Distribution Agreement

In presenting this thesis or dissertation as a partial fulfillment of the requirements for an advanced degree from Emory University, I hereby grant to Emory University and its agents the non-exclusive license to archive, make accessible, and display my thesis or dissertation in whole or in part in all forms of media, now or hereafter known, including display on the world wide web. I understand that I may select some access restrictions as part of the online submission of this thesis or dissertation. I retain all ownership rights to the copyright of the thesis or dissertation. I also retain the right to use in future works (such as articles or books) all or part of this thesis or dissertation.

Signature:

_____ _____

Jiabei He Date

The Association of Urinary Phthalate Concentrations with Asthma-Related Outcomes among Adults: NHANES 2003-2012

By

Jiabei He

Master of Public Health

Environmental Health

Dana Boyd Barr, PhD

Committee Chair

Terryl J. Hartman, PhD, MPH, RD

Committee Member

Paige Tolbert, PhD

Committee Member

The Association of Urinary Phthalate Concentrations with Asthma-Related Outcomes among Adults: NHANES 2003-2012

By

Jiabei He

B.Med.

Southern Medical University

2015

Thesis Committee Chair: Dana Boyd Barr, Ph.D.

An abstract of

A thesis submitted to the Faculty of the
Rollins School of Public Health of Emory University

in partial fulfillment of the requirements for the degree of
Master of Public Health
in Environmental Health

2017

Abstract

The Association of Urinary Phthalate Concentrations with Asthma-Related Outcomes among Adults: NHANES 2003-2012

By Jiabei He

Background: Phthalates are a family of synthetic chemicals used in a wide spectrum of plastic products, such as personal care products, building materials, food packages, medical devices and pharmaceuticals. Phthalates are classified into low and high molecular weight compounds. Previous studies indicate that phthalates are related to allergic and inflammatory processes in humans. Asthma is a common pulmonary disease characterized by airway hypersensitivity. We conducted this analysis to evaluate the relationship between urinary phthalate metabolite concentrations and asthma-related outcomes.

Methods: Five 2-year cycles of the National Health and Nutrition Examination Survey (NHANES 2003-2012) were merged for our analysis dataset. A total of 7,298 adult participants with available urinary phthalates metabolites, asthma, and covariate data were included. After stratification and confounder adjustment, logistic regression was performed that incorporated sample weights to account for the complex, multistage probability sampling design used to select participants that represent the non-institutionalized US population.

Results: Mono-n-methyl phthalate (MNM) was positively associated (OR: 1.25, CI: 1.04-1.50) with ever asthma among all adults. Among adult females, MCNP, MCOP and MCPP were positively associated with ever asthma (ORs=1.26-1.34). Among females reporting ever asthma, MiBP was positively associated with past asthma (OR: 1.83, CI: 1.10-3.04). Among black females, MCPP and MEHP were associated with ever asthma (ORs: 1.44, CI: 1.02-2.05 and OR: 1.55, CI 1.08-2.21, respectively). Among white females reporting a history of asthma, MiBP was positively associated with past asthma (OR: 2.29, CI: 1.11-4.71).

Conclusion: In this cross-sectional sample of US adults we did not observe a consistent pattern of association with asthma-related outcomes across the phthalate metabolites measured, however, the associations we did observe were positive. Given the cross-sectional design of the survey, we cannot infer causality and because of the variable associations observed, the observations may be a result of chance alone.

The Association of Urinary Phthalate Concentrations with Asthma-Related Outcomes among Adults: NHANES 2003-2012

By

Jiabei He

B.Med.

Southern Medical University

2015

Thesis Committee Chair: Dana Boyd Barr, Ph.D.

An abstract of

A thesis submitted to the Faculty of the
Rollins School of Public Health of Emory University

in partial fulfillment of the requirements for the degree of
Master of Public Health
in Environmental Health

2017

Tables of Contents

The Association of Urinary Phthalate Concentrations with Asthma-Related Outcomes among Adults: NHANES 2003-2012

BACKGROUND

Phthalates

Phthalates, dialkyl- or alkyl/aryl esters of 1,2-benzenedicarboxylic acid (Wolff et al., 2014), are a family of synthetic chemicals used in a wide spectrum of products, including personal care products, plastics, building materials, food packages, medical devices and pharmaceuticals (ATSDR, 1995, 1997, 2001, 2002; Latini, 2005; North, Takaro, Diamond, & Ellis, 2014). Phthalates are used in industrial manufacturing to help polyvinyl chloride (PVC) or vinyl plastics maintain flexibility and durability with cost-effectiveness and to hold color, fragrance and shine (Barr et al., 2003; Jurewicz & Hanke, 2011; Sanchez-Prado, Llompart, Lamas, Garcia-Jares, & Lores, 2011). Selected phthalates have been used to enable "time release" in certain medications (ATSDR, 1995, 2001; David, McKee, Butala, Barter, & Kayser, 2001).

Phthalates are commonly classified into two groups to better define their sources and uses: low molecular weight phthalates (LMWP) with 3-6 carbon atoms in their backbone, and high molecular weight phthalates (HMWP) with more than 6 carbons in their backbone. LMW phthalates include diethylphthalate (DEP), dibutylphthalate (DBP) and benzylbutyl phthalate (BBzP) which generate the monoester metabolites monoethyl phthalate (MEP), mono-n-butyl phthalate (MnBP), mono-isobutyl phthalate (MiBP) and mono-n-methyl phthalate (MMP). HMW phthalates include di-2-ethylhexyl phthalate (DEHP) which forms several metabolites including mono-2-ethyl-5-carboxypentyl phthalate (MECPP), mono-(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP), mono-(2-ethyl)-hexyl phthalate (MEHP), and mono-(2-ethyl-5-oxyhexyl) phthalate (MEOHP) (Hoppin et al., 2013). LMW phthalates are commonly found in cosmetics, personal care

products, and solvents for cellulose acetate, such as capsule coating to provide timed releases in certain pharmaceuticals (ATSDR, 1995, 2001; David et al., 2001). In contrast, HMW phthalates are primarily used as plasticizers, particularly in building materials, such as flooring and wall coverings, in food contact applications, and in components of medical devices (ATSDR, 1997, 2002; David et al., 2001).

Humans can be exposed to phthalates via multiple routes and pathways. Ingestion is an important route of exposure to the HMW phthalates, which can be consumed as a result of frequent contact of food and beverages with packaging containing PVC (Koch et al., 2013; Rudel et al., 2011; Whyatt et al., 2014). Exposure to phthalates can also occur through inhalation, dermal absorption and transplacental transfer (ATSDR, 1995, 1997, 2001, 2002; Braun et al., 2016; Holland et al., 2016). After entering the human body, phthalates are rapidly metabolized and excreted by urination and defecation with biologic half-lives ranging from 3-18 hours (Genuis, Beesoon, Lobo, & Birkholz, 2012).

Non-occupational exposure of the general U.S. population to phthalates is widespread, although variation related to demographic covariates such as age, gender, race/ethnicity, geographic location and temporal factors may be present (Koch et al., 2016). The average urinary concentration of the DEHP metabolite was estimated to be 85.47 µg/L in the US population. Average concentrations of its composition metabolites MECPP, MEHP and MEORP was estimated to be 38.28 µg/L, 25.35 µg/L and 16.14 µg/L, respectively, according to the summary analyses of NHANES 2005 – 2006 (Hoppin et al., 2013). Although LMW products have gradually been replaced by HMW phthalates or phthalates-free products, HMW phthalates still play a major role in the US market. In the United States and Germany, exposure to the most common phthalates has decreased from 1999 to 2012, but exposures to HMW phthalates substituting for DEHP (such as DiNP) have dramatically increased, particularly in the United States (Koch et al., 2016).

Asthma

Since phthalates are well-known endocrine disruptors, a large amount of research focuses on effects on the reproductive system, the thyroid and metabolism (Huang, Kuo, Guo, Liao, & Lee, 2007; Lyche et al., 2009). We are interested in its influence on respiratory system.

Asthma, characterized by recurrent bronchial hyperresponsiveness, is one of most common pulmonary diseases in the United States. In 2011-2014, asthma prevalence was 8.8% among adults. Overall asthma prevalence among adults increased from 2001-2002 (7.1%) to 2013-2014 (9.2%) (CDC). Common clinical symptoms of asthma include coughing, wheezing, shortness of breath and chest tightness. Symptoms are typically exacerbated at night and in the early morning or related to exercise or inspiration of cold air in winter. Airway inflammation, airflow obstruction, and irreversible airway remodeling are believed to be involved in asthma development.

The respiratory system of a human contains two lungs, the trachea, the bronchi in the mediastinum, and the bronchial trees (bronchi branches). The trachea and its large proximal branches form a passageway for air exchange between the lung and the external environment (Gilroy, 2013). Asthma primarily targets at the bronchi and its subdivisions and conducting bronchioles.

The pathogenesis of asthma can be classified into two categories. One is intrinsic asthma mainly caused by viral infection (e.g., rhinovirus, parainfluenza virus, respiratory syncytial virus), air pollutants, aspirin or other nonsteroidal medication use (Goljan, 2013). The other one is extrinsic asthma which involves a classical inflammation process. Mast cells, IgE antibodies, leukotrienes, chemokines and cytokines participate in the inflammation process and lead to histologic changes, such as thickening of the basement membranes, infiltration of inflammatory cells, hypertrophy of mucous glands, hypertrophy of smooth muscle cells and hyperplasia (Kumar, Abbas, & Aster, 2012). Narrowing of the bronchi causes expiratory wheezing in asthma, and the air trapping in

distal bronchioles leads to the increased anteroposterior thoracic diameter, especially in long-term untreated asthma patients.

Although the mechanisms have not yet been elucidated, there are several encouraging laboratory research findings pertaining to the cellular mechanisms through which phthalates may influence inflammation and allergic response. For example, the production of pro-inflammatory chemokines and cytokines in human macrophages increased when human macrophages were exposed to the diethyl hexyl phthalate (DEHP) (Nishioka et al., 2012). DEHP can also stimulate mast cells to degranulate and release histamine and beta-hexosaminidase, which is related to asthma severity (Kuo et al., 2013). In addition, i*n vitro* studies indicate diethyl phthalate (DEP) could increase the production of pro-allergic TH-2 cytokines including IL-4 and TNF-alpha in human macrophages (Ochiai et al., 2014).

A cross-sectional study in 2004 found associations between urinary levels of MBP and lower pulmonary function (forced vital capacity (FVC), forced expiratory volume in 1 second (FEV1), peak expiratory flow (PEF), and maximum mid-expiratory flow (MMEF)) in the National Health and Nutrition Examination (NHANES) III. Results for a group of 240 adults suggested that MEP was associated with lower FVC and FEV1 in men but not women. The underlying mechanisms for these observations are still unclear (Hoppin, Ulmer, & London, 2004). A more recent study in elderly Koreans observed an inverse association between the sum of mono-(2-ethyl-5-hydroxyhexyl) phthalate and mono-(2-ethyl-5-oxohexyl) phthalate (ΣDEHP) levels and FEV1/FVC or FEF25-75 after adjusting for potential confounders (H. Y. Park, Kim, Lim, Bae, & Hong, 2013). FEV1/FVC is considered as essential spirometry evidence for asthma diagnosis. In NHANES 2005-2006, MBzP, one of the HMW phthalate metabolites, was positively associated with allergic symptoms including asthma, wheeze, allergy, rhinitis and hay fever in adults (asthma: OR:1.46, CI: 1.01-2.11; wheeze: OR:1.78, CI:1.22-2.60; hay fever: OR:1.68, CI:1.09-2.59; rhinitis: OR: 1.27, CI: 1.01-1.52) but the associations were all nonsignificant and inconsistent among

children 7-19 years of age (asthma: OR:1.06, CI: 0.33-3.45; wheeze: OR:0.92, CI:0.35-2.37; hay fever: OR:0.42, CI:0.22-0.79; rhinitis: OR: 1.02, CI: 0.62-1.67) (Hoppin et al., 2013).

Hypothesis

Limited epidemiological evidence supports an association between phthalate esters and asthma symptoms. In the last few decades, phthalates have become ubiquitous environmental contaminants while the prevalence of asthma has increased correspondingly. Thus, we hypothesize that phthalate metabolite concentrations are positively associated with asthma-related outcomes in a cross-sectional, representative sample of the US population (NHANES 2003-2012).

METHODS

Data Source and Study Population

The data used to evaluate the association between phthalate exposure and asthma-related outcomes in this study were obtained from the National Health and Nutrition Examination Survey (NHANES). NHANES is an ongoing cross-sectional survey fielded since 1999 by the Centers for Disease Control and Prevention (CDC). It is based on a complex multistage sampling method, designed to collect data on the health and nutritional status of the civilian, non-institutionalized US population (CDC, 2013a, 2013b). Demographic information, dietary data, questionnaires, a physical examination and laboratory data were collected every two years for each survey cycle. All data were de-identified before being released and made publicly accessible.

Five survey cycles from 2003 to 2012 were merged to ensure sufficient sample size. Although about 2,500 participants were sampled for each 2-year study cycle, phthalates were only measured on a 1/3 subset; the subset was randomly selected to maintain representativeness.

Our analysis was restricted to the adult participants 20 years and older at the time of recruitment who had complete information on asthma-related outcomes, urinary phthalates concentrations and model covariates. A total of 8,457 adult NHANES participants aged 20 and older had data on 11 urinary phthalates metabolites (Figure 1). CNP and COP were not measured in 2003-2004, and MCP and MOP were not measured in 2011-2012. We did not exclude participants based on data availability for CNP, COP, MCP and MOP. After excluding 11 individuals with missing values in the "ever asthma" question and 1,148 with missing values in covariates, we had a final sample of 7,298 participants.

Measurement of Exposures and Outcomes

A total of 15 urinary phthalates metabolite concentrations were obtained from the NHANES laboratory dataset. Spot urine samples were collected at one of three daily sampling times (morning, early afternoon, late afternoon) in NHANES mobile examination centers from participants aged 6 years and older (CDC, 2013c). Urinary phthalate metabolites were measured using isotope dilution high-performance liquid chromatography-electrospray ionization-tandem mass spectrometry (HPLC-ESI-MS/MS) at the National Center for Environmental Health laboratory.

Asthma and wheeze information were extracted from two participant questionnaires (Medical Conditions and Respiratory Health). To assess asthma prevalence, participants (or their proxies) who completed the Medical Conditions questionnaire were asked in consecutive questions "Has a doctor or other health professional ever told {you/sample person (SP)} that {you have/s/he SP has} asthma" (ever asthma) and if "yes" "{Do you/ Does SP} still have asthma?" The latter question resulted in mutually exclusive responses which we used to define asthma persistence and were coded as current asthma or past asthma. Respondents who indicated they had current asthma were queried "During the past 12 months, {have you/ has SP"} had an episode of asthma or an asthma attack?" (asthma attack in past year). The Respiratory Health

questionnaire asked participants “In the past 12 months {have you/ has SP} had wheezing or whistling in {your/his/her} chest?” (wheezing in past year).

Covariates

Covariates of interest included: age (continuous), ethnicity (white/black/Hispanic/other ethnicities), gender (male/female), poverty income ratio (PIR), body mass index (BMI), urinary creatinine and serum cotinine. Family PIR was estimated using guidelines and adjustment for family size, year and state (CDC, 2013a, 2013b). PIR was categorized as 0.00–1.30, >1.30–3.50, and >3.50 and above. BMI was calculated as weight in kilograms divided by height in meters squared ($kg/(m)^2$) and classified as underweight (≤18), normal (18-25), overweight (25-30), or obese (≥30). In our models, age, BMI, PIR, urinary creatinine and serum cotinine are continuous variables, while ethnicity and gender are categorical variables.

Statistical Analysis

All data cleaning, descriptive and statistical analyses were performed using SAS 9.4 (SAS Institute, Cary, NC). The significance level was set as 0.05 for all hypothesis tests.

Multivariate logistic regression models were used to evaluate the association between phthalate metabolites and asthma-related outcomes after controlling for important confounders. In sequential analyses we evaluated the associations of phthalates with ever having asthma in the overall population, then among the subset reporting asthma, we assessed persistence (past v. current) and severity (attack, in the past year). The analyses for presence of wheeze in the past year was conducted using the subset of the overall population who completed the Respiratory Health questionnaire and also had available data for ever having asthma, phthalate metabolites and covariates of interest.

Normality assumptions for each metabolite were checked by histograms, normal probability plots, and Kolmogorov-Smirnov tests. In our analyses, urinary phthalates metabolite

concentrations were log10-transformed because they were not normally distributed. Logistic models were fitted for each phthalate metabolite separately for the associations with ever asthma, current asthma, past asthma, asthma attack in past year and wheeze in past year. The phthalate subset-specific population weights were applied in our analyses (CDC, 2013a).

A priori we planned to stratify analyses by sex because previous results had observed different effect estimates by gender (C. Park et al., 2017). We used a forward selection procedure to evaluate potential confounders (Kleinbaum, Kupper, Nizam, & Rosenberg, 2013). The confounders included in our final models were age, BMI, PIR, serum cotinine and urinary creatinine. We evaluated potential interaction by ethnicity/race by including the categorical ethnicity/race variables and their cross-product terms with the continuous phthalate metabolite variables in separate multivariate models with the likelihood ratio tests. Results are reported as multivariate adjusted odds ratios (OR) with 95% confidence intervals (CI).

RESULTS

Descriptive statistics

The availability of data for 15 phthalate metabolites levels by each survey cycle is reported in Table 1. Table 2 presents the study participant characteristics. The final number of samples included in the analyses was 7298, or approximately two-thirds (68.8%) of the total population in the phthalate data subset. Overall, 3715 (35.6%) participants were female and 3583 (33.3%) participants were male. The average age was 46.5 years old with a standard error of 0.3 years. Of the participants, 3534 (48.8%) were non-Hispanic white, 1482 (7.3%) were non-Hispanic black, 1761 (8.5%) were Hispanic and 521 (4.2%) were other ethnicities/races. Among the population studied, 19.7% had serum cotinine levels > 10 ng/mL, 24.0% were obese, and

30.1% had a family poverty income ratio above 3.5. For the asthma related outcome variables, 965 (9.2%) of adult participants reported a history of asthma, 576 (5.3%) reported current asthma, and 389 (3.9%) had asthma in the past. In the past year, 264 (2.5%) had an asthma attack and 199 (1.9%) had wheezing episodes.

The geometric means, geometric standard errors and distribution percentiles of the sample population are shown in Tables 3, 3-1, 3-2. MEP was the most frequently detected (99.9% >LOD) metabolite among LMWP and MECCP was the most frequently detected metabolite (99.9% >LOD) among HMWP. The frequencies of detection of MCHP, MiNP and MOP were less than 25%, thus they were not included in regression models.

Logistic regression

Tables 4 presents the results of logistic regression analyses for all adults. After adjusting for covariates, Mono-n-methyl phthalate (MNM) was positively associated (OR: 1.25, CI: 1.04-1.50) with ever asthma among all adults.

The effect estimates stratified by gender and ethnicity are reported in Tables 5-12. We observed several differences by gender and ethnicity, but the stratified results of some ethnicities were not reported due to small sample sizes. Among adult females (Table 6), MCNP, MCOP, and MCPP were positively associated with ever asthma (MCNP: OR:1.26, CI:1.00-1.57; MCOP: OR:1.29, CI:1.03-1.61; MCPP: OR: 1.34, CI: 1.08-1.65). Among those who reported ever asthma, MiBP was positively associated with past asthma (OR: 1.83, CI: 1.10-3.04), but was negatively associated with current asthma (OR: 0.52, CI: 0.30-0.90). Among male adults (Table 7), MnBP was positively associated with wheeze in past year (OR: 2.08, CI: 1.01-4.30). MnBP was negatively associated with asthma attack in past year (OR: 0.41, CI: 0.18-0.95) among male adults who reporting current asthma.

In our stratified analyses, among black females (Table 7), MCPP and MEHP were associated with ever asthma (MCPP: OR:1.55, CI: 1.08-2.21; MEHP: OR: 1.44, CI: 1.02-2.05). Among white females who reported ever asthma, MiBP was positively associated with past asthma (OR: 2.29, CI: 1.11-4.71), but was negatively associated with current asthma (OR: 0.42, CI: 0.20-0.90). Any strata where the number of participants reporting asthma-related outcomes (ever asthma, current asthma, past asthma, asthma attack in the past year, and the wheezing in the past year) of less than 50 was considered exploratory and therefore was not reported in our results, but were displayed in our tables listed in the appendix.

DISCUSSION

In this cross-sectional study, the associations between urinary phthalate metabolites and asthma related outcomes were evaluated in adults sampled in NHANES 2003-2012. Analyses were stratified by gender and ethnicity and controlled for several important confounders including age, BMI, PIR, serum cotinine and urinary creatinine.

Although the urinary phthalate metabolite concentrations are similar to those reported previously for NHANES 2005-2006 (Hoppin et al., 2013), our conclusions based on a larger sample with the inclusion of additional survey cycles, are somewhat different. We did not observe a significant association between MBzP and current asthma or current wheezing among overall adults as previously reported (Hoppin et al., 2013). Although ethnicity was considered as an interaction term in their models, stratified results were not included. In our analyses, MNM was positively associated with ever asthma among all adults. Among respondent with asthma, MBzP was positively associated with asthma attacks in the past year and wheezing in the past year but only among the black female population. However, since the number of black females reporting

asthma attacks in the past year and wheezing in the past year was small, these results should be interpreted cautiously.

There may be a number or reasons why we observed different results by sex and race /ethnicity. Such differences may in part be explained by selected behaviors associated with higher phthalate levels in minorities. In a recently published research, Zola and colleagues reported a dose-response relationship between fast food consumption and exposure to phthalates. 35% Hispanic and 43.8% non-Hispanic black consumed fast food, while 32.5% non-Hispanic white consumed fast food. Fast food-derived dietary fat intake was also positively associated with ΣDEHP and DiNP (Zota, Phillips, & Mitro, 2016). In addition, according to a CDC 2008-2010 report, there was a higher asthma prevalence among blacks (11.2%), when compared to whites (7.7%) (Akinbami et al., 2012). These sex and ethnic differences need to be further explored in large populations.

One strength in our study is that 5 survey cycles were combined to improve our study power and to conduct analyses stratified by sex and ethnicity. Another strength of our study is that "ever asthma" was categorized into two variables: current asthma and past asthma. Since asthma is not curable but is controllable with medication and technology, it is appropriate to separate past history and current asthma. Additionally, the laboratory responsible for NHANES urinary analyses has extensive quality control procedures in place.

By design NHANES is a cross-sectional study which is a limitation of our analyses. The lack of longitudinal data in NHANES does not allow causal inferences to be drawn from the data. Reverse causation may be a consideration. For example, asthma inhalers may increase the exposure to phthalates or vice versa. A priori we planned to stratify analyses by sex and race/ethnicity. Although we found significant associations between phthalates metabolites and asthma related outcomes in some strata of sex and race/ethnicity, these were at times based on more modest sample sizes and such should be considered exploratory and interpreted with

caution. In addition, since CNP and COP were not measured during 2003-2004 survey cycle, and MCP and MOP were not measured during 2011-2012 survey cycle, and our results might be influenced by these unavailable data.

In conclusion, in this cross-sectional sample of US adults we did not observe a consistent pattern of direction or magnitude of association with asthma-related outcomes across the phthalate metabolites measured. For example, only mono-n-methyl phthalate (MNM) was positively associated (OR: 1.25, CI: 1.04-1.50) with ever asthma among all adults. We found some positive associations for HMWP and ever asthma among females but results for white and black females were not entirely consistent. There were no significant associations between phthalate exposure and ever asthma among men. The role of environmental chemicals, like phthalates, in the development of respiratory diseases is an important topic of emerging research and additional research utilizing large sample sizes is needed in this area.

REFERENCES

Akinbami, L. J., Moorman, J. E., Bailey, C., Zahran, H. S., King, M., Johnson, C. A., & Liu, X. (2012). Trends in asthma prevalence, health care use, and mortality in the United States, 2001–2010. *NCHS data brief, 94*(94), 1-8.

ATSDR. (1995). Toxicological profile for diethylphthalate. from U.S. Department of Health and Human Services, Public Health Service

ATSDR. (1997). Toxicological profile for di-n-octylphthalate. from U.S. Department of Health and Human Services, Public Health Service

ATSDR. (2001). Toxicological profile for di-n-butyl phthalate. from U.S. Department of Health and Human Services, Public Health Service

ATSDR. (2002). Toxicological profile for di(2-ethylhexyl)phthalate. from U.S. Department of Health and Human Services, Public Health Service

Barr, D. B., Silva, M. J., Kato, K., Reidy, J. A., Malek, N. A., Hurtz, D., . . . Calafat, A. M. (2003). Assessing human exposure to phthalates using monoesters and their oxidized metabolites as biomarkers. *Environmental Health Perspectives, 111*(9), 1148.

Braun, J. M., Bellinger, D. C., Hauser, R., Wright, R. O., Chen, A., Calafat, A. M., . . . Lanphear, B. P. (2016). Prenatal phthalate, triclosan, and bisphenol A exposures and child visual-spatial abilities. *Neurotoxicology, 58*, 75-83. doi:10.1016/j.neuro.2016.11.009

CDC. (2013a). National Health and Nutrition Examination Survey: Analytic Guidelines, 1999-2010.

CDC. (2013b). *National Health and Nutrition Examination Survey: Analytic Guidelines, 2011-2012*. Retrieved from https://www.cdc.gov/nchs/data/nhanes/analytic_guidelines_11_12.pdf

CDC. (2013c). *National Health and Nutrition Examination Survey: Plan and Operations*. Retrieved from Hyattsville, Maryland: https://www.cdc.gov/nchs/data/series/sr_01/sr01_056.pdf

David, R. M., McKee, R. H., Butala, J. H., Barter, R. A., & Kayser, M. (2001). Esters of Aromatic Mono-, Di-, and Tricarboxylic Acids, Aromatic Diacids and Di-, Tri-, Or Polyalcohols. *Patty's toxicology*.

Genuis, S. J., Beesoon, S., Lobo, R. A., & Birkholz, D. (2012). Human elimination of phthalate compounds: blood, urine, and sweat (BUS) study. *ScientificWorldJournal, 2012*, 615068. doi:10.1100/2012/615068

Gilroy, A. M. (2013). Anatomy: An Essential Textbook.

Goljan, E. F. (2013). *Rapid review pathology: with student consult online access*: Elsevier Health Sciences.

Holland, N., Huen, K., Tran, V., Street, K., Nguyen, B., Bradman, A., & Eskenazi, B. (2016). Urinary Phthalate Metabolites and Biomarkers of Oxidative Stress in a Mexican-American Cohort: Variability in Early and Late Pregnancy. *Toxics, 4*(1). doi:10.3390/toxics4010007

Hoppin, J. A., Jaramillo, R., London, S. J., Bertelsen, R. J., Salo, P. M., Sandler, D. P., & Zeldin, D. C. (2013). Phthalate exposure and allergy in the U.S. population: results from NHANES 2005-2006. *Environ Health Perspect, 121*(10), 1129-1134. doi:10.1289/ehp.1206211

Hoppin, J. A., Ulmer, R., & London, S. J. (2004). Phthalate exposure and pulmonary function. *Environ Health Perspect, 112*(5), 571-574.

Huang, P.-C., Kuo, P.-L., Guo, Y.-L., Liao, P.-C., & Lee, C.-C. (2007). Associations between urinary phthalate monoesters and thyroid hormones in pregnant women. *Human reproduction, 22*(10), 2715-2722.

Jurewicz, J., & Hanke, W. (2011). Exposure to phthalates: reproductive outcome and children health. A review of epidemiological studies. *Int J Occup Med Environ Health, 24*(2), 115-141. doi:10.2478/s13382-011-0022-2

Kleinbaum, D., Kupper, L., Nizam, A., & Rosenberg, E. (2013). *Applied regression analysis and other multivariable methods*: Nelson Education.

Koch, H. M., Lorber, M., Christensen, K. L., Palmke, C., Koslitz, S., & Bruning, T. (2013). Identifying sources of phthalate exposure with human biomonitoring: results of a 48h fasting study with urine collection and personal activity patterns. *Int J Hyg Environ Health, 216*(6), 672-681. doi:10.1016/j.ijheh.2012.12.002

Koch, H. M., Ruther, M., Schutze, A., Conrad, A., Palmke, C., Apel, P., . . . Kolossa-Gehring, M. (2016). Phthalate metabolites in 24-h urine samples of the German Environmental Specimen Bank (ESB) from 1988 to 2015 and a comparison with US NHANES data from 1999 to 2012. *Int J Hyg Environ Health*. doi:10.1016/j.ijheh.2016.11.003

Kumar, V., Abbas, A. K., & Aster, J. C. (2012). *Robbins basic pathology*: Elsevier Health Sciences.

Kuo, C. H., Hsieh, C. C., Kuo, H. F., Huang, M. Y., Yang, S. N., Chen, L. C., . . . Hung, C. H. (2013). Phthalates suppress type I interferon in human plasmacytoid dendritic cells via epigenetic regulation. *Allergy, 68*(7), 870-879. doi:10.1111/all.12162

Latini, G. (2005). Monitoring phthalate exposure in humans. *Clin Chim Acta, 361*(1-2), 20-29. doi:10.1016/j.cccn.2005.05.003

Lyche, J. L., Gutleb, A. C., Bergman, Å., Eriksen, G. S., Murk, A. J., Ropstad, E., . . . Skaare, J. U. (2009). Reproductive and developmental toxicity of phthalates. *Journal of Toxicology and Environmental Health, Part B, 12*(4), 225-249.

Nishioka, J., Iwahara, C., Kawasaki, M., Yoshizaki, F., Nakayama, H., Takamori, K., . . . Iwabuchi, K. (2012). Di-(2-ethylhexyl) phthalate induces production of inflammatory

molecules in human macrophages. *Inflamm Res, 61*(1), 69-78. doi:10.1007/s00011-011-0390-x

North, M. L., Takaro, T. K., Diamond, M. L., & Ellis, A. K. (2014). Effects of phthalates on the development and expression of allergic disease and asthma. *Ann Allergy Asthma Immunol, 112*(6), 496-502. doi:10.1016/j.anai.2014.03.013

Ochiai, S., Roediger, B., Abtin, A., Shklovskaya, E., Fazekas de St Groth, B., Yamane, H., . . . Ronchese, F. (2014). CD326(lo)CD103(lo)CD11b(lo) dermal dendritic cells are activated by thymic stromal lymphopoietin during contact sensitization in mice. *J Immunol, 193*(5), 2504-2511. doi:10.4049/jimmunol.1400536

Park, C., Choi, W., Hwang, M., Lee, Y., Kim, S., Yu, S., . . . Choi, K. (2017). Associations between urinary phthalate metabolites and bisphenol A levels, and serum thyroid hormones among the Korean adult population - Korean National Environmental Health Survey (KoNEHS) 2012-2014. *Sci Total Environ*. doi:10.1016/j.scitotenv.2017.01.144

Park, H. Y., Kim, J. H., Lim, Y. H., Bae, S., & Hong, Y. C. (2013). Influence of genetic polymorphisms on the association between phthalate exposure and pulmonary function in the elderly. *Environ Res, 122*, 18-24. doi:10.1016/j.envres.2012.11.004

Rudel, R. A., Gray, J. M., Engel, C. L., Rawsthorne, T. W., Dodson, R. E., Ackerman, J. M., . . . Brody, J. G. (2011). Food packaging and bisphenol A and bis(2-ethyhexyl) phthalate exposure: findings from a dietary intervention. *Environ Health Perspect, 119*(7), 914-920. doi:10.1289/ehp.1003170

Sanchez-Prado, L., Llompart, M., Lamas, J. P., Garcia-Jares, C., & Lores, M. (2011). Multicomponent analytical methodology to control phthalates, synthetic musks, fragrance allergens and preservatives in perfumes. *Talanta, 85*(1), 370-379. doi:10.1016/j.talanta.2011.03.079

Whyatt, R. M., Perzanowski, M. S., Just, A. C., Rundle, A. G., Donohue, K. M., Calafat, A. M., . . . Miller, R. L. (2014). Asthma in inner-city children at 5-11 years of age and

prenatal exposure to phthalates: the Columbia Center for Children's Environmental Health Cohort. *Environ Health Perspect, 122*(10), 1141-1146. doi:10.1289/ehp.1307670

Wolff, M. S., Teitelbaum, S. L., McGovern, K., Windham, G. C., Pinney, S. M., Galvez, M., . . . Environment Research, P. (2014). Phthalate exposure and pubertal development in a longitudinal study of US girls. *Hum Reprod, 29*(7), 1558-1566. doi:10.1093/humrep/deu081

Zota, A. R., Phillips, C. A., & Mitro, S. D. (2016). Recent Fast Food Consumption and Bisphenol A and Phthalates Exposures among the U.S. Population in NHANES, 2003-2010. *Environ Health Perspect, 124*(10), 1521-1528. doi:10.1289/ehp.1510803

Appendix:

Figure 1. Schematic diagram of inclusion criteria and sample counts

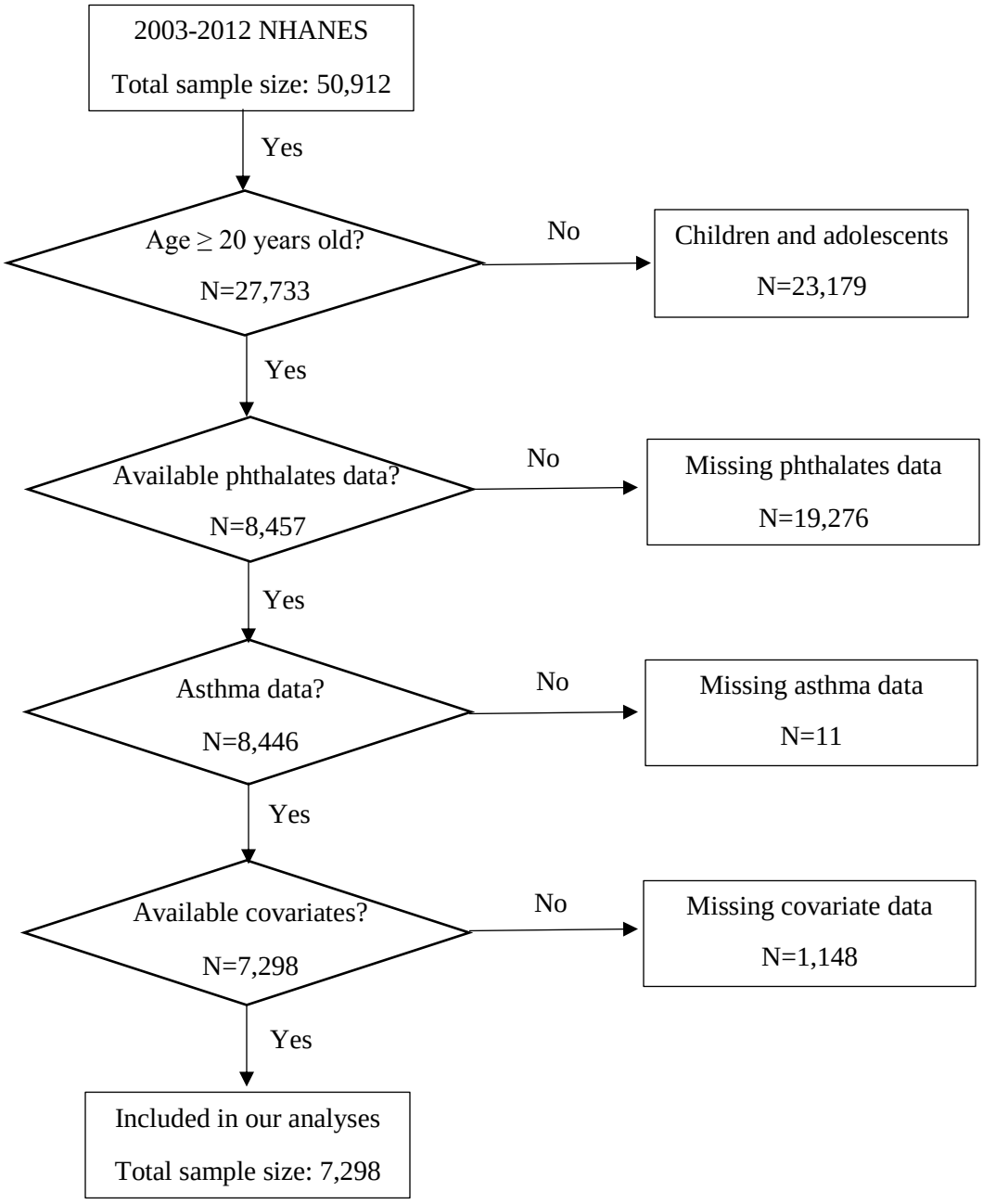

Table 1. Availability of 15 phthalate metabolites by each survey cycle

Survey circle	total n	MBP		MCP		MEP		MHP		MNP	
		n AVL	% AVL	n AVL	% AVL	n AVL	% AVL	n AVL	% AVL	n AVL	% AVL
Total	50912	12995		10506		12995		12995		12995	
2003-2004	10122	2605	25.74	2605	25.74	2605	25.74	2605	25.74	2605	25.74
2005-2006	10348	2548	24.62	2548	24.62	2548	24.62	2548	24.62	2548	24.62
2007-2008	10149	2604	25.66	2604	25.66	2604	25.66	2604	25.66	2604	25.66
2009-2010	10537	2749	26.09	2749	26.09	2749	26.09	2749	26.09	2749	26.09
2011-2012	9756	2489	25.51	0	0	2489	25.51	2489	25.51	2489	25.51

Survey circle	total n	MOP		MZP		MNM		MC1		MHH	
		n AVL	% AVL	n AVL	% AVL	n AVL	% AVL	n AVL	% AVL	n AVL	% AVL
Total	50912	10506		12995		12995		12995		12995	
2003-2004	10122	2605	25.74	2605	25.74	2605	25.74	2605	25.74	2605	25.74
2005-2006	10348	2548	24.62	2548	24.62	2548	24.62	2548	24.62	2548	24.62
2007-2008	10149	2604	25.66	2604	25.66	2604	25.66	2604	25.66	2604	25.66
2009-2010	10537	2749	26.09	2749	26.09	2749	26.09	2749	26.09	2749	26.09
2011-2012	9756	0	0	2489	25.51	2489	25.51	2489	25.51	2489	25.51

Survey circle	total n	MOH		MIB		ECP		CNP		COP	
		n AVL	% AVL	n AVL	% AVL	n AVL	% AVL	n AVL	% AVL	n AVL	% AVL
Total	50912	12995		12995		12995		10390		10390	
2003-2004	10122	2605	25.74	2605	25.74	2605	25.74	0	0	0	0
2005-2006	10348	2548	24.62	2548	24.62	2548	24.62	2548	24.62	2548	24.62
2007-2008	10149	2604	25.66	2604	25.66	2604	25.66	2604	25.66	2604	25.66
2009-2010	10537	2749	26.09	2749	26.09	2749	26.09	2749	26.09	2749	26.09
2011-2012	9756	2489	25.51	2489	25.51	2489	25.51	2489	25.51	2489	25.51

*AVL: available

Table 2. Characteristics of study participants with weighted percentages and standard errors

	Overall		Female		Male	
Characteristic	N	Weighted percent (SE)	N	Weighted percent (SE)	N	Weighted percent (SE)
Sample size	7298	68.8 (0.7)	3715	35.6 (0.6)	3583	33.3 (0.5)
Age, mean (SE)		46.52 (0.3)		46.9 (0.4)		45.9 (0.4)
Ethnicity						
Non-Hispanic White	3534	48.8 (1.3)	1777	25.0 (0.8)	1757	23.8 (0.8)
Non-Hispanic Black	1482	7.3 (0.6)	756	4.1 (0.3)	726	3.2 (0.2)
Hispanic	1761	8.5 (0.7)	917	4.2 (0.4)	844	4.3 (0.4)
Others	521	4.2 (0.3)	265	2.3 (0.2)	256	1.9 (0.2)
Cotinine (ng/mL)						
<LOD (<0.015)	1554	16.7 (0.7)	965	10.4 (0.5)	589	6.3 (0.4)
Low (0.015 - 10)	3852	36.7 (0.8)	1973	19.0 (0.6)	1879	17.7 (0.5)
High(>10)	1892	19.7 (0.6)	777	8.4 (0.4)	1115	11.3 (0.5)
BMI (kg/(m^2))						
Underweight (<18.5)	117	1.1 (0.1)	82	0.9 (0.1)	35	0.2 (0.1)
Normal (18.5 -25)	2069	20.9 (0.6)	1108	12.1 (0.4)	961	8.8 (0.4)
Overweight (25 -30)	2461	22.8 (0.7)	1097	10.3 (0.4)	1364	12.6 (0.4)
Obese(≥30)	2651	24.0 (0.6)	1428	12.3 (0.4)	1223	11.7 (0.5)
PIR						
0.0-1.3	2302	14.8 (0.6)	1282	8.4 (0.3)	1020	6.1 (0.3)
>1.3-3.5	2676	24.3 (0.7)	1317	12.5 (0.5)	1359	11.8 (0.4)
>3.5	2320	30.1 (0.9)	1116	14.7 (0.6)	1204	15.4 (0.5)
Ever have asthma (% yes)	965	9.2 (0.4)	555	5.3 (0.3)	410	3.9 (0.3)
- Current asthma (% yes)	576	5.3 (0.3)	352	3.3 (0.2)	224	2.1 (0.2)
- Past asthma (% yes)	389	3.9 (0.2)	203	2.0 (0.2)	186	1.8 (0.2)
Asthma attack past year (% yes)	264	2.5 (0.2)	180	1.6 (0.2)	84	0.9 (0.1)
Wheeze past year (% yes)	199	1.9 (0.2)	102	1.1 (0.1)	97	0.8 (0.1)

*SE: Standard error

Table 3. Urinary phthalate metabolites concentration (ug/L), adult overall

Metabolites		> LOD (%)	GM (GSE)	Percentile				
				5th	25th	50th	75th	95th
*LMW			1.70 (0.04)	0.24	0.82	1.74	3.56	11.47
MEP	Mono-ethyl phthalate	99.9	81.42 (2.54)	7.00	26.29	75.49	228.77	1348.35
MiBP	Mono-isobutyl phthalate	98.0	5.25 (0.13)	0.59	2.60	5.79	11.69	31.11
MnBP	Mono-n-butyl phthalate	98.1	13.78 (0.38)	1.49	6.90	15.38	30.85	85.23
MNM	Mono-n-methyl phthalate	49.2	1.31 (0.03)	<LOD	<LOD	<LOD	2.59	11.71
*HWM			0.34 (0.01)	0.06	0.16	0.32	0.66	2.60
MBzP	Mono-benzyl phthalate	98.4	5.95 (0.17)	0.57	2.60	6.40	14.65	47.34
MCHP	Mono-cyclohexyl phthalate	4.4	0.36 (0.00)	<LOD	<LOD	<LOD	<LOD	<LOD
MCNP	Mono-(carboxynonyl) phthalate	94.0	2.53 (0.06)	0.41	1.20	2.49	4.89	17.21
MCOP	monocarboxyoctyl phthalte	97.5	9.36 (0.37)	0.96	3.49	8.00	22.95	126.89
MCPP	Mono-(3-carboxypropyl) phthalate	97.3	2.47 (0.07)	0.30	1.19	2.49	4.99	18.14
MiNP	Mono-isononyl phthalate	24.0	1.08 (0.02)	<LOD	<LOD	<LOD	<LOD	7.98
MOP	Mono-n-octyl phthalate	1.2	1.05 (0.01)	<LOD	<LOD	<LOD	<LOD	<LOD
∑DEHP	Di-2-ethylhexyl phthalate		68.72 (1.20)	9.52	29.31	64.31	142.28	672.68
MECPP	Mono-(2-ethyl-5-carboxypentyl) phthalate	99.9	23.62 (0.68)	3.38	10.39	22.40	48.20	217.41
MEHHP	Mono-(2-ethyl-5-hydroxyhexyl) phthalate	69.2	2.00 (0.06)	<LOD	<LOD	1.70	4.20	22.50
MEHP	Mono-2-ethylhexyl phthalate	99.5	15.15 (0.45)	1.82	6.30	14.40	32.69	167.79
MEOHP	Mono-(2-ethyl-5-oxohexyl) phthalate	98.9	9.34 (0.28)	1.10	4.02	8.98	20.06	97.85

*Unit of LMWP and HMWP is mmol/L urine; GM: geometric mean; GSE: geometric standard error of mean.

Table 3-1. Urinary phthalate metabolites concentration (ug/L), adult female

Metabolites		> LOD (%)	GM (GSE)	Percentile				
				5th	25th	50th	75th	95th
*LMW			1.64 (0.05)	0.22	0.74	1.72	3.63	11.63
MEP	Mono-ethyl phthalate	99.9	79.93 (3.00)	6.86	25.55	76.55	222.88	1214.90
MiBP	Mono-isobutyl phthalate	97.6	4.85 (0.136)	0.50	2.28	5.39	11.39	29.84
MnBP	Mono-n-butyl phthalate	98.1	13.69 (0.45)	1.29	6.39	15.22	32.02	101.79
MNM	Mono-n-methyl phthalate	41.3	1.25 (0.04)	<LOD	<LOD	<LOD	2.37	11.47
*HWM			0.31 (0.01)	0.05	0.14	0.29	0.59	2.08
MBzP	Mono-benzyl phthalate	98.0	5.546 (0.19)	0.49	2.30	5.97	14.31	47.53
MCHP	Mono-cyclohexyl phthalate	4.3	0.36 (0.01)	<LOD	<LOD	<LOD	<LOD	<LOD
MCNP	Mono-(carboxynonyl) phthalate	92.4	2.19 (0.07)	0.35	1.00	2.12	4.31	14.78
MCOP	monocarboxyoctyl phthalte	96.8	8.16 (0.35)	0.79	3.17	7.20	20.58	99.79
MCPP	Mono-(3-carboxypropyl) phthalate	96.7	2.18 (0.06)	0.29	1.00	2.19	4.60	15.27
MiNP	Mono-isononyl phthalate	21.9	1.01 (0.02)	<LOD	<LOD	<LOD	<LOD	5.43
MOP	Mono-n-octyl phthalate	1.1	1.05 (0.01)	<LOD	<LOD	<LOD	<LOD	<LOD
∑DEHP	Di-2-ethylhexyl phthalate		61.56 (2.07)	8.90	26.18	58.72	133.49	530.81
MECPP	Mono-(2-ethyl-5-carboxypentyl) phthalate	99.9	21.33 (0.71)	3.02	9.37	20.46	44.98	173.83
MEHHP	Mono-(2-ethyl-5-hydroxyhexyl) phthalate	66.0	1.78 (0.06)	0.35	0.77	1.42	3.78	16.99
MEHP	Mono-2-ethylhexyl phthalate	99.5	13.31 (0.47)	1.59	5.52	12.87	30.49	132.57
MEOHP	Mono-(2-ethyl-5-oxohexyl) phthalate	98.8	8.43 (0.30)	1.00	3.59	8.29	19.07	78.87

*Unit of LMWP and HMWP is mmol/L urine; GM: geometric mean; GSE: geometric standard error of mean.

Table 3-2. Urinary phthalate metabolites concentration (ug/L), adult male

Metabolites		> LOD (%)	GM (GSE)	Percentile				
				5th	25th	50th	75th	95th
*LMW			1.76 (0.05)	0.28	0.89	1.75	3.52	11.28
MEP	Mono-ethyl phthalate	99.9	83.04 (3.12)	7.61	27.22	73.87	234.30	1419.83
MiBP	Mono-isobutyl phthalate	98.4	5.72 (0.17)	0.68	2.99	6.13	11.88	32.17
MnBP	Mono-n-butyl phthalate	98.2	13.89 (0.46)	1.69	7.59	15.48	29.23	69.17
MNM	Mono-n-methyl phthalate	51.1	1.39 (0.04)	<LOD	<LOD	0.78	2.79	12.00
*HWM			0.38 (0.01)	0.06	0.18	0.35	0.70	3.25
MBzP	Mono-benzyl phthalate	98.8	6.43 (0.21)	0.64	2.98	6.98	15.01	47.20
MCHP	Mono-cyclohexyl phthalate	4.6	0.36 (0.00)	<LOD	<LOD	<LOD	<LOD	<LOD
MCNP	Mono-(carboxynonyl) phthalate	95.7	2.93 (0.09)	0.42	1.49	2.79	5.59	19.34
MCOP	monocarboxyoctyl phthalte	98.2	10.83 (0.50)	1.18	3.89	9.29	26.86	156.47
MCPP	Mono-(3-carboxypropyl) phthalate	98.0	2.83 (0.105)	0.39	1.39	2.69	5.51	22.01
MiNP	Mono-isononyl phthalate	26.3	1.16 (0.02)	<LOD	<LOD	<LOD	<LOD	9.40
MOP	Mono-n-octyl phthalate	1.4	1.05 (0.01)	<LOD	<LOD	<LOD	<LOD	<LOD
∑DEHP	Di-2-ethylhexyl phthalate		77.29 (2.63)	10.61	34.26	69.89	150.10	855.92
MECPP	Mono-(2-ethyl-5-carboxypentyl) phthalate	99.8	26.35 (0.90)	3.89	11.80	23.94	51.30	284.57
MEHHP	Mono-(2-ethyl-5-hydroxyhexyl) phthalate	72.5	2.28 (0.08)	0.35	0.81	1.99	4.79	29.11
MEHP	Mono-2-ethylhexyl phthalate	99.6	17.40 (0.62)	2.10	7.38	16.09	36.65	223.31
MEOHP	Mono-(2-ethyl-5-oxohexyl) phthalate	99.1	10.42 (0.37)	1.26	4.59	9.51	21.20	125.34

*Unit of LMWP and HMWP is mmol/L urine; GM: geometric mean; GSE: geometric standard error of mean.

Table 4. Adjusted odds ratio of having asthma outcome related to phthalate exposure, overall adult participants; NHANES 2003-2012

Metabolites	Ever asthma (n=965)	Current asthma (n=576)	Past asthma (n=389)	Asthma attack past year (n=264)	Wheeze past year (n=199)
LMW					
MEP	0.94 (0.81, 1.08)	1.08 (0.82, 1.42)	0.94 (0.72, 1.23)	0.51 (0.84, 1.61)	0.99 (0.70, 1.41)
MiBP	0.98 (0.78, 1.23)	0.70 (0.46, 1.07)	1.41 (0.94, 2.13)	1.19 (0.52, 1.22)	0.95 (0.52, 1.75)
MnBP	0.94 (0.78, 1.14)	0.80 (0.54, 1.20)	1.25 (0.84, 1.84)	0.99 (0.42, 1.00)	1.40 (0.81, 2.43)
MNM	1.25 (1.04, 1.50)	1.02 (0.72, 1.44)	1.03 (0.72, 1.48)	0.75 (0.54, 1.20)	0.96 (0.75, 1.23)
HWM					
MBzP	1.14 (0.95, 1.38)	1.13 (0.76, 1.66)	0.88 (0.61, 1.29)	0.68 (0.59, 1.50)	1.10 (0.65, 1.86)
MCNP	1.13 (0.94, 1.35)	0.83 (0.54, 1.27)	1.19 (0.78, 1.82)	1.04 (0.50, 1.24)	1.04 (0.72, 1.51)
MCOP	1.10 (0.94, 1.29)	1.04 (0.77, 1.40)	0.96 (0.72, 1.28)	0.56 (0.58, 1.43)	1.05 (0.70, 1.58)
MCPP	1.18 (0.97, 1.42)	1.12 (0.79, 1.58)	0.90 (0.64, 1.27)	0.63 (0.59, 1.41)	1.27 (0.83, 1.96)
∑DEHP	1.02 (0.85, 1.22)	0.98 (0.69, 1.39)	1.07 (0.76, 1.52)	0.76 (0.50, 1.42)	1.06 (0.58, 1.93)
MECPP	0.98 (0.81, 1.19)	0.95 (0.67, 1.35)	1.10 (0.78, 1.55)	0.77 (0.53, 1.44)	1.13 (0.67, 1.91)
MEHHP	1.06 (0.89, 1.26)	1.02 (0.74, 1.40)	1.01 (0.74, 1.39)	0.65 (0.69, 1.77)	0.94 (0.59, 1.49)
MEHP	1.04 (0.89, 1.22)	0.95 (0.69, 1.33)	1.10 (0.79, 1.52)	0.73 (0.51, 1.31)	1.04 (0.59, 1.81)
MEOHP	1.01 (0.85, 1.20)	0.98 (0.69, 1.39)	1.06 (0.75, 1.50)	0.75 (0.48, 1.34)	1.02 (0.52, 1.97)

* Adjusted ORs (95%CIs) were reported for 1-log10 increase in urinary phthalate concentration. All models were adjusted for age, BMI, PIR, urinary creatinine, and serum cotinine. n= the number of participants reporting ever asthma, current asthma, past asthma, asthma attack in the past year, and wheezing in the past year. ORs with n less than 50 were not reported in our results, but were still displayed in the tables.

Table 5. Adjusted odds ratio of having asthma outcome related to phthalate exposure, adult female; NHANES 2003-2012

Metabolites	Ever asthma (n=555)	Current asthma (n=352)	Past asthma (n=203)	Asthma attack past year (n=180)	Wheeze past year (n=102)
LMW					
MEP	0.85 (0.68, 1.05)	1.15 (0.76, 1.75)	0.89 (0.60, 1.33)	1.19 (0.78, 1.80)	1.05 (0.69, 1.62)
MiBP	1.02 (0.74, 1.39)	0.52 (0.30, 0.90)	1.83 (1.10, 3.04)	0.84 (0.42, 1.70)	0.67 (0.25, 1.80)
MnBP	0.85 (0.65, 1.13)	0.71 (0.41, 1.23)	1.36 (0.81, 2.29)	0.70 (0.38, 1.31)	0.99 (0.49, 1.98)
MNM	1.25 (0.98, 1.59)	1.17 (0.72, 1.90)	0.92 (0.56, 1.52)	0.82 (0.48, 1.41)	0.99 (0.70, 1.42)
HWM					
MBzP	1.03 (0.81, 1.32)	1.16 (0.69, 1.94)	0.84 (0.51, 1.38)	0.96 (0.53, 1.74)	1.03 (0.48, 2.20)
MCNP	1.26 (1.00, 1.57)	0.67 (0.35, 1.30)	1.50 (0.76, 2.77)	0.83 (0.46, 1.50)	0.93 (0.56, 1.54)
MCOP	1.29 (1.03, 1.61)	0.87 (0.55, 1.36)	1.13 (0.73, 1.74)	1.05 (0.62, 1.78)	0.83 (0.50, 1.38)
MCPP	1.34 (1.08, 1.65)	1.03 (0.65, 1.64)	0.98 (0.62, 1.55)	0.95 (0.49, 1.84)	1.18 (0.75, 1.83)
∑DEHP	1.09 (0.86, 1.39)	0.91 (0.58, 1.42)	1.15 (0.74, 1.79)	0.72 (0.38, 1.37)	0.86 (0.34, 2.16)
MECPP	1.05 (0.82, 1.33)	0.90 (0.58, 1.39)	1.17 (0.76, 1.80)	0.77 (0.42, 1.42)	1.06 (0.52, 2.14)
MEHHP	1.15 (0.94, 1.41)	0.80 (0.52, 1.22)	1.28 (0.84, 1.96)	1.08 (0.62, 1.90)	0.72 (0.34, 1.52)
MEHP	1.12 (0.90, 1.38)	0.90 (0.59, 1.35)	1.16 (0.77, 1.76)	0.70 (0.39, 1.26)	0.83 (0.35, 1.94)
MEOHP	1.08 (0.85, 1.38)	0.90 (0.58, 1.39)	1.14 (0.74, 1.74)	0.64 (0.35, 1.19)	0.75 (0.28, 2.02)

* Adjusted ORs (95%CIs) were reported for 1-log10 increase in urinary phthalate concentration. All models were adjusted for age, BMI, PIR, urinary creatinine, and serum cotinine. n= the number of participants reporting ever asthma, current asthma, past asthma, asthma attack in the past year, and wheezing in the past year. ORs with n less than 50 were not reported in our results, but were still displayed in the tables.

Table 6. Adjusted odds ratio of having asthma outcome related to phthalate exposure, adult male; NHANES 2003-2012

Metabolites	Ever asthma (n=410)	Current asthma (n=224)	Past asthma (n=186)	Asthma attack past year (n=84)	Wheeze past year (n=97)
LMW					
MEP	1.00 (0.82, 1.22)	0.87 (0.57, 1.32)	1.16 (0.77, 1.74)	1.01 (0.54, 1.89)	0.90 (0.55, 1.47)
MiBP	0.84 (0.61, 1.16)	0.79 (0.40, 1.54)	1.28 (0.66, 2.47)	0.66 (0.28, 1.52)	1.36 (0.68, 2.70)
MnBP	0.90 (0.64, 1.25)	0.67 (0.39, 1.16)	1.50 (0.88, 2.58)	0.41 (0.18, 0.95)	2.08 (1.01, 4.29)
MNM	1.22 (0.93, 1.59)	0.74 (0.40, 1.37)	1.38 (0.76, 2.52)	0.60 (0.26, 1.38)	0.91 (0.62, 1.34)
HWM					
MBzP	1.17 (0.84, 1.62)	0.85 (0.49, 1.48)	1.18 (0.68, 2.04)	0.76 (0.35, 1.65)	1.02 (0.53, 1.96)
MCNP	0.95 (0.66, 1.36)	1.03 (0.56, 1.89)	0.99 (0.54, 1.81)	0.65 (0.24, 1.75)	1.43 (0.54, 3.74)
MCOP	0.89 (0.68, 1.17)	1.22 (0.81, 1.85)	0.84 (0.55, 1.28)	0.73 (0.33, 1.59)	1.52 (0.74, 3.12)
MCPP	0.97 (0.71, 1.34)	1.16 (0.73, 1.84)	0.86 (0.54, 1.36)	0.82 (0.41, 1.65)	1.47 (0.76, 2.82)
∑DEHP	0.89 (0.68, 1.17)	0.95 (0.53, 1.73)	1.10 (0.61, 1.99)	0.98 (0.39, 2.43)	1.34 (0.54, 3.32)
MECPP	0.86 (0.64, 1.17)	0.91 (0.52, 1.58)	1.14 (0.66, 1.98)	0.97 (0.40, 2.35)	1.29 (0.51, 3.30)
MEHHP	0.94 (0.75, 1.19)	1.39 (0.79, 2.43)	0.74 (0.42, 1.28)	1.13 (0.52, 2.44)	1.27 (0.77, 2.08)
MEHP	0.92 (0.73, 1.16)	0.94 (0.53, 1.69)	1.11 (0.62, 1.99)	0.98 (0.43, 2.23)	1.30 (0.57, 2.94)
MEOHP	0.88 (0.69, 1.13)	0.95 (0.52, 1.73)	1.11 (0.61, 2.01)	0.99 (0.42, 2.31)	1.34 (0.55, 3.26)

* Adjusted ORs (95%CIs) were reported for 1-log10 increase in urinary phthalate concentration. All models were adjusted for age, BMI, PIR, urinary creatinine, and serum cotinine. n= the number of participants reporting ever asthma, current asthma, past asthma, asthma attack in the past year, and wheezing in the past year. ORs with n less than 50 were not reported in our results, but were still displayed in the tables.

Table 7. Adjusted odds ratio of having asthma outcome related to phthalate exposure, adult black female; NHANES 2003-2012

Metabolites	Ever asthma (n=113)	Current asthma (n=64)	Past asthma (n=49)	Asthma attack past year (n=25)	Wheeze past year (n=27)
LMW					
MEP	0.78 (0.57, 1.06)	1.37 (0.70, 2.68)	0.81 (0.44, 1.49)	1.26 (0.61, 2.61)	1.80 (0.86, 3.79)
MiBP	0.92 (0.50, 1.65)	2.21 (0.74, 6.62)	0.44 (0.16, 1.20)	0.92 (0.28, 3.05)	2.78 (1.06, 7.28)
MnBP	1.06 (0.59, 1.90)	1.20 (0.41, 3.49)	0.66 (0.25, 1.77)	1.44 (0.43, 4.85)	3.51 (0.96, 12.79)
MNM	1.00 (0.53, 1.89)	1.03 (0.41, 2.59)	0.92 (0.36, 2.32)	1.29 (0.49, 3.42)	1.79 (0.78, 4.10)
HWM					
MBzP	1.33 (0.86, 2.04)	1.82 (0.79, 4.21)	0.46 (0.21, 1.00)	3.55 (1.48, 8.53)	12.27 (6.41, 23.51)
MCNP	1.03 (0.68, 1.57)	1.21 (0.38, 3.83)	1.05 (0.36, 3.10)	1.95 (0.54, 7.06)	1.32 (0.48, 3.64)
MCOP	1.29 (0.85, 1.96)	1.92 (0.84, 4.38)	0.70 (0.33, 1.51)	1.59 (0.59, 4.29)	2.22 (1.10, 4.50)
MCPP	1.55 (1.08, 2.21)	1.71 (0.54, 5.46)	0.73 (0.25, 2.11)	1.07 (0.37, 3.13)	2.22 (1.42, 3.46)
∑DEHP	1.40 (0.96, 2.05)	1.68 (0.75, 3.76)	0.73 (0.35, 1.50)	0.80 (0.27, 2.39)	6.87 (1.83, 25.80)
MECPP	1.36 (0.93, 2.00)	1.52 (0.68, 3.42)	0.82 (0.39, 1.71)	0.77 (0.27, 2.22)	6.60 (1.62, 26.84)
MEHHP	1.25 (0.86, 1.82)	1.55 (0.73, 3.29)	0.76 (0.36, 1.59)	1.22 (0.48, 3.07)	2.26 (0.61, 8.40)
MEHP	1.44 (1.02, 2.05)	1.68 (0.78, 3.62)	0.71 (0.36, 1.39)	0.82 (0.29, 2.34)	4.87 (1.43, 16.58)
MEOHP	1.40 (0.97, 2.02)	1.66 (0.74, 3.75)	0.72 (0.35, 1.49)	0.80 (0.25, 2.51)	5.47 (1.38, 21.73)

* Adjusted ORs (95%CIs) were reported for 1-log10 increase in urinary phthalate concentration. All models were adjusted for age, BMI, PIR, urinary creatinine, and serum cotinine. n= the number of participants reporting ever asthma, current asthma, past asthma, asthma attack in the past year, and wheezing in the past year. ORs with n less than 50 were not reported in our results, but were still displayed in the tables.

Table 8. Adjusted odds ratio of having asthma outcome related to phthalate exposure, adult Hispanic female; NHANES 2003-2012

Metabolites	Ever asthma (n=118)	Current asthma (n=72)	Past asthma (n=46)	Asthma attack past year (n=40)	Wheeze past year (n=20)
LMW					
MEP	1.14 (0.78, 1.66)	1.57 (0.76, 3.23)	0.63 (0.31, 1.30)	1.07 (0.51, 2.24)	0.78 (0.38, 1.60)
MiBP	1.16 (0.68, 1.98)	0.84 (0.31, 2.24)	1.19 (0.45, 3.19)	0.52 (0.12, 2.31)	1.80 (0.30, 10.98)
MnBP	0.93 (0.51, 1.68)	0.45 (0.16, 1.28)	2.21 (0.77, 6.30)	0.21 (0.04, 1.09)	1.48 (0.37, 5.92)
MNM	1.46 (0.80, 2.66)	1.51 (0.84, 2.70)	0.66 (0.37, 1.18)	0.35 (0.09, 1.28)	2.26 (0.60, 8.48)
HWM					
MBzP	0.98 (0.54, 1.79)	0.89 (0.32, 2.43)	1.10 (0.40, 3.00)	0.72 (0.20, 2.57)	3.81 (0.65, 22.27)
MCNP	0.84 (0.45, 1.57)	0.56 (0.15, 2.05)	1.73 (0.47, 6.31)	3.05 (0.34, 27.43)	0.42 (0.08, 2.29)
MCOP	1.49 (0.93, 2.39)	0.60 (0.25, 1.48)	1.62 (0.67, 3.94)	7.68 (1.85, 31.85)	1.31 (0.53, 3.23)
MCPP	1.41 (0.70, 2.82)	1.28 (0.44, 3.73)	0.77 (0.27, 2.23)	2.60 (0.53, 12.72)	1.34 (0.62, 2.92)
∑DEHP	0.93 (0.43, 2.02)	0.64 (0.24, 1.71)	1.52 (0.57, 4.03)	0.18 (0.03, 0.94)	1.99 (0.30, 13.41)
MECPP	0.88 (0.40, 1.92)	0.52 (0.20, 1.35)	1.89 (0.73, 4.93)	0.36 (0.08, 1.57)	1.94 (0.33, 11.52)
MEHHP	0.90 (0.52, 1.59)	0.88 (0.34, 2.28)	1.13 (0.44, 2.90)	0.18 (0.04, 0.75)	1.56 (0.40, 6.02)
MEHP	0.98 (0.50, 1.94)	0.76 (0.29, 1.97)	1.28 (0.50, 3.30)	0.14 (0.03, 0.71)	1.63 (0.23, 11.67)
MEOHP	0.98 (0.48, 2.01)	0.73 (0.29, 1.87)	1.33 (0.52, 3.38)	0.17 (0.03, 0.81)	1.65 (0.20, 13.87)

* Adjusted ORs (95%CIs) were reported for 1-log10 increase in urinary phthalate concentration. All models were adjusted for age, BMI, PIR, urinary creatinine, and serum cotinine. n= the number of participants reporting ever asthma, current asthma, past asthma, asthma attack in the past year, and wheezing in the past year. ORs with n less than 50 were not reported in our results, but were still displayed in the tables.

Table 9. Adjusted odds ratio of having asthma outcome related to phthalate exposure, adult white female; NHANES 2003-2012

Metabolites	Ever asthma (n=271)	Current asthma (n=173)	Past asthma (n=98)	Asthma attack past year (n=88)	Wheeze past year (n=45)
LMW					
MEP	0.83 (0.62, 1.11)	1.03 (0.60, 1.77)	0.98 (0.58, 1.64)	1.33 (0.72, 2.46)	1.15 (0.60, 2.23)
MiBP	1.07 (0.71, 1.61)	0.42 (0.20, 0.90)	2.29 (1.11, 4.71)	0.80 (0.31, 2.03)	0.45 (0.09, 2.24)
MnBP	0.89 (0.62, 1.29)	0.61 (0.28, 1.35)	1.71 (0.78, 3.73)	0.79 (0.31, 2.00)	0.75 (0.30, 1.88)
MNM	1.27 (0.93, 1.74)	1.11 (0.60, 2.06)	0.97 (0.52, 1.81)	0.88 (0.45, 1.72)	0.73 (0.34, 1.55)
HWM					
MBzP	1.01 (0.74, 1.40)	1.16 (0.57, 2.36)	0.90 (0.45, 1.81)	0.86 (0.37, 2.03)	0.68 (0.23, 2.00)
MCNP	1.35 (1.00, 1.82)	0.58 (0.25, 1.38)	1.61 (0.70, 3.72)	0.57 (0.23, 1.42)	0.87 (0.47, 1.64)
MCOP	1.18 (0.89, 1.55)	0.88 (0.49, 1.59)	1.07 (0.61, 1.87)	0.79 (0.37, 1.67)	0.66 (0.34, 1.26)
MCPP	1.21 (0.92, 1.59)	0.90 (0.47, 1.73)	1.06 (0.56, 1.99)	0.79 (0.31, 2.03)	1.06 (0.55, 2.03)
∑DEHP	1.08 (0.76, 1.52)	0.92 (0.51, 1.67)	1.09 (0.62, 1.95)	0.96 (0.42, 2.17)	0.35 (0.10, 1.25)
MECPP	1.03 (0.73, 1.44)	0.96 (0.54, 1.73)	1.04 (0.59, 1.84)	0.97 (0.45, 2.07)	0.54 (0.21, 1.40)
MEHHP	1.20 (0.89, 1.61)	0.75 (0.42, 1.32)	1.30 (0.73, 2.32)	1.47 (0.67, 3.20)	0.320 (0.09, 1.09)
MEHP	1.10 (0.80, 1.50)	0.87 (0.50, 1.52)	1.15 (0.67, 1.99)	0.92 (0.43, 1.95)	0.40 (0.13, 1.22)
MEOHP	1.08 (0.76, 1.53)	0.90 (0.51, 1.56)	1.13 (0.66, 1.94)	0.85 (0.37, 1.92)	0.38 (0.09, 1.57)

* Adjusted ORs (95%CIs) were reported for 1-log10 increase in urinary phthalate concentration. All models were adjusted for age, BMI, PIR, urinary creatinine, and serum cotinine. n= the number of participants reporting ever asthma, current asthma, past asthma, asthma attack in the past year, and wheezing in the past year. ORs with n less than 50 were not reported in our results, but were still displayed in the tables.

Table 10. Adjusted odds ratio of having asthma outcome related to phthalate exposure, adult black male; NHANES 2003-2012

Metabolites	Ever asthma (n=113)	Current asthma (n=64)	Past asthma (n=49)	Asthma attack past year (n=25)	Wheeze past year (n=27)
LMW					
MEP	0.83 (0.59, 1.18)	1.48 (0.60, 3.61)	0.67 (0.27, 1.64)	0.60 (0.24, 1.55)	0.82 (0.38, 1.78)
MiBP	1.0 (0.61, 1.80)	2.32 (0.70, 7.65)	0.42 (0.12, 1.39)	1.18 (0.37, 3.80)	1.27 (0.54, 2.94)
MnBP	1.26 (0.65, 2.43)	2.46 (0.69, 8.77)	0.39 (0.10, 1.47)	0.73 (0.24, 2.18)	0.83 (0.27, 2.59)
MNM	0.96 (0.62, 1.49)	2.22 (0.72, 6.91)	0.51 (0.18, 1.44)	0.21 (0.05, 0.96)	1.63 (0.86, 3.09)
HWM					
MBzP	1.29 (0.86, 1.94)	1.73 (0.63, 4.77)	0.53 (0.19, 1.49)	0.87 (0.26, 2.90)	1.24 (0.47, 3.31)
MCNP	1.06 (0.55, 2.02)	1.75 (0.53, 5.79)	0.61 (0.18, 2.05)	0.60 (0.10, 3.64)	0.68 (0.23, 2.00)
MCOP	1.00 (0.66, 1.54)	0.75 (0.32, 1.79)	1.39 (0.58, 3.30)	0.29 (0.08, 1.03)	0.96 (0.43, 2.12)
MCPP	0.81 (0.47, 1.38)	1.20 (0.46, 3.10)	0.80 (0.31, 2.03)	0.37 (0.10, 1.29)	1.00 (0.31, 3.27)
∑DEHP	0.98 (0.65, 1.46)	0.90 (0.31, 2.56)	1.16 (0.42, 3.22)	0.68 (0.16, 2.90)	2.10 (0.46, 9.65)
MECPP	0.96 (0.61, 1.49)	0.92 (0.39, 2.16)	1.14 (0.50, 2.59)	0.59 (0.15, 2.31)	1.95 (0.44, 8.67)
MEHHP	0.87 (0.62, 1.22)	1.38 (0.52, 3.64)	0.74 (0.28, 1.95)	0.70 (0.18, 2.83)	0.86 (0.23, 3.28)
MEHP	0.98 (0.68, 140.)	0.91 (0.33, 2.55)	1.12 (0.41, 3.09)	0.78 (0.19, 3.17)	2.24 (0.60, 8.33)
MEOHP	0.95 (0.65, 1.41)	0.99 (0.35, 2.84)	1.06 (0.39, 2.91)	0.64 (0.17, 2.45)	2.07 (0.49, 8.79)

* Adjusted ORs (95%CIs) were reported for 1-log10 increase in urinary phthalate concentration. All models were adjusted for age, BMI, PIR, urinary creatinine, and serum cotinine. n= the number of participants reporting ever asthma, current asthma, past asthma, asthma attack in the past year, and wheezing in the past year. ORs with n less than 50 were not reported in our results, but were still displayed in the tables.

Table 11. Adjusted odds ratio of having asthma outcome related to phthalate exposure, adult Hispanic male; NHANES 2003-2012

Metabolites	Ever asthma (n=58)	Current asthma (n=27)	Past asthma (n=31)	Asthma attack past year (n=11)	Wheeze past year (n=13)
LMW					
MEP	0.96 (0.59, 1.56)	1.02 (0.36, 2.88)	0.98 (0.35, 2.78)	0.86 (0.16, 4.77)	1.46 (0.37, 5.68)
MiBP	1.02 (0.46, 2.27)	1.20 (0.12, 9.85)	0.91 (0.10, 8.15)	9.14 (0.59, 142.08)	1.37 (0.25, 7.61)
MnBP	1.03 (0.56, 1.92)	0.86 (0.13, 5.50)	1.18 (0.18, 7.66)	0.46 (0.06, 3.47)	0.76 (0.08, 6.97)
MNM	0.81 (0.47, 1.39)	0.89 (0.19, 4.16)	1.12 (0.24, 5.22)	0.40 (0.03, 5.17)	0.89 (0.38, 2.06)
HWM					
MBzP	1.21 (0.61, 2.43)	0.86 (0.24, 3.10)	1.16 (0.32, 4.17)	0.09 (0.01, 1.87)	1.57 (0.16, 15.79)
MCNP	0.77 (0.30, 1.98)	2.33 (0.59, 9.27)	0.43 (0.11, 1.71)	0.83 (0.07, 10.69)	0.92 (0.13, 6.50)
MCOP	0.99 (0.51, 1.91)	2.82 (1.04, 7.63)	0.35 (0.13, 0.96)	2.98 (0.14, 62.61)	1.71 (0.49, 6.00)
MCPP	0.99 (0.56, 1.74)	2.14 (0.84, 5.46)	0.47 (0.18, 1.20)	0.73 (0.09, 5.92)	1.40 (0.56, 3.51)
∑DEHP	0.90 (0.56, 1.45)	6.17 (1.09, 34.83)	0.16 (0.03, 0.92)	0.48 (0.11, 2.12)	1.15 (0.36, 3.71)
MECPP	0.78 (0.46, 1.31)	5.39 (1.05, 27.76)	0.19 (0.04, 0.96)	0.45 (0.09, 2.20)	1.14 (0.37, 3.53)
MEHHP	0.98 (0.59, 1.62)	4.54 (1.14, 18.12)	0.22 (0.06, 0.88)	0.70 (0.19, 2.60)	0.96 (0.29, 3.15)
MEHP	1.01 (0.67, 1.53)	5.24 (1.16, 23.65)	0.19 (0.04, 0.86)	0.55 (0.15, 2.02)	1.20 (0.41, 3.54)
MEOHP	1.04 (0.67, 1.63)	6.81 (1.10, 42.04)	0.15 (0.02, 0.91)	0.45 (0.10, 2.13)	1.29 (0.42, 3.90)

* Adjusted ORs (95%CIs) were reported for 1-log10 increase in urinary phthalate concentration. All models were adjusted for age, BMI, PIR, urinary creatinine, and serum cotinine. n= the number of participants reporting ever asthma, current asthma, past asthma, asthma attack in the past year, and wheezing in the past year. ORs with n less than 50 were not reported in our results, but were still displayed in the tables.

Table 12. Adjusted odds ratio of having asthma outcome related to phthalate exposure, adult white male; NHANES 2003-2012

Metabolites	Ever asthma (n=210)	Current asthma (n=123)	Past asthma (n=87)	Asthma attack past year (n=44)	Wheeze past year (n=47)
LMW					
MEP	1.02 (0.79, 1.33)	0.85 (0.50, 1.44)	1.19 (0.71, 1.99)	0.99 (0.45, 2.15)	0.98 (0.47, 2.03)
MiBP	0.82 (0.53, 1.27)	0.75 (0.32, 1.75)	1.36 (0.59, 3.12)	0.45 (0.17, 1.18)	1.80 (0.68, 4.77)
MnBP	0.82 (0.54, 1.25)	0.58 (0.26, 1.29)	1.76 (0.79, 3.89)	0.29 (0.10, 0.83)	2.72 (1.15, 6.45)
MNM	1.39 (0.98, 1.98)	0.61 (0.29, 1.30)	1.68 (0.81, 3.50)	0.70 (0.26, 1.89)	0.91 (0.49, 1.71)
HWM					
MBzP	1.06 (0.70, 1.61)	0.77 (0.37, 1.62)	1.32 (0.63, 2.75)	0.62 (0.23, 1.65)	1.04 (0.43, 2.50)
MCNP	0.90 (0.58, 1.40)	0.93 (0.39, 2.18)	1.07 (0.46, 2.53)	0.55 (0.11, 2.81)	1.35 (0.40, 4.54)
MCOP	0.90 (0.62, 1.29)	1.25 (0.71, 2.23)	0.81 (0.45, 1.47)	0.69 (0.24, 1.99)	1.54 (0.67, 3.57)
MCPP	1.00 (0.65, 1.54)	1.03 (0.55, 1.91)	0.97 (0.52, 1.80)	1.03 (0.41, 2.56)	1.41 (0.68, 2.91)
∑DEHP	0.87 (0.60, 1.25)	0.71 (0.30, 1.70)	1.48 (0.62, 3.51)	1.00 (0.28, 3.57)	1.40 (0.38, 5.13)
MECPP	0.88 (0.58, 1.30)	0.67 (0.31, 1.49)	1.55 (0.70, 3.43)	1.09 (0.31, 3.85)	1.38 (0.36, 5.32)
MEHHP	0.97 (0.71, 1.32)	1.11 (0.50, 2.45)	0.93 (0.43, 2.02)	1.11 (0.41, 3.04)	1.78 (0.88, 3.58)
MEHP	0.88 (0.64, 1.20)	0.74 (0.31, 1.72)	1.44 (0.62, 3.35)	0.91 (0.29, 2.84)	1.23 (0.38, 3.90)
MEOHP	0.84 (0.60, 1.16)	0.72 (0.29, 1.77)	1.47 (0.60, 3.60)	0.98 (0.30, 3.21)	1.44 (0.41, 5.02)

* Adjusted ORs (95%CIs) were reported for 1-log10 increase in urinary phthalate concentration. All models were adjusted for age, BMI, PIR, urinary creatinine, and serum cotinine. n= the number of participants reporting ever asthma, current asthma, past asthma, asthma attack in the past year, and wheezing in the past year. ORs with n less than 50 were not reported in our results, but were still displayed in the tables.