

Report to the
U.S. Consumer Product Safety Commission
by the
CHRONIC HAZARD ADVISORY PANEL ON PHTHALATES
AND PHTHALATE ALTERNATIVES

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APPENDIX E1
MODELING CONSUMER EXPOSURE TO
PHTHALATE ESTERS



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CONSUMER PRODUCT SAFETY COMMISSION
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Memorandum

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SUBJECT : Modeling consumer exposure to phthalate esters (PEs)^{*†}

The attached report provides the U.S. Consumer Product Safety Commission's (CPSC's) Health Sciences' staff assessment of consumer exposures to phthalate esters from all sources and routes of exposure, including diet, teething and toys, child care articles, and personal care products. This work was performed at the request of the Chronic Hazard Advisory Panel (CHAP) on phthalates and phthalate substitutes.

* These comments are those of the CPSC staff, have not been reviewed or approved by, and may not necessarily reflect the views of, the Commission.

† Leslie E. Patton, Ph.D., Toxicologist, who is no longer with CPSC, contributed to this report.

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ABBREVIATIONS*

3 β -HSD	3 β -hydroxysteroid dehydrogenase
AA	antiandrogenicity; antiandrogenic
ADHD	attention deficit hyperactivity disorder
ADI	acceptable daily intake
AGD	anogenital distance
AGI	anogenital index
ASD	Autistic Spectrum Disorders
ASTDR	Agency for Toxic Substances and Disease Registry
ATBC	acetyl tributyl citrate
BASC-PRS	Behavior Assessment System for Children-Parent Rating Scales
BBP	butylbenzyl phthalate
BIBRA	British Industrial Biological Research Association
BMD	benchmark dose
BMDL	benchmark dose (lower confidence limit)
BNBA	Brazelton Neonatal Behavioral Assessment
BRIEF	Behavior Rating Inventory of Executive Function
BSI	behavioral symptoms index
CBCL	Child Behavior Check List
CDC	Centers for Disease Control and Prevention, U.S.
CERHR	Center for the Evaluation of Risks to Human Reproduction
CF	consumption factor
CHAP	Chronic Hazard Advisory Panel
CHO	Chinese hamster ovary
CNS	central nervous system
CPSC	Consumer Product Safety Commission, U.S.
CPSIA	Consumer Product Safety Improvement Act of 2008
CRA	cumulative risk assessment
CSL	cranial suspensory ligament
cx-MIDP	mono(carboxy-isononyl) phthalate (also, CNP, MCNP)
cx-MINP	mono(carboxy-isoctyl) phthalate (also COP, MCOP)
DAP	diallyl phthalate
DBP	dibutyl phthalate
DCHP	dicyclohexyl phthalate
DDP	di- <i>n</i> -decyl phthalate
DEHA	di(2-ethylhexyl) adipate
DEHP	di(2-ethylhexyl) phthalate
DEHT	di(2-ethylhexyl) terephthalate
DEP	diethyl phthalate
DHEPP	di- <i>n</i> -heptyl phthalate
DHEXP	di- <i>n</i> -hexyl phthalate
DHT	dihydrotestosterone

* List applies to main report and all appendices.

DI	daily intake
DIBP	diisobutyl phthalate
DIDP	diisodecyl phthalate
DIHEPP	diisoheptyl phthalate
DIHEXP	diisoheptyl phthalate
DINP	diisononyl phthalate
DINCH [®]	1,2-cyclohexanedicarboxylic acid, diisononyl ester
DINX	1,2-cyclohexanedicarboxylic acid, diisononyl ester
DIOP	diisooctyl phthalate
DIPP	diisopropyl phthalate
DMP	dimethyl phthalate
DNHEXP	di- <i>n</i> -hexyl phthalate
DNOP	di- <i>n</i> -octyl phthalate
DOTP	di(2-ethylhexyl) terephthalate
DPENP	di- <i>n</i> -pentyl phthalate
DPHP	di(2-propylheptyl) phthalate
DPS	delayed preputial separation
DSP	decrease spermatocytes and spermatids
DVO	delayed vaginal opening
ECHA	European Chemicals Agency
ECMO	extracorporeal membrane oxygenation
ED ₅₀	median effective dose
EPA	Environmental Protection Agency, U.S.
EPW	epididymal weight
FDA	Food and Drug Administration, U.S.
f _{ue}	urinary excretion factor
GD	gestational day
GGT	gamma-glutamyl transferase
GLP	good laboratory practices
grn	granulin
HBM	human biomonitoring
hCG	human chorionic gonadotrophin
HI	hazard index
HMW	high molecular weight
HPV	high production volume
HQ	hazard quotient
IARC	International Agency for Research on Cancer
ICH	International Conference on Harmonisation
insl3	insulin-like factor 3
IP	intraperitoneally
JRC	Joint Research Centre
LD	lactation day
LH	luteinizing hormone
LMW	low molecular weight
LOAEL	lowest observed adverse effect level
LOD	level/limit of detection

LOQ	level/limit of quantitation
MBP	monobutyl phthalate
MBZP	monobenzyl phthalate
MCPP	mono(3-carboxypropyl) phthalate
MDI	mental development index
MECPP	mono(2-ethyl-5-carboxypentyl) phthalate
MEHP	mono(2-ethylhexyl) phthalate
MEHHP	mono(2-ethyl-5-hydroxyhexyl) phthalate
MEOHP	mono(2-ethyl-5-oxohexyl) phthalate
MEP	monoethyl phthalate
MIBP	monoisobutyl phthalate
MINP	mono(isononyl) phthalate
MIS	Mullerian inhibiting substance
MMP	monomethyl phthalate
MNG	multinucleated gonocyte
MNOP	mono- <i>n</i> -octyl phthalate
MOE	margin of exposure
MSSM	Mount Sinai School of Medicine
MW	molecular weight
NA	not available
NAE	no antiandrogenic effects observed
NCEA	National Center for Environmental Assessment
NHANES	National Health and Nutritional Examination Survey
NNNS	NICU Network Neurobehavioral Scale
NOAEL	no observed adverse effect level
NOEL	no observed effect level
NR	nipple retention
NRC	National Research Council, U.S.
NTP	National Toxicology Program, U.S.
OECD	Organisation for Economic Cooperation and Development
OH-MIDP	mono(hydroxy-isodecyl) phthalate
OH-MINP	mono(hydroxy-isononyl) phthalate
OR	odds ratio
oxo-MIDP	mono(oxo-isodecyl) phthalate
oxo-MINP	mono(oxo-isononyl) phthalate
PBR	peripheral benzodiazepine receptor
PDI	psychomotor developmental index
PE	phthalate ester
PEAA	potency estimates for antiandrogenicity
PND	postnatal day
PNW	postnatal week
POD	point of departure
PODI	point of departure index
PPAR α	peroxisome proliferator-activated receptor alpha
PPS	probability proportional to a measure of size
PSU	primary sampling unit

PVC	polyvinyl chloride
RfD	reference dose
RTM	reproductive tract malformation
SD	Sprague-Dawley
SDN-POA	sexually dimorphic nucleus of the preoptic area
SFF	Study for Future Families
SR-B1	scavenger receptor class B1
SRS	social responsiveness scale
StAR	steroidogenic acute regulatory protein
SVW	seminal vesicle weight
TCDD	2,3,7,8-tetrachlorodibenzo-p-dioxin
TDI	tolerable daily intake
TDS	testicular dysgenesis syndrome
TEF	toxicity equivalency factors
TOTM	tris(2-ethylhexyl) trimellitate
TPIB	2,2,4-trimethyl-1,3 pentanediol diisobutyrate
T PROD	testosterone production
TXIB [®]	2,2,4-trimethyl-1,3 pentanediol diisobutyrate
UF	uncertainty factor

1 Introduction

The Consumer Product Safety Improvement Act (CPSIA)^{*} of 2008 (CPSIA, 2008) was enacted on August 14, 2008. Section 108 of the CPSIA permanently prohibits the sale of any “children’s toy or child care article” individually containing concentrations of more than 0.1% of dibutyl phthalate (DBP), butylbenzyl phthalate (BBP), or di(2-ethylhexyl) phthalate (DEHP). Section 108 prohibits on an interim basis the sale of “any children’s toy that can be placed in a child’s mouth” or “child care article” containing concentrations of more than 0.1% of di-*n*-octyl phthalate (DNOP), diisononyl phthalate (DINP), or diisodecyl phthalate (DIDP). In addition, Section 108 of the CPSIA directs the Consumer Product Safety Commission (CPSC) to convene a Chronic Hazard Advisory Panel (CHAP) “to study the effects on children’s health of all phthalates and phthalate alternatives as used in children’s toys and child care articles.” The CHAP will recommend to the Commission whether any phthalates or phthalate alternatives other than those permanently banned should be declared banned hazardous substances.

This report describes scenario-based estimates of phthalate exposure, which were performed by CPSC staff under the direction of the CHAP. The CHAP selected eight phthalates for study (Table E1-1) because they are subject to the CPSIA, are found in human tissue, and/or exposure data are available. Data sources included reviews of phthalate exposure data (Clark, 2009; Versar/SRC, 2010; Clark *et al.*, 2011). In addition, the CHAP requested the CPSC staff to:

- Include new concentration data that were not available to Clark or Versar/SRC;
- Emphasize the most recent concentration data, rather than the entire historical database;
- Include mouthing exposure to phthalate alternatives; and
- Perform additional sensitivity analyses.

We estimated exposures of four subpopulations (women of reproductive age, infants, toddlers, and children) to eight phthalate esters (PEs) selected by the CHAP. Exposure to phthalate alternatives is described in a separate report.

^{*}Public Law 110-314.

Table E1-1 Phthalate esters in this report.

Name	Abbr. ^a	CAS	MF	MW (range) ^b
Diethyl phthalate	DEP	84-66-2	C12H14O4	222.2
Di- <i>n</i> -butyl phthalate ^c	DBP	84-74-2	C16H22O4	278.4
Diisobutyl phthalate	DIBP	84-69-5	C16H22O4	278.4
Butylbenzyl phthalate ^c	BBP	85-68-7	C19H20O4	312.4
Di- <i>n</i> -octyl phthalate ^d	DNOP	117-84-0	C24H38O4	390.6
Di(2-ethylhexyl) phthalate ^c	DEHP	117-81-7	C24H38O4	390.6
Diisononyl phthalate ^d	DINP	28553-12-0 68515-48-0	C26H42O4	418.6 (390.6–446.7)
Diisodecyl phthalate ^d	DIDP	26761-40-0 68515-49-1	C28H46O4	446.7 (418.6–474.7)

^a Abbr., abbreviation; CAS, Chemical Abstracts Service number, MF, molecular formula; MW, molecular weight.

^b DINP includes isomers with C8 – C10 ester groups; DIDP includes isomers with C9 – C11 ester groups.

^c Subject to a permanent ban in child care articles and children’s toys.

^d Subject to an interim ban in child care articles and toys that can be placed in a child’s mouth.

2 Methodology

In this report, we estimated human exposure to selected PEs by identifying and evaluating relevant exposure scenarios. This approach required knowledge of all relevant sources of PE exposure, data on concentrations of PEs in environmental media and products, physiological parameters, and consumer use information. The scenario-based (indirect) approach is complementary to the biomonitoring approach, which is also employed by the CHAP. The biomonitoring (direct) approach provides robust estimates of total human exposure to PEs but does not provide information regarding the sources of exposure. The scenario-based approach, employed for this report, estimates the relative contributions of various sources of PE exposure.

2.1 Sources and Scenarios

Humans are exposed to PEs from many sources and through multiple pathways and scenarios (Wormuth *et al.*, 2006; Versar/SRC, 2010; Clark *et al.*, 2011). PEs are ubiquitous environmental contaminants present in air, water, soil, food, personal care products (cosmetics), drugs and medical devices, automobiles, and consumer products. * PEs were also commonly used in toys

* In this report, “consumer product” refers to products under the jurisdiction of the CPSC. This includes products used in and around the home, recreational settings, and schools that are not regulated by other federal agencies, for example, food, drugs, personal care products (cosmetics), and medical devices. The terms “personal care products” and “cosmetics” are used interchangeably in this report. Most of the personal care products discussed in the report fall under the Food and Drug Administration’s definition of “cosmetic.”

and child care articles before their use was restricted by the European Commission and the United States. The sources and scenarios that may contribute significantly to human exposure were identified by CPSC staff and are listed in Table E1-2.

Table E1-2 Sources of exposure to phthalate esters included by exposure route.

Source	Target Population (age range)			
	Women (15 to 44) ^a	Infants (0 to <2)	Toddlers (2 to <3)	Children (3 to 12)
Children's Products				
Teethers & toys	D ^b	O, D	O, D	D
Changing pad	--	D	D	--
Play pen	--	D	D	--
Household Products				
Air freshener, aerosol	I (direct) ^c	I (indirect) ^d	I (indirect)	I (indirect)
Air freshener, liquid	I (indirect)	I (indirect)	I (indirect)	I (indirect)
Vinyl upholstery	D	--	D	D
Gloves, vinyl	D	--	--	--
Adhesive, general purpose	D	--	--	--
Paint, aerosol	I, D	--	I (indirect) ^d	I (indirect) ^d
Adult toys	Internal	--	--	--
Personal Care Products				
Soap/body wash	D	D	D	D
Shampoo	D	D	D	D
Skin lotion/cream	D	D	D	D
Deodorant, aerosol	D, I (direct)	I (indirect)	I (indirect)	D, I (direct) ^e
Perfume, aerosol	D, I (direct)	I (indirect)	I (indirect)	D, I (direct) ^e
Hair spray, aerosol	D, I (direct)	I (indirect)	I (indirect)	D, I (direct) ^e
Nail polish	D	--	--	D
Environmental Media				
Outdoor air	I	I	I	I

Source	Target Population (age range)			
	Women	Infants	Toddlers	Children
	(15 to 44) ^a	(0 to <2)	(2 to <3)	(3 to 12)
Indoor air	I	I	I	I
Dust	O	O	O	O
Soil	O	O	O	O
Diet				
Food	O	O	O	O
Water	O	O	O	O
Beverages	O	O	O	O
Prescription drugs	O	--	O	O

^a Age range, years.

^b D, dermal; O, oral; I, inhalation.

^c Includes direct exposure from product use.

^d Indirect exposure from product use by others in the home.

^e Females only.

2.2 Calculations

Exposures were calculated with equations specific to the exposure route and the physico-chemical processes by which exposure may occur. Exposure from direct ingestion was estimated by:

$$E_{O,1} = C \times M \times N \times B \times F/W \quad (1)$$

where: $E_{O,1}$, estimated oral exposure by ingestion, $\mu\text{g}/\text{kg}\cdot\text{d}$; C, concentration in product or environmental medium, $\mu\text{g}/\text{g}$; M, mass ingested per event, g; N, frequency of exposure, events per day, d^{-1} ; B, fraction absorbed by the gastrointestinal tract, unitless; F, fraction of population exposed by this scenario, unitless; W, body weight, kg.

Exposure from mouthing soft plastic teethingers and toys was estimated by:

$$E_{O,2} = R \times T \times N \times B \times F/W \quad (2)$$

where: $E_{O,2}$, estimated oral exposure from mouthing, $\mu\text{g}/\text{kg}\cdot\text{d}$; R, migration rate, $\mu\text{g}/\text{h}$; T, exposure duration, h; N, frequency of exposure, d^{-1} ; B, fraction absorbed, unitless; F, fraction of population exposed by this scenario, unitless; W, body weight, kg.

The migration rate (R) is for a 10-cm^2 disk. A standard surface area of 10 cm^2 was assumed for the surface area of the article in the child's mouth (Simoneau *et al.*, 2001; CPSC, 2002).

Inhalation exposure was calculated by:

$$E_I = C \times I \times T \times N \times B \times F/W \quad (3)$$

where: E_i , estimated inhalation exposure, $\mu\text{g}/\text{kg}\cdot\text{d}$; C , concentration in air, $\mu\text{g}/\text{m}^3$; I , inhalation rate, m^3/h ; T , exposure duration, h; N , frequency of exposure, d^{-1} ; B , fraction absorbed, unitless; F , fraction of population exposed by this scenario, unitless; W , body weight, kg.

Percutaneous exposure* from non-polyvinyl chloride (PVC) products was estimated by:

$$E_{D,1} = C \times M \times D \times T \times N \times F/W \quad (4)$$

where: $E_{D,1}$, estimated dermal exposure, $\mu\text{g}/\text{kg}\cdot\text{d}$; C , concentration in the medium of interest, $\mu\text{g}/\text{g}$; M , mass of medium in contact with the skin, g; D , dermal absorption rate, h^{-1} ; T , exposure duration, h; N , frequency of exposure, events per day, d^{-1} ; F , fraction of population exposed, unitless; W , body weight, kg.

For dermal contact with PVC films or solid products, exposure was estimated by (Deisinger *et al.*, 1998; Wormuth *et al.*, 2006):

$$E_{D,2} = DT \times S \times \left(\frac{D_{PE}}{D_{DEHP}} \right) \times T \times N \times F/W \quad (5)$$

where: $E_{D,2}$, estimated dermal exposure from contact with PVC, $\mu\text{g}/\text{kg}\cdot\text{d}$; DT , rate of dermal transfer and absorption for DEHP, $0.24 \mu\text{g}/\text{cm}^2\cdot\text{h}$ (Deisinger *et al.*, 1998); S , surface area of exposed skin, cm^2 ; D_{PE} , dermal absorption rate of the PE of interest, h^{-1} ; D_{DEHP} , dermal absorption rate of DEHP, h^{-1} ; T , exposure duration per event, h; N , frequency of exposure, d^{-1} ; F , exposed fraction of the population, unitless; W , body weight, kg.

Internal exposure from PVC adult toys was estimated by:

$$E_A = R \times A \times T \times N \times B \times F/W \quad (6)$$

where: E_A , estimated internal exposure, $\mu\text{g}/\text{kg}\cdot\text{d}$; R , migration rate, $\mu\text{g}/\text{cm}^2\cdot\text{h}$; A , product surface area, cm^2 ; T , exposure duration, h; N , frequency of exposure, d^{-1} ; B , fraction absorbed, unitless; F , exposed fraction of the population; W , body weight, kg.

Average values (means) for all parameters were used to estimate the average population exposure. The 95th percentile concentrations (or for toys, migration rates) were generally used to estimate upper bound exposures. In selected scenarios, we also calculated exposures using the mean concentration (or migration rate) with the 95th percentile value for exposure frequency or duration. Data were not available to estimate upper bound exposures for some scenarios.

For some products, such as aerosols and air fresheners, it was necessary to estimate indoor PE concentrations. For aerosols, the initial PE concentration in a room was estimated by:

$$C_0 = M_P \times C_P \times F_0/V \quad (7)$$

* Strictly speaking, equations (4) and (5) calculate absorbed doses, rather than exposures.

where: C_0 , initial concentration in room air, $\mu\text{g}/\text{m}^3$; M_P , mass of product per use, g; C_P , PE concentration in the product, $\mu\text{g}/\text{g}$; F_O , overspray fraction, unitless; V , room volume, m^3 .

The time-dependent PE concentration was given by:

$$C_T = C_0 \times e^{-(ACH+K) \times T} \quad (8)$$

where: C_T , PE concentration in room air at time= T , $\mu\text{g}/\text{m}^3$; C_0 , initial concentration in room air, $\mu\text{g}/\text{m}^3$; ACH, air exchange rate, h^{-1} ; K , first order decay rate, h^{-1} ; and T , time, h.

For aerosol products (deodorant, hair spray, perfume, air freshener, and paint) the PE concentration in the user's breathing zone was estimated by assuming a 1 m^3 breathing zone (Thompson and Thompson, 1990) that exchanges air with room air at a rate of 10 h^{-1} .

For liquid air fresheners, it was assumed that the PE is released into air at a constant rate. Thus, the PE source strength was estimated by:

$$S = \frac{M_P \times C_P}{L_P \times 24} \quad (7)$$

where: S , PE source strength, $\mu\text{g}/\text{h}$; M_P , mass of product, g; C_P , PE concentration in the product, $\mu\text{g}/\text{g}$; L_P , product lifetime, days; 24, conversion factor, h/d.

The steady-state PE concentration in room air was given by:

$$C_{SS} = \frac{S/V}{ACH+K} \quad (8)$$

where: C_{SS} , steady-state PE concentration in room air, $\mu\text{g}/\text{m}^3$; S , source strength, $\mu\text{g}/\text{h}$; V , room volume, m^3 ; ACH, air exchange rate, h^{-1} ; K , first order decay rate, h^{-1} .

2.3 Input Data

Data on PE concentrations in environmental media and products were identified from all available sources, including the primary scientific literature, government reports (*e.g.*, Danish Ministry of the Environment), literature reviews (Versar/SRC, 2010), CPSC studies (Dreyfus, 2010), previously published exposure assessments (Wormuth *et al.*, 2006; Clark *et al.*, 2011), and a database prepared for the Phthalate Ester Panel of the American Chemistry Council (Clark, 2009). Priority was given to studies that were of the highest quality, the most recent, and the most relevant to the U.S. population. We recorded or calculated summary statistics for these concentrations including the mean, 95th percentile, and detection frequency. Nondetects in environmental media and food were assumed to equal one-half the detection limit. Nondetects in consumer and personal care products were regarded as zero because we consider PEs to be intentionally added in these products. Nondetects and zero values were included in the calculation of the summary statistics. Data on personal care products (Table E1-3), household products (Tables E1-4 and E1-5), and environmental media (Table E1-6) are summarized below.

Table E1-3 Phthalate ester concentrations in personal care products ($\mu\text{g/g}$).^a

Product		DEP	DBP
Shampoo (shampoo/body wash)	n	13	NR
	mean	26	
	0.95	143	
	DF (%)	23	
Shampoo/body wash, infant use	n	13	NR
	mean	26	
	0.95	143	
	DF (%)	23	
Soap/body wash	n	3	NR
	mean	175	
	0.95	313	
	DF (%)	67	
Skin lotion/cream	n	18	NR
	mean	30	
	0.95	108	
	DF (%)	33	
Skin lotion/cream, infant use	n	11	NR
	mean	32	
	0.95	174	
	DF (%)	18	
Perfume/fragrance	n	22	NR
	mean	12545	
	0.95	27453	
	DF (%)	100	
Deodorant	n	35	NR
	mean	441	
	0.95	11462	
	DF (%)	57	
Hair spray, gel, mousse	n	49	NR
	mean	112	
	0.95	328	

Product		DEP	DBP
	DF (%)	67	
Nail polish	n	6	6
	mean	189	19207
	0.95	852	60077
	DF (%)	17	56

^a Mean and 95th percentile concentrations ($\mu\text{g/g}$). Nondetects were assumed to equal zero. Abbreviations: n, number of products tested; DF, phthalate ester detection frequency (%), NR, not reported (not present). Sources: Hubinger (2010); Hubinger & Havery (2006); Houlihan *et al.* (2008).

Table E1-4 Phthalate ester concentrations in household products ($\mu\text{g/g}$).^a

Product		DEP	DBP	DIBP	BBP	DINP	Reference
Air freshener, aerosol	n	8	8	NR ^b	NR	NR	NRDC (2007)
	mean	294	0.19				
	0.95	952	0.24				
	DF (%)	63	25				
	range	1.0 – 1100	0.12 – 0.25				
Air freshener, liquid	n	5	5	5	NR	NR	NRDC (2007)
	mean	2436	1.5	1.1			
	0.95	6571	3.9	1.6			
	DF (%)	60	80	60			
	range	0.78 – 7300	0.19 – 4.5	0.24 – 1.6			
Adhesive, general purpose	n	NR	NR	NR	4	NR	NLM (2012)
	mean				9,050		
	0.95				30,800		
	DF (%)				25		
	range				36,200		
Paint/coating, aerosol	n	NR	NR	NR	96	96	NLM (2012)
	mean				1,040	400	
	0.95				0	0	
	DF (%)				2.1	1.0	
	range				50,000	39,000	

^a n, number of products tested; mean, mean concentration; 0.95, 95th percentile concentration; DF, detection frequency (%); range, range of concentrations in products containing phthalates. Summary statistics include zero values.

^b NR, not reported. The phthalate ester was not present in the product.

Table E1-5 Phthalate esters used in PVC products.^a

Product	DNOP	DEHP	DINP	DIDP	Reference
Teethers & toys	?	X	X	?	Assumed
Changing pad	X	X	X	X	Assumed
Play pen	X	X	X	X	Assumed
Furniture	X	--	X	X	Godwin (2010)
Gloves^b	X	X	X	X	Godwin (2010)
Adult toys	X	X	X	--	Nilsson <i>et al.</i> (2006)

^a X, PE present; ?, PE present, but no migration data available; --, PE not present.

^b Assumes similar PEs as used in medical exam gloves.

Table E1-6 Phthalate ester concentrations in environmental media.^a

Medium	DEP	DBP	DIBP	BBP	DNOP	DEHP	DINP	DIDP
Indoor Air ($\mu\text{g}/\text{m}^3$)^b								
mean	0.57	0.20	0.11	0.022	3.5×10^{-4}	0.089	NR	NR
95 th percentile	1.4	0.44	0.26	0.053	ND	0.17	NR	NR
Outdoor Air ($\mu\text{g}/\text{m}^3$)^c								
mean	0.060	0.0035	0.0036	0.0030	3.5×10^{-4}	0.020	NR	NR
95 th percentile	0.16	0.015	0.011	0.0048	ND	0.12	NR	NR
Dust ($\mu\text{g}/\text{g}$)^d								
mean	8.5	27	2.9	120	NR	510	130	34
95 th percentile	11.0	44	5.0	280	NR	850	1,000	110
Soil ($\mu\text{g}/\text{g}$)^e								
mean	NR	3.5×10^{-2}	NR	6.5×10^{-3}	1.3×10^{-2}	2.7×10^{-1}	7.8×10^{-2}	NR
95 th percentile	NR	1.6×10^{-1}	NR	2.6×10^{-2}	4.2×10^{-2}	1.1	3.0×10^{-1}	NR

^a ND, not detected; value shown is one-half the detection limit. NR, not reported.

^b Rudel *et al.* (2003; 2010).

^c Rudel *et al.* (2010).

^d Abb *et al.* (2009); Rudel *et al.* (2003).

^e Vikelsøe *et al.* (1999).

For the purpose of this report, it is assumed that DEHP and DINP are still used in teething toys and toys, even though DEHP use in these products is permanently prohibited by the CPSIA and DINP is banned on an interim basis (Table E1-5). This is to assess the potential impact of PE use in these products, as specified in the CPSIA. Currently, toys and child care articles should not contain prohibited PEs; the prohibitions became effective in 2009. Biomonitoring data used to estimate total PE exposure (CHAP Report, Section 2.5) predate the PE prohibition. Exposure from mouthing toys containing other PEs, such as DNOP and DIDP, were not included because migration data for estimating oral exposure were not available. For the same reasons given above, it is assumed that DNOP, DEHP, DINP, and DIDP are used in changing pads and play pens. Only general information on the use of PEs in PVC products is available (Godwin, 2010). Information on PE use in household products (Godwin, 2010) and adult toys (Nilsson *et al.*, 2006) is summarized in Table E1-5.

Data on physiological parameters (Table E1-7) (such as body weight, inhalation rate, and skin surface area) and product use information (Tables E1-8 – E1-11) (amount of product used, frequency and duration of exposure) were generally derived from a standard reference (EPA 2011). Information on infant mouthing duration (Greene, 2002) and PE migration rates from teething toys and toys (Chen, 2002) were from CPSC studies (Table E1-12). Migration rates were measured by the Joint Research Centre method (Simoneau *et al.*, 2001). PE migration rates from adult toys were from Nilsson *et al.* (2006) (Table E1-13). Dermal absorption rates (Table E1-14) were estimated from published data (Stoltz and El-hawari, 1983; Stoltz *et al.*, 1985; Elsisi *et al.*, 1989). For cases in which use data were not available, it was necessary to make reasonable assumptions regarding use parameters.

We applied a default value of 1.0, assumed for oral, inhalation, and internal (*i.e.*, intravaginal for adult toys) absorption/bioavailability (Table E1-7) (see Discussion).

For estimating inhalation exposures, we assumed a value of 38 m³ for the size of an average bedroom in a small home (Persily *et al.*, 2006; small homes). The air exchange rate is the median value for U.S. homes (Murray and Burmaster, 1995). The hypothetical breathing zone had a volume of 1 m³ (Thompson and Thompson, 1990) and 10 air changes per hour (assumed), which is equivalent to a linear air flow of 0.01 km/h. The first order decay rate of 1 h⁻¹ is appropriate for particles in the general range of 1 to 10 µm in diameter (EPA, 2011, Table 19-29).

Information on exposure to diethyl phthalate (DEP) in prescription drugs (Table E1-14) is from the U.S. Food and Drug Administration (FDA) (Jacobs, 2011). The maximum daily DEP dose (mg/kg-d) and number of prescriptions per year were available for four age groups, although these age groups do not correspond exactly to the age groups in this study. The number of prescriptions was divided by the U.S. population for the age range of interest (Census, 2010) as a rough estimate of the fraction of the population taking a given drug.

2.4 Dietary Exposures

The methods for estimating dietary exposure are described in detail in a separate report (Carlson and Patton, 2012; Appendix E3). Food residue data are from a total diet study from the United Kingdom (Bradley, 2011) that contains the most recently reported food residues available.

Table E1-7 Physiological parameters.

Parameter	Units	Women	Infants	Toddlers	Children	Reference
Age range		15 to 44	0 to <1	1 to <3	3 to 12	
Body weight^{a, b}	kg	75	7.8	12.4	30.7	EPA (2011), Table 8-25 (women); Table 8-1 (juveniles)
Inhalation rate^{b, c}		0.60	0.36	0.55	0.53	EPA (2011), Table 6-15
Surface areas:^b						
Total	cm ²	18,500	3,990	5,700	9,200	EPA (2011), Table-7-13 (women); Tables 7-1 & 7-8 (juveniles)
Hands		900	180	270	420	
Palms, both hands^d		300	60	90	140	
Exposed legs, arms^e		1600	260	380	680	
Changing pad^f		N/A	90	130	N/A	
Playpen^g		N/A	60	90	N/A	
Toys^h		25	10	10	25	Assumed
Dust consumption	g/d	0.03	0.03	0.06	0.06	EPA (2011), Table 5-1
Soil consumption	g/d	0.02	0.03	0.05	0.05	EPA (2011), Table 5-1
Bioavailability:						
Oral	unitless	1	1	1	1	Assumed (see text)
Inhalation		1	1	1	1	
Internalⁱ		1	--	--	--	

^a Mean body weight for females age 18 to 65, NHANES IV.

^b Weighted averages were used to average age ranges with different intervals.

^c Average daily inhalation rate for females, age 16 to 41. Males and females combined for age 0 to <1; 1 to <3; and 3 to <11 years.

^d One-third of total hand area.

^e Estimated skin surface area in contact with a sofa, while sitting, and wearing short pants and short sleeves. Assumes two-thirds of the arms and legs are exposed and one-quarter of exposed area contacts the sofa.

^f Estimated skin surface area in contact with a changing pad. Assumes one-third of genitals, plus buttocks contact the pad.

^g Estimated skin surface area in contact with a playpen. Assumes one-third of hand surface area exposed.

^h Estimated skin surface area in contact with a small (teether or rattle, 10 cm²) or medium (action figure, 25 cm²) toy.

ⁱ Adult toys.

Table E1-8 Product use parameters for women.

Product	Mass per use ^a (g)	Mass on skin (g)	Exposure duration (h)		Over-spray fraction	Uses per day (d ⁻¹)	Fraction exposed	Reference
			Skin	Air				
<i>Personal Care Products</i>								
Shampoo ^b	16	0.16	24	--	--	0.82	1	EPA (2011), Table 17-3
Soap/body wash ^b	2.6	0.026	24	--	--	1.5	1	
Lotion/cream	0.5	0.5	24	--	--	1	1	
Deodorant ^c	0.5	0.5	24	0.1	0.5	1	1	
Perfume, spray ^c	0.23	0.23	24	0.1	0.5	0.29	1	
Nail polish ^d	0.33	0.033	24	--	--	0.16	1	
Hairspray ^c	1.0	0.5	24	0.1	--	0.25	1	Mass is assumed.
<i>Household Products</i>								
Paint, aerosol ^{c, e}	200	2.0	24	0.25	0.5	0.012	0 or 1	EPA (2011), Tables 17-4,
Adhesive ^d	25	0.25	24	0.25	0.5	0.012	0 or 1	17-5, 17-6
Aerosol air freshener ^f	1	--	--	0.1	1.0	1	0.5	
Liquid air freshener ^f	1	--	--	--	--	1	0.5	
<i>Dermal Contact</i>								
Handling toys	--	--	0.1	--	--	1	1	Assumed
Vinyl furniture ^g	--	--	4.0	--	--	1	0 or 1	Babich & Thomas (2001)

Product	Mass per use ^a (g)	Mass on skin (g)	Exposure duration (h)		Over-spray fraction	Uses per day (d ⁻¹)	Fraction exposed	Reference
			Skin	Air				
Vinyl gloves ^h	--	--	0.011	--	--	1	1	EPA (2011), Table 17-12
Adult toys	--	--	0.25	--	--	0.019	0.5	Nilsson <i>et al.</i> (2006)
<i>Time indoors/outdoorsⁱ</i>	--	--	21/3	--	--	--	--	EPA (2011), Table 16-1

^a Mass per use, amount of product per use, g; mass on skin, residual amount of product remaining on skin after use, g; exposure duration, time that product remains on the skin (dermal) or time user is exposed in the breathing zone (air), h; overspray fraction, fraction of aerosol that does not contact the intended surface, unitless; uses per day (frequency of use), number of times the product is used per day, d⁻¹; fraction exposed, fraction of the population that is exposed to the product, unitless.

^b For shampoo and soap/body wash, it was assumed that 1% of the product remained on the skin for 24 hours. For all other personal care products, it was assumed that the amount used remains on the skin for 24 hours.

^c For aerosol products, it was assumed that the user is exposed in a breathing zone during product use. The listed exposure duration for air is the time exposed in the breathing zone. Indirect exposure from room air occurs for the time indoors (21 hours).

^d For nail polish and adhesive, it was assumed that 1% of mass contacts the skin.

^e For aerosol paint and lacquer, it was assumed that 1% of mass contacts the skin. The overspray fraction was assumed. The fraction exposed was assumed to equal either 0 (non-users) or 1 (users of products containing phthalates). The use parameters available were for users only. The fraction of products containing phthalate esters is unknown.

^f Daily use of aerosol air freshener or continuous use of liquid air freshener was assumed. The fraction exposed was assumed to equal 0.5 for each.

^g Time spent sitting while reading or watching television. The prevalence of vinyl-covered furniture is unknown. Assume average person is unexposed and that an exposed individual represents the upper bound exposure.

^h Average dish detergent use is 107 hours per year.

ⁱ Average time outdoors rounded to the nearest hour. Time indoors was assumed to equal 24 minus time outdoors.

Table E1-9 Product use parameters for infants.

Product	Mass per use ^a	Mass on skin	Exposure duration (h)		Frequency of use	Fraction exposed	Reference
	(g)	(g)	mean	0.95	(d ⁻¹)	(unitless)	
Personal Care Products							
Soap/body wash ^b	1	0.01	24	--	1	1	
Lotion/cream ^c	1.4	1.4	24	--	1	1	EPA (2011), Table 17-3 (baby use)
Dermal Contact						1	
Teethers & toys ^d	--	--	4.3	--	1	0.3	EPA (2011), Table 16-62
Changing pad ^e	--	--	0.08	0.17	6	1	O'Reilly (1989)
Play pen ^f	--	--	4.3	12.6	1	0.3	EPA (2011), Table 16-62
Mouthing							
Teethers & toys ^g	--	--	0.073	0.292	1	1	Greene (2002)
Time indoors/outdoors ^h	--	--	23/1	--	1	1	EPA (2011), Table 16-1

^a Mass per use, amount of product per use, g; mass on skin, residual amount of product remaining on skin after use, g; exposure duration, time that product remains in contact with skin (mean and 95th percentile), h; frequency of use, number of times the product is used per day, d⁻¹; fraction exposed, fraction of the population that is exposed to the product, unitless.

^b For soap/body wash, it was assumed that 1% of the product remained on the skin for 24 hours. Frequency and amount per use for soap/body wash are assumed.

^c For lotion/cream, it was assumed that the amount used remains on the skin for 24 hours. Parameters are for baby use.

^d Time “playing games” for 3- to 6-month olds.

^e Exposure duration is assumed to be 5 minutes (mean) or 10 minutes (upper bound). Frequency of use is from O'Reilly (1989).

^f Average duration is the time playing games; upper bound is the time sleeping/napping. EPA (2011), Table 16-62.

^g Time spent mouthing “all soft plastic articles except pacifiers” (Greene, 2002).

^h Average time outdoors rounded to the nearest hour. Time indoors was assumed to equal 24 minus time outdoors. Indirect (room air) exposures to aerosol products occur during the time indoors (23 h).

Table E1-10 Product use parameters for toddlers.

Product	Mass per use ^a	Mass on skin	Exposure duration (h)		Frequency of use	Fraction exposed	Reference
	(g)	(g)	mean	0.95	(d ⁻¹)	(unitless)	
Personal Care Products^b							
Shampoo ^c	0.5	0.005	24	--	0.27	1	EPA (2011), Table 17-3
Soap/body wash ^c	2.6	0.026	24	--	1.2	1	
Lotion/cream ^d	1.4	1.4	24	--	1.0	1	
Dermal Contact						1	
Teethers & toys ^e	--	--	3.2	--	1	0.64	EPA (2011), Table 16-62
Changing pad ^f	--	--	0.08	0.17	5	1	O'Reilly 1989
Play pen ^g	--	--	3.2	11.8	1	0.64	EPA (2011), Table 16-62
Vinyl-covered furniture ^h	--	--	1.6	--	1	0 or 1	
Mouthing							
Teethers & toys ⁱ	--	--	0.067	0.263	--	1	Greene (2002)
Time indoors/outdoors^j	--	--	23/1	--	--	1	EPA (2011), Table 16-1

^a Mass per use, amount of product per use, g; mass on skin, residual amount of product remaining on skin after use, g; exposure duration, time that product remains in contact with skin (mean and 95th percentile), h; frequency of use, number of times the product is used per day, d⁻¹; fraction exposed, fraction of the population that is exposed to the product, unitless.

^b Use infant/baby use parameters, where available.

^c For shampoo and soap, it was assumed that 1% of the product remained on the skin for 24 hours. For lotion/cream, it assumed that the amount used remains on the skin for 24 hours.

^d For lotion/cream, it was assumed that the amount used remains on the skin for 24 hours. Parameters are for baby use.

^e Time playing games, 1-year-olds.

^f Exposure duration is assumed to be 5 minutes (mean) or 10 minutes (upper bound). Frequency is from O'Reilly (1989).

^g Average duration is the time playing. Upper bound is the time sleeping/napping. EPA (2011), Table 16-62. One-year olds.

^h Time watching television. EPA (2011), Table 16-77.

ⁱ Time spent mouthing "all soft plastic articles except pacifiers" (Greene, 2002).

^j Average time outdoors rounded to the nearest hour. Time indoors was assumed to equal 24 minus time outdoors. Indirect (room air) exposures to aerosol products occur during the time indoors (23 h).

Table E1-11 Product use parameters for children.

Product	Mass per use ^a	Mass on skin	Exposure duration (h)		Over-spray	Uses per day	Fraction exposed	Reference
	(g)	(g)	skin	air	fraction	(d ⁻¹)	(unitless)	
Personal Care Products^b								
Shampoo^c	16	0.16	24	--	--	0.82	1	EPA (2011), Table 17-3
Soap/body wash^c	2.6	0.026	24	--	--	1.5	1	
Lotion/cream^c	0.5	0.5	24	--	--	1	1	
Deodorant^d	0.5	0.5	24	0.1	0.5	1	1	
Perfume, spray^d	0.23	0.23	24	0.1	0.5	0.29	0.5	
Nail polish^e	0.33	0.033	24	--	--	0.16	0.5	
Hairspray^d	1.0	0.5	24	0.1	--	0.25	0.5	Mass is assumed
Dermal Contact							1	
Toys^f	--	--	2.1	--	--	1	0.4	EPA (2011), Table 16-62
Vinyl-covered furniture^g	--	--	2.7	--	--	--	0 or 1	
Time indoors/outdoors^h	--	--	22/2	--	--	--	1	EPA (2011), Table 16-1

^a Mass per use, amount of product per use, g; mass on skin, residual amount of product remaining on skin after use, g; exposure duration, time that product remains on the skin (skin) or time user is exposed in the breathing zone (air), h; overspray fraction, fraction of aerosol that does not contact the intended surface, unitless; uses per day (frequency of use), number of times the product is used per day, d⁻¹; fraction exposed, fraction of the population that is exposed to the product, unitless.

^b Use adult use parameters for children ages 3 to 12.

^c For shampoo and soap, it was assumed that 1% of the product remained on the skin for 24 hours. For lotion/cream, it was assumed that the amount used remains on the skin for 24 hours.

- ^d For aerosol products, it was assumed that the user is exposed in a breathing zone during product use (duration listed under air) and exposure from room air occurs for the time indoors (22 h).
- ^e For nail polish, it was assumed that 1% of mass contacts the skin.
- ^f Time playing games, average of 3- to 11-year olds.
- ^g Average time outdoors rounded to the nearest hour. Time indoors was assumed to equal 24 minus time outdoors.

Table E1-12 Phthalate ester migration into artificial saliva.^a

Phthalate ester	n ^b	Migration rate (µg/h)	
		Mean	95th Percentile
DINP	25	4.2	10.1
DEHP	3	1.3	1.9

^a Chen (2002). Migration rate (µg/10 cm²-h) measured by a modification of the Joint Research Centre method (Simoneau *et al.*, 2001).

^b n, number of products tested.

Table E1-13 Phthalate ester migration from adult toys.^a

Phthalate ester	Lubricant	Migration rate (µg/cm ² -h)
DNOP	none	0.08
DEHP	none	0.04
DEHP	water-based	0.04
DEHP	oil-based	54.8

^a Nilsson *et al.* (2006).

Table E1-14 Estimated percutaneous absorption rates (h^{-1}) for phthalate esters.

Phthalate ester	Absorption rate	Reference
Diethyl phthalate (DEP)	1.1×10^{-2}	Elsisi <i>et al.</i> (1989) ^a
Dibutyl phthalate (DBP)	5.3×10^{-3}	Elsisi <i>et al.</i> (1989)
Diisobutyl phthalate (DIBP)	3.2×10^{-3}	Elsisi <i>et al.</i> (1989)
Butylbenzyl phthalate (BBP)	1.7×10^{-3}	Elsisi <i>et al.</i> (1989)
Di-<i>n</i>-octyl phthalate (DNOP)	2.4×10^{-4}	Same as DEHP (assumed)
Di(2-ethylhexyl) phthalate (DEHP)	2.4×10^{-4}	Elsisi <i>et al.</i> (1989)
Diisononyl phthalate (DINP)	2.0×10^{-4}	Stoltz & El-hawari (1983); Stoltz <i>et al.</i> (1985)
Diisodecyl phthalate (DIDP)	3.4×10^{-5}	Elsisi <i>et al.</i> (1989)

^a Rates were estimated from the absorption at 24 hours in Elsis *et al.* (1989), Figure 2.

Table E1-15 Maximum diethyl phthalate (DEP) exposure (mg/d) from prescription drugs by age group.^a

Drug	Adults			0–6 Years			7–11 Years		
	Dose ^b	No.	F	Dose	No.	F	Dose	No.	F
A	134	9.6 x 10 ⁵	4.1 x10 ⁻³	67	2.5 x 10 ³	8.6 x10 ⁻⁵	67	1.1 x 10 ⁴	5.6 x10 ⁻⁴
B	20	4.4 x 10 ⁶	1.9 x10 ⁻²	5	4.0 x 10 ³	1.4 x10 ⁻⁴	10	9.0 x 10 ³	4.5 x10 ⁻⁴
C	7	2.4 x 10 ⁶	1.0 x10 ⁻²	7	2.9 x 10 ²	9.6 x10 ⁻⁶	7	1.4 x 10 ³	7.1 x10 ⁻⁵
D	3	4.6 x 10 ⁵	2.0 x10 ⁻³	3	1.7 x 10 ²	5.6 x10 ⁻⁶	3	2.7 x 10 ³	1.3 x10 ⁻⁴
E	19	9.6 x 10 ⁴	4.1 x10 ⁻⁴	7	1.0 x 10 ²	3.4 x10 ⁻⁶	7	7.1 x 10 ¹	3.5 x10 ⁻⁶
F	34	4.4 x 10 ⁴	1.9 x10 ⁻⁴				11	1.4 x 10 ¹	6.8 x10 ⁻⁷
G	8	1.1 x 10 ⁵	4.6 x10 ⁻⁴				8	3.8 x 10 ¹	1.9 x10 ⁻⁶
H	5	1.5 x 10 ⁵	6.4 x10 ⁻⁴	5	4.0 x 10 ¹	1.4 x10 ⁻⁶	5	6.0 x 10 ¹	3.0 x10 ⁻⁶
I	15	1.8 x 10 ⁴	7.7 x10 ⁻⁵	6	3.3 x 10 ¹	1.1 x10 ⁻⁶	8	2.5 x 10 ²	1.2 x10 ⁻⁵
J	12	1.4 x 10 ²	5.9 x10 ⁻⁷	8	6.3	2.1 x10 ⁻⁷	10	1.0 x 10 ¹	5.0 x10 ⁻⁷
K	22	4.4 x 10 ¹	1.9 x10 ⁻⁷						
L	20	5.0 x 10 ¹	2.2 x10 ⁻⁷						
M	4	3.8 x 10 ¹	1.6 x10 ⁻⁷						
Total		8.7 x10 ⁶	3.7 x10 ⁻²		7.2 x10 ³	2.4 x10 ⁻⁴		2.5 x10 ⁴	1.2 x10 ⁻³
Population		2.3 x10 ⁸			3.0 x10 ⁷			2.0 x10 ⁷	

^a Source: Personal communication from Abigail Jacobs, U.S. Food and Drug Administration, Center for Drug Evaluation and Research (Jacobs, 2011). All are oral medications. Data for male and females are combined.

^b Dose; maximum daily DEP exposure, mg/d; No., number of prescriptions per year; F, fraction of population exposed.

Table E1-16 Mean and 95th percentile concentrations of selected phthalate esters in food commodities (µg/g).^a

Food Commodity		DEP	DBP	DIBP	BBP	DNOP	DEHP	DINP	DIDP
Grain	<i>Mean</i>	5.1	12.3	25.2	9.0	12	78	639	393
	<i>0.95</i>	11.4	35.4	91.6	25.7	35	234	2984	1198
Dairy	<i>Mean</i>	21.1	6.8	18.2	7.1	12	173	508	326
	<i>0.95</i>	89.2	17.2	69.9	16.4	26	554	1394	943
Fish	<i>Mean</i>	13.6	12.8	10.0	14.7	17	98	819	377
	<i>0.95</i>	40.2	51.5	40.7	46.6	45	286	2174	1281
Meat	<i>Mean</i>	5.1	6.8	5.5	12.2	11	54	298	236
	<i>0.95</i>	16.1	28.3	14.2	35.0	38	191	927	986
Fat	<i>Mean</i>	7.2	20.8	17.3	108.8	47	689	1481	1055
	<i>0.95</i>	29.2	54.2	46.5	93.2	133	2784	2851	2397
Eggs	<i>Mean</i>	4.7	5.2	5.7	9.4	20	24	385	259
	<i>0.95</i>	8.2	8.8	10.9	19.8	71	39	742	407

^a Mean and 95th percentile concentrations were estimated from data in Bradley (2011) as described in Carlson and Patton (2012). Nondetects were treated as one-half the detection limit.

Two hundred and sixty-one retail food items were analyzed for 15 phthalate esters (diesters), nine phthalate monoesters, and phthalic acid. Only the data on the eight diesters listed in Table E1-1 were used. Nondetects were regarded as one-half the detection limit. The mean and 95th percentile concentrations were calculated for each food category (Table E1-16).

Food items in this study were categorized as either grain products, dairy products, fish products, meat products, fat products, or eggs (EPA, 2007). A few of the food categories were not represented by food item/residue data because these data were not present in the Bradley (2011) study. These included vegetable, fruit, soy, and nuts. Categories that were not represented by at least one food item were excluded from further analysis.

PE concentrations in food (Table E1-16) and consumption estimates (Table E1-17) for these categories were used to estimate per capita (population) dietary exposures (EPA, 2007). For each population and PE, mean and 95th percentile dietary exposures ($\mu\text{g}/\text{kg}\cdot\text{d}$) were calculated by summing the contribution from each food category, using equation (1). For dietary exposures only, we used the body weights appropriate for the age-specific consumption estimates (EPA, 2007).

Table E1-17 Average daily food consumption (g/d) by age group (EPA, 2007).

Food Type	Women	Infants	Toddlers	Children
Grain	135.05	18.57	86.7	120.58
Dairy	221.92	107.36	420.4	406.84
Fish	15.48	0.29	4.29	5.88
Meat	127.02	10.56	62.04	87.62
Fat	62.71	34.32	45.11	58.21
Eggs	23.4	2.53	15.98	15.65
Age (y):	≥ 20	0 to <1	1 to 5	6 to 11
Body weight (kg)	73	8.8	15.15	29.7

3 Results

3.1 Total Exposure

Estimates of mean and 95th percentile exposures to eight phthalate esters are shown in Table E1-18 and Figure E1-1. For women, mean PE exposures ranged from 0.15 µg/kg-d (DIBP) to 18.1 µg/kg-d (DEP). Estimated mean DINP exposures were higher than those of any other PE for infants (21 µg/kg-d), toddlers (31 µg/kg-d), and children (14 µg/kg-d). For infants, toddlers, and children, the estimated 95th percentile DINP exposures were as high as 93 µg/kg-d, which is close to the acceptable daily intake for DINP derived by the 2001 CHAP on DINP of 120 µg/kg-d (CPSC, 2001). DEP, DEHP, and DIDP also contributed substantially to the total PE exposure in all subpopulations.

3.2 General Sources of Phthalate Ester Exposure

Exposure sources and scenarios were grouped into seven categories: diet, prescription drugs, toys, child care articles, personal care products, indoor environment, and outdoor environment. The categories are defined in Table E1-19. Tables E1-20 to E1-23 and Figure E1-2 give the relative contributions (as percent of total exposure) of the seven sources for each PE and for each subpopulation. Overall, diet was the predominant source of exposure to DIBP, BBP, DNOP, DEHP, DINP, and DIDP. Personal care products were the major source of exposure to DEP and DBP.

For women (Table E1-20), diet contributes more than 50% of the exposure to DIBP, DNOP, DEHP, DINP, and DIDP. Based on the mean (population mean) exposure, prescription drugs are the greatest source of DEP exposure. However, prescription drugs containing DEP are taken by less than 5% of the population. Therefore, most women are not exposed to DEP in prescription drugs. Because of the skewed distribution for exposure from drugs, we used the average DEP exposure for women who take prescription drugs containing DEP to estimate an upper bound exposure for the whole population. As with the average, this value overestimates the 95th percentile exposure because it represents less than 5% of the population. In the absence of prescription drugs, personal care products contributed significantly to women's DEP exposure. Personal care products, specifically nail polish, were a significant source of DBP exposure (see Section 3.3. below).

For infants and toddlers (Tables E1-21, E1-22), more than 50% of DIBP, DINP, and DIDP exposure and more than 40% of DEHP exposure was from the diet. Dermal contact with child care articles (play pen and changing pad) contributed roughly 90% of the estimated DNOP exposure and contributed substantially to the estimated exposures from DEHP and DINP. However, the methodology used to estimate PE exposure for this scenario is uncertain, and data on DNOP exposure from other sources are limited (see Discussion). Toys (including both mouthing and handling) contributed modestly to DINP and DEHP exposures in infants (about 9 to 13%) and toddlers (about 5%). Currently, DINP and DEHP are not allowed in toys and child

Table E1-18 Estimated mean and 95th percentile total phthalate ester exposure ($\mu\text{g}/\text{kg}\cdot\text{d}$) by subpopulation.

PE	Women		Infants		Toddler		Children	
	(15 to <45)		(0 to <1)		(1 to <3)		(3 to 12)	
	<i>mean</i>	<i>0.95</i>	<i>Mean</i>	<i>0.95</i>	<i>mean</i>	<i>0.95</i>	<i>mean</i>	<i>0.95</i>
DEP	18.1	398	3.1	14.9	2.8	2188	2.8	1149
DBP	0.29	5.7	0.51	1.2	0.69	1.6	0.55	7.4
DIBP	0.15	0.50	0.48	1.5	0.86	3.0	0.45	1.6
BBP	1.1	2.6	1.8	4.0	2.4	5.8	1.1	2.4
DNOP	0.17	21.0	4.4	9.6	5.4	16.0	0.52	15.4
DEHP	1.6	5.6	12.2	33.8	15.7	46.7	5.4	16.5
DINP	5.1	32.5	20.7	57.4	30.8	93.3	14.3	55.1
DIDP	3.2	12.2	10.0	26.4	16.6	47.6	9.1	28.1

Table E1-19 Categories of exposure sources.

Category	Exposure Source
Diet	Food, beverages, water
Prescription Drugs	Prescription drugs only
Toys ^a	Mouthing (infants and toddlers) and dermal (all) exposure to teething toys and toys
Child-care Articles ^a	Dermal contact with PVC changing pads, play pens
Personal Care Products	Soap, shampoo, lotion, deodorant, perfume, hair spray, and nail polish
Indoor Environment ^a	Indoor air, household dust, furniture, vinyl gloves, air fresheners, adhesive, aerosol paint, and adult toys
Outdoor Environment	Outdoor air and soil

^a These categories include products under CPSC jurisdiction.

Table E1-20 Sources of phthalate ester exposure (percent of total exposure) for women.

PE		Diet ^a	Drugs	Toys ^b	Child Care ^b	Personal Care	Indoors ^b	Outdoors
DEP	<i>mean</i>	0.5	76.4	0	0	21.8	1.2	<0.1
	<i>0.95</i>	0.1	92.8	0	0	6.9	0.2	<0.1
DBP	<i>mean</i>	26.4	0	0	0	58.6	14.9	<0.1
	<i>0.95</i>	4.0	0	0	0	94.4	1.6	<0.1
DIBP	<i>mean</i>	87.0	0	0	0	0	12.9	<0.1
	<i>0.95</i>	90.9	0	0	0	0	9.1	<0.1
BBP	<i>mean</i>	14.3	0	0	0	0	85.7	<0.1
	<i>0.95</i>	9.8	0	0	0	0	90.2	<0.1
DNOP	<i>mean</i>	75.8	0	4.7	0	0	19.5	<0.1
	<i>0.95</i>	1.7	0	<0.1	0	0	98.3	<0.1
DEHP	<i>mean</i>	84.2	0	0.5	0	0	15.2	<0.1
	<i>0.95</i>	87.8	0	0.1	0	0	11.9	<0.1
DINP	<i>mean</i>	95.3	0	0.1	0	0	4.6	<0.1
	<i>0.95</i>	44.6	0	<0.1	0	0	55.3	<0.1
DIDP	<i>mean</i>	99.4	0	<0.1	0	0	0.6	<0.1
	<i>0.95</i>	75.8	0	<0.1	0	0	24.2	<0.1

^a Categories are defined in Table E1-19. Values are rounded to the nearest 0.1%.

^b These categories include products under CPSC jurisdiction.

Table E1-21 Sources of phthalate ester exposure (percent of total exposure) for infants.

PE		Diet ^a	Drugs	Toys ^b	Child Care ^b	Personal Care	Indoors ^b	Outdoors
DEP	<i>mean</i>	9.7	0	0	0	64.8	25.3	0.1
	<i>0.95</i>	8.4	0	0	0	78.1	13.5	<0.1
DBP	<i>mean</i>	39.1	0	0	0	0	60.9	0.1
	<i>0.95</i>	45.6	0	0	0	0	54.3	0.1
DIBP	<i>mean</i>	73.6	0	0	0	0	26.4	<0.1
	<i>0.95</i>	80.8	0	0	0	0	19.1	<0.1
BBP	<i>mean</i>	30.8	0	0	0	0	69.1	<0.1
	<i>0.95</i>	16.8	0	0	0	0	81.1	<0.1
DNOP	<i>mean</i>	8.5	0	0	91.5	0	<0.1	<0.1
	<i>0.95</i>	10.2	0	0	89.8	0	<0.1	<0.1
DEHP	<i>mean</i>	41.1	0	9.2	33.0	0	16.7	<0.1
	<i>0.95</i>	54.3	0	9.8	25.6	0	10.3	<0.1
DINP	<i>mean</i>	66.89	0	12.8	16.5	0	3.8	<0.1
	<i>0.95</i>	62.4	0	16.6	12.7	0	8.3	<0.1
DIDP	<i>mean</i>	93.0	0	0	5.7	0	1.3	0
	<i>0.95</i>	93.8	0	0	4.6	0	1.6	0

^a Categories are defined in Table E1-19. Values are rounded to the nearest 0.1%.

^b These categories include products under CPSC jurisdiction.

Table E1-22 Sources of phthalate ester exposure (percent of total exposure) for toddlers.

PE		Diet	Drugs	Toys	Child Care ^b	Personal Care	Indoors	Outdoor
DEP	<i>mean</i>	24.2	19.1	0	0	25.3	31.3	0.1
	<i>0.95</i>	0.1	99.6	0	0	0.2	0.1	<0.1
DBP	<i>mean</i>	51.9	0	0	0	0	48.0	<0.1
	<i>0.95</i>	59.7	0	0	0	0	40.2	0.1
DIBP	<i>mean</i>	85.5	0	0	0	0	14.5	<0.1
	<i>0.95</i>	90.2	0	0	0	0	9.7	<0.1
BBP	<i>mean</i>	26.8	0	0	0	0	73.2	<0.1
	<i>0.95</i>	18.2	0	0	0	0	81.8	<0.1
DNOP	<i>mean</i>	11.3	0	0	88.7	0	<0.1	<0.1
	<i>0.95</i>	9.8	0	0	90.2	0	<0.1	<0.1
DEHP	<i>mean</i>	48.0	0	5.2	30.6	0	16.1	<0.1
	<i>0.95</i>	55.5	0	4.4	30.9	0	9.2	<0.1
DINP	<i>mean</i>	77.9	0	5.4	13.2	0	3.5	<0.1
	<i>0.95</i>	74.4	0	5.9	13.0	0	6.7	<0.1
DIDP	<i>mean</i>	94.9	0	0	4.1	0	1.0	0
	<i>0.95</i>	94.6	0	0	4.3	0	1.1	0

^a Categories are defined in Table E1-19. Values are rounded to the nearest 0.1%.

^b These categories include products under CPSC jurisdiction.

Table E1-23 Sources of phthalate ester exposure (percent of total exposure) for children.

PE		Diet ^a	Drugs	Toys ^b	Child Care ^b	Personal Care	Indoors ^b	Outdoors
DEP	<i>mean</i>	12.4	50.9	0	0	24.9	11.7	0.1
	<i>0.95</i>	0.1	99.3	0	0	0.5	0.1	<0.1
DBP	<i>mean</i>	38.2	0	0	0	38.4	23.3	<0.1
	<i>0.95</i>	7.9	0	0	0	88.7	3.4	<0.1
DIBP	<i>mean</i>	89.6	0	0	0	0	10.3	<0.1
	<i>0.95</i>	93.1	0	0	0	0	6.9	<0.1
BBP	<i>mean</i>	36.9	0	0	0	0	63.0	<0.14
	<i>0.95</i>	25.9	0	0	0	0	74.0	0.1
DNOP	<i>mean</i>	68.2	0	31.7	0	0	0.0	<0.1
	<i>0.95</i>	5.9	0	1.1	0	0	93.0	<0.1
DEHP	<i>mean</i>	78.0	0	3.0	0	0	18.9	<0.1
	<i>0.95</i>	88.5	0	1.0	0	0	10.5	<0.1
DINP	<i>mean</i>	96.1	0	1.0	0	0	3.0	<0.1
	<i>0.95</i>	73.3	0	0.3	0	0	26.5	<0.1
DIDP	<i>mean</i>	99.0	0	0.3	0	0	0.7	0
	<i>0.95</i>	91.9	0	0.1	0	0	8.0	0

^a Categories are defined in Table E1-19. Values are rounded to the nearest 0.1%.

^b These categories include products under CPSC jurisdiction.

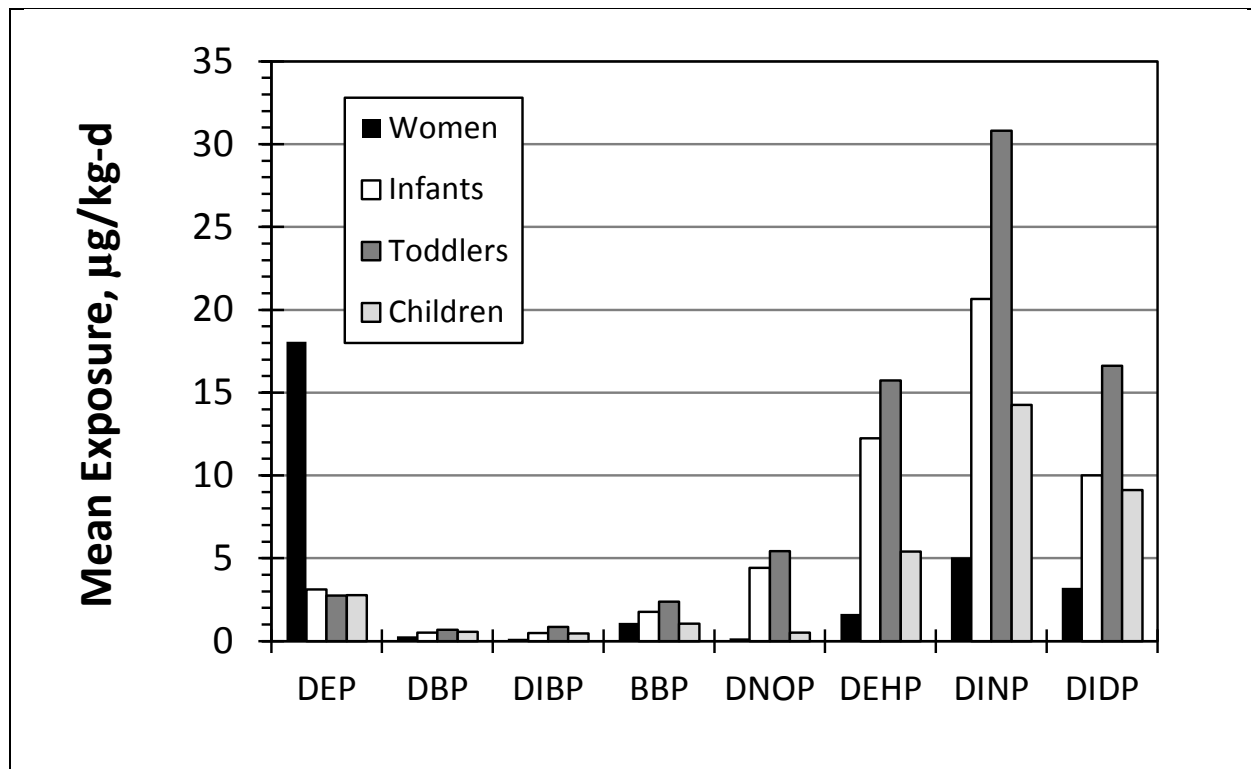


Figure E1-1 Estimated phthalate ester exposure ($\mu\text{g}/\text{kg}\cdot\text{d}$) for eight phthalates and four subpopulations.

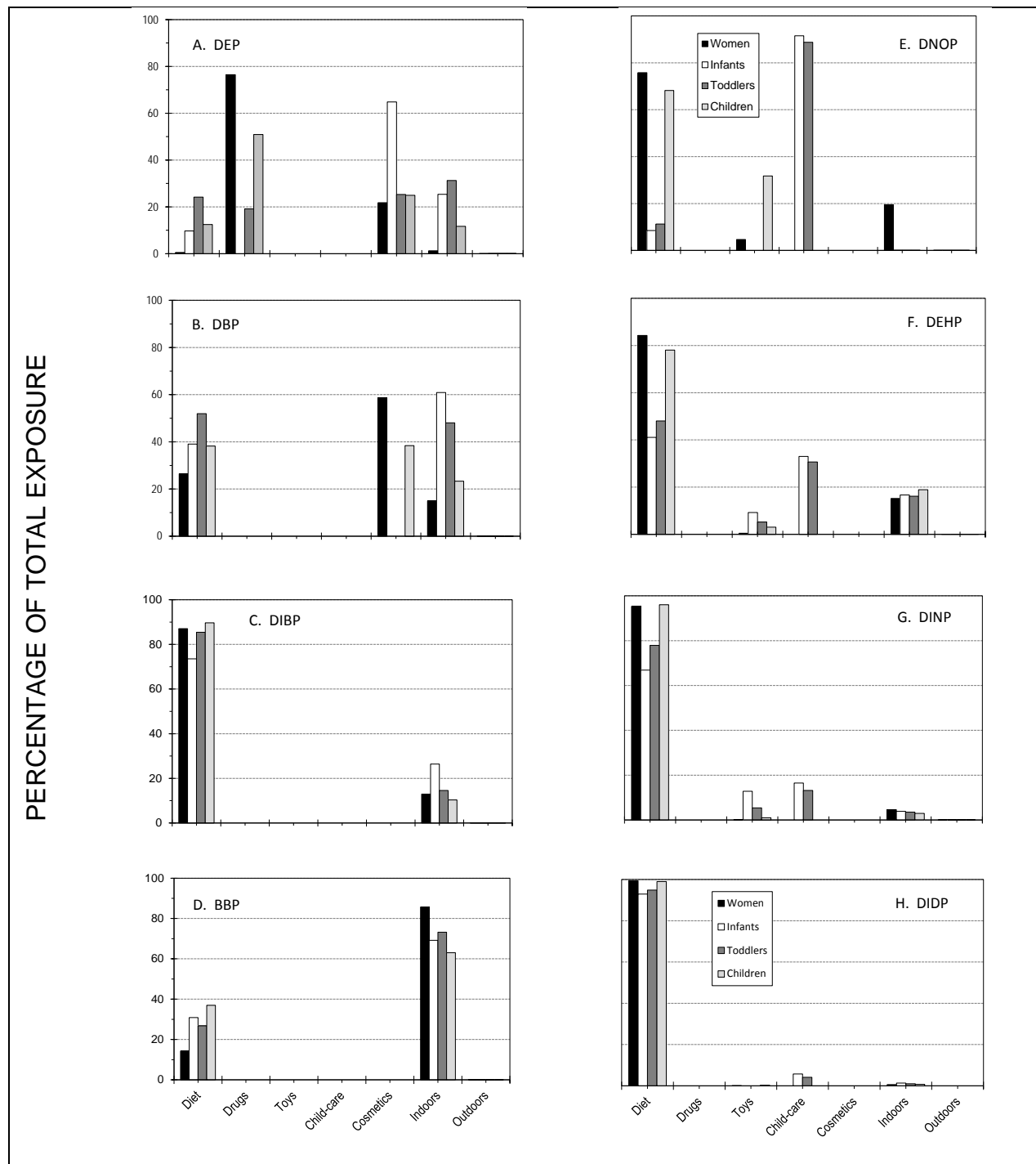


Figure E1-2 Sources of phthalate ester exposure. Percentage of total exposure for seven sources: (1) diet, (2) prescription drugs, (3) toys, (4) child care articles, (5) personal care products (cosmetics), (6) indoor sources, and (7) outdoor sources. Sources are defined in Table E1-19. Solid black bars, women; white bars, infants; dark gray bars, toddlers; and light gray bars, children.

care articles; the estimates described here are based on older residue data for these products. The indoor environment (including indoor air, household dust, air fresheners, and indirect exposure from aerosol paints) contributed substantially (15% to 73%) to infant and toddler exposures to lower molecular weight PEs, including DEP, DBP, DIBP, and BBP. Personal care products (including indirect exposure from the mother's use) contributed more than 50% of DEP exposure to infants.

For children (Table E1-23), diet accounted for more than 50% of DIBP, DNOP, DINP, and DIDP exposure and more than 35% of DBP and BBP exposure. Handling toys contributed modestly (less than 5%) to DEHP, DINP, and DIDP exposure, and over 30% to DNOP exposure. Exposures to DNOP, DEHP, DINP, and DIDP from toys are hypothetical because these PEs currently are not allowed in toys. Personal care products were a significant source of DBP and DEP exposure. The indoor environment contributed more than 60% of exposure to BBP. The indoor environment includes indoor air, household dust, home furnishings, and indirect exposure from aerosol paints.

3.3 Individual Scenarios for Phthalate Ester Exposure

The estimated exposure from each specific scenario is provided in supplementary data Tables E1-S1 to E1-S4. For women, three scenarios presented potentially high exposures: (i) aerosol paint products (BBP and DINP); (ii) dermal contact with PVC products, such as home furnishings and household gloves (BBP, DNOP, DEHP, DINP, and DIDP); and (iii) adult toy use in combination with an oil-based lubricant (upper bound exposure to DEHP) (Table E1-S1). For various reasons, these scenarios are also more uncertain relative to most other sources, as discussed below (see Discussion).

For infants and toddlers, incidental ingestion of household dust contributed roughly 25% to the total BBP exposure and 15% to total DEHP exposure (Tables E1-S2, E1-S3). The sources of PEs in household dust are unknown but may include consumer products (see Discussion). Indoor air contributed roughly one-fourth of the total exposure to the lower molecular weight PEs DEP, DBP, and DIBP.

For children, dust was a significant source of exposure to DEHP (18%). Other significant indoor sources were indirect exposure to aerosol paints (BBP, DINP), nail polish (DBP), and indoor air (DBP) (Table E1-S4).

Individual scenarios that contribute more than 10% of the total exposure for a given PE are summarized in Table E1-24. Overall, diet was the primary source of exposure to DIBP, BBP, DNOP, DEHP, DINP, and DIDP. Personal care products were the primary source of exposure to DEP and DBP. Drugs, air fresheners, and perfume also contributed to DEP exposure. Indoor air contributed to total DIBP exposure. Dust contributed to DEHP and BBP exposure. Mouthing and handling toys contributed to total DINP exposure. Use of particular products containing BBP, DNOP, or DINP resulted in substantial exposures in certain scenarios.

Table E1-24 Scenarios contributing >10% of the total exposure to individual phthalate esters.

PE	Women	Infants	Toddlers	Children
DEP	drugs > perfume	lotion >indoor air > hair spray, diet	diet > indoor air, drugs, perfume	drugs > diet, perfume
DBP	nail polish >diet > indoor air	diet >indoor air, dust	diet >indoor air > dust	nail polish, diet > indoor air
DIBP	diet >indoor air	diet >indoor air	diet > indoor air	diet
BBP	aerosol paint > gloves > diet	aerosol paint > diet, dust	aerosol paint > diet, dust	aerosol paint, diet > dust
DNOP	diet > gloves	play pen >changing pad >diet	play pen >changing pad >diet	diet >handling toys
DEHP	diet > dust	diet > play pen, dust, changing pad	diet >play pen >dust	diet >dust
DINP	diet	diet > mouthing teethers & toys, play pen	diet >play pen	diet
DIDP	diet	diet	diet	diet

Table E1-25 Comparison of modeled estimates of total phthalate ester exposure ($\mu\text{g}/\text{kg}\cdot\text{d}$).

PE	Study	Adult female		Infants		Toddlers		Children	
		Ave. ^a	U.B.	Ave.	U.B.	Ave.	U.B.	Ave.	U.B.
DEP	Wormuth ^b	1.4	65.7	3.5	19.4	1.5	8.1	0.7	4.6
	Clark ^c	--	--	0.3	1.2	1.2	3.8	0.9	2.8
	This study ^d	18.1	398	3.1	14.9	2.8	2188	2.8	1149
DBP	Wormuth	3.5	38.4	7.6	43.0	2.7	24.9	1.2	17.7
	Clark	--	--	1.5	5.7	3.4	12.0	2.4	8.1
	This study	0.3	5.7	0.5	1.2	0.7	1.6	0.5	7.4
DIBP	Wormuth	0.4	1.5	1.6	5.7	0.7	2.7	0.3	1.2
	Clark	--	--	1.3	5.5	2.6	6.2	2.1	4.8
	This study	0.1	0.5	0.5	1.5	0.9	3.0	0.5	1.6
BBP	Wormuth	0.3	1.7	0.8	7.9	0.3	3.7	0.0	1.1
	Clark	--	--	0.5	6.1	1.5	6.1	1.0	4.0
	This study	1.1	2.6	1.8	4.0	2.4	5.8	1.1	2.4
DEHP	Wormuth	1.4	65.7	3.5	19.4	1.5	8.1	0.7	4.6
	Clark	--	--	5.0	27.0	30.0	124	20.0	81.0
	This study	1.6	5.6	12.2	33.8	15.7	46.7	5.4	16.5
DINP	Wormuth	0.004	0.3	21.7	139.7	7.1	66.3	0.2	5.4
	Clark	--	--	0.8	9.9	2.1	8.7	1.3	5.5
	This study	5.1	32.5	20.7	57.4	30.8	93.3	14.3	55.1

^a Ave., average; U.B., upper bound.

^b Wormuth *et al.* (2006). Mean and maximum exposure estimates. Women (female adults; 18 to 80 years); infants (0 to 12 months); toddlers (1 to 3 years); children (4 to 10 years).

^c Clark *et al.* (2011). Median and 95th percentile exposure estimates. Combined male and female adults (20 to 70 years; not shown here); infants (neonates; 0 to 6 months); toddlers (0.5 to 4 years); children (5 to 11 years).

^d This study. Mean and 95th percentile exposure estimates. Women (women of reproductive age; 15 to 44 years); infants (0 to <1 year); toddlers (1 to <3 years); children (3 to 12 years).

3.4 Comparison with Other Studies

Other authors have estimated human exposures to PEs by either modeling or biomonitoring approaches. Clark *et al.* (2011) and Wormuth *et al.* (2006) employed a modeling approach to estimate exposure to various subpopulations. Six PEs were common to the Clark, the Wormuth, and the current study. The metrics used to estimate average and upper bound exposures, and the age ranges of the subpopulations, differed somewhat among the three studies. Clark *et al.* (2011) did not include separate estimates for female adults. Differences in total PE exposure are, in part, due to differences in the methods for estimating dietary exposure because diet is a primary source of PE exposure. Despite these differences, total exposure estimates generally agreed within an order of magnitude.

The CHAP estimated human exposure to PEs using a human biomonitoring approach. Biomonitoring is the most direct method for estimating total PE exposure, and in this case, it can be considered the most reliable (CHAP Report). The CHAP used biomonitoring data from the Study for Future Families (SFF; n=339), which includes biomonitoring data on mothers (prenatal and postnatal data) and their infants (Sathyanarayana *et al.*, 2008a; 2008b). The CHAP also used data from the National Health and Nutritional Survey (NHANES; 2005–2006) to estimate exposures to adult women (n=605). On average, the estimated exposures for individual PEs in the present study were 1.2-fold greater than the biomonitoring results from the SFF data and 2.4-fold greater than the results from the NHANES data (Table E1-26; Figure E1-3). The correlation coefficient between the NHANES results and the current study is 0.93 (Table E1-26). The correlation coefficients between the present study and the SFF results are 0.52 for infants and 0.28 for women.

Table E1-26 Comparison of modeled estimates of total phthalate ester exposure ($\mu\text{g}/\text{kg}\text{-d}$) with estimates from biomonitoring studies.

PE	Study ^a	Women		Infants	
		Ave. ^b	0.95	Ave.	0.95
DEP	This study	18.1	398.0	3.1	14.9
	SFF ^c	NR	NR	NR	NR
	NHANES	3.4	67.7	NR	NR
DBP	This study	0.3	5.7	0.5	1.2
	SFF	0.7	2.4	2.6	10.4
	NHANES	0.8	3.9	NR	NR
DIBP	This study	0.1	0.5	0.5	1.5
	SFF	0.1	0.6	0.4	2.1
	NHANES	0.2	1.1	NR	NR
BBP	This study	1.1	2.6	1.8	4.0
	SFF	0.5	2.4	1.9	8.5
	NHANES	0.3	1.3	NR	NR
DEHP	This study	1.6	5.6	12.2	33.8
	SFF	2.8	19.1	7.6	28.7
	NHANES	3.6	156.2	NR	NR
DINP	This study	5.1	32.5	20.7	57.4
	SFF	0.8	5.4	3.6	18.0
	NHANES	1.1	15.6	NR	NR
DIDP	This study	3.2	12.2	10.0	26.4
	SFF	2.0	21.3	6.1	28.7
	NHANES	1.7	5.6	NR	NR
r²	SFF	0.28		0.52	
	NHANES	0.93		--	

^a Biomonitoring results calculated by the CHAP, based on data from NHANES (adult women; 2005–2006) and the Study for Future Families (SFF).

^b Ave., average, mean (this study) or median (NHANES and SFF); 0.95, 95th percentile; NR, not reported; r², correlation coefficient for this study compared to either NHANES or SFF (average and upper bound exposures combined).

^c Data for women are the average of prenatal and postnatal values.

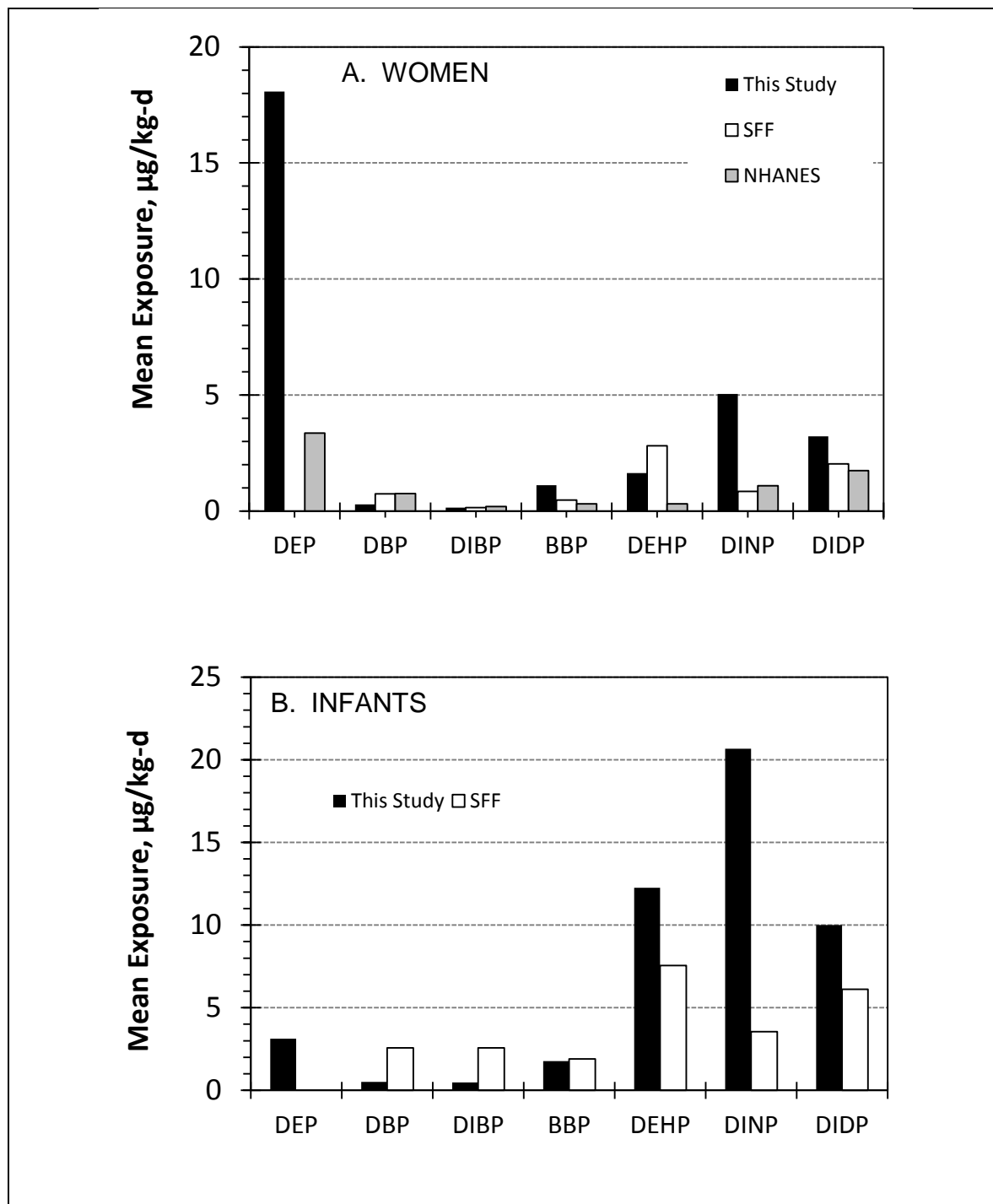


Figure E1-3 Comparison of modeled exposure estimates (this study) with exposures derived from human biomonitoring studies. A. Women; B. Infants. Biomonitoring results from the CHAP report, based on data from NHANES and the Study for Future Families (SFF). SFF data for women are the average of prenatal and postnatal values. Exposure estimates from this study are means; exposures from NHANES and SFF are medians. DEP was not reported for SFF.

4 Discussion

4.1 Uncertainty and Limitations

The modeling approach for estimating human exposure is subject to a number of uncertainties and limitations. This approach is highly dependent on concentration data in environmental media, food, and products, as well as information on consumer behavior. It is also subject to methodological limitations in that it relies on mathematical models and their underlying assumptions.

4.1.1 Scope

4.1.1.1 Phthalate Esters

This report includes exposure estimates for eight PEs of primary interest to the CHAP because there are known human exposures from biomonitoring studies, data for assessing exposure are available, and/or there are concerns about possible health effects in humans (CHAP Report). Approximately 50 PEs are produced at an annual rate of at least 25 million pounds per year, of which half are produced at more than 1 million pounds per year (EPA, 2006). Adequate data for estimating human exposure are not available for most PEs.

Limited data on the presence of phthalate monoesters (metabolites or impurities of PEs) in food (Bradley, 2011) and environmental media (Clark, 2009) are available. Monoesters are not included in this report.

4.1.1.2 Sources

Any consideration of the relative importance of different sources must be made with caution because the quality of the underlying data varies for different sources. Overall, confidence in the dietary, environmental, and mouthing exposure estimates is high. Confidence is lower in exposure estimates from other sources, such as dermal contact with PVC products, aerosol paints, and adult toys.

We attempted to include all relevant sources of PE exposure. We excluded sources for which there is limited direct contact with consumers, such as wall coverings and shower curtains. Indirect exposures from these sources are likely to occur from indoor air and household dust. There have been reports that PEs may occur naturally in marine flora and medicinal plants (reviewed in Patton, 2011). However, most of these studies fail to rule out possible contamination from anthropogenic sources. Even if some PEs are naturally occurring, there is insufficient information to estimate their impact on human exposure.

Exposure from medical devices containing DEHP is not included. These exposures are limited to individuals undergoing invasive medical procedures, such as thoracic surgery and kidney dialysis, and infants in neonatal intensive care units. The medical conditions in these patients may outweigh concerns about possible health effects of DEHP.

The indoor environment contributed significantly to total PE exposure estimates. The ultimate source of PEs in indoor air and house dust probably includes outdoor sources (air and soil). It is also likely that consumer products and home furnishings contribute to indoor sources. As semi-

volatile compounds, PEs may volatilize from PVC products and then adsorb to airborne particles or surfaces (Lioy, 2006; Xu and Little, 2006; Weschler and Nazaroff, 2010). Abraded particles from PVC products also may contribute to PE levels in household dust. Although the dynamics of these processes are not fully understood, it appears likely that much of the indoor exposure presented here ultimately derives from consumer products and personal care products.

Occupational exposures are outside the scope of this report.

4.1.2 Modeling Assumptions

4.1.2.1 Exposure Models

Exposure assessment relies on mathematical models and numerous assumptions. These necessary limitations may either overestimate or underestimate exposure. Accounting for exposures from multiple sources may lead to overlapping exposure estimates, which is double counting of some exposures. For example, PE levels in indoor air most likely include contributions from personal care products and air fresheners. Because separate exposure estimates were also derived for inhalation exposure from personal care products and air fresheners, there is likely some double-counting of these sources of indoor air exposures. In some scenarios (mouthing and handling of toys, dermal contact with child articles and furniture, aerosol paints), we assumed simultaneous exposure to multiple versions of the same product containing different PEs. A more realistic scenario would be to consider each product as having a single PE or else a mixture with roughly the same total PE. Furthermore, six PEs are currently prohibited in toys and child care articles. Thus, PE exposure from teething rings, toys, and child care articles is largely hypothetical.

4.1.2.2 Bioavailability

Although oral toxicokinetic data are available for several phthalates, we assumed a default value of 1.0 for oral, inhalation, and internal (*i.e.*, intravaginal for adult toys) bioavailability (Table E1-7). This was done for several reasons: (1) most of the bioavailability factors used by Wormuth *et al.* (2006) were greater than 0.5 and, thus, have a less than two-fold effect on absorbed dose estimates; (2) because the relevant hazard data are based on applied doses, rather than biologically available doses, it is appropriate to estimate exposure using the same metric; (3) human biomonitoring data are used to estimate applied oral doses in humans. Thus, disregarding the bioavailability adjustment aids in the comparison to biomonitoring results; (4) our approach is conservative in that it tends slightly to overestimate dose.

4.1.2.3 Percutaneous Absorption

Animal data were used to estimate percutaneous absorption rates (Stoltz and El-hawari, 1983; Stoltz *et al.*, 1985; Elsisi *et al.*, 1989). Percutaneous absorption rates may be 5- to 10-fold greater in animals than in adult human skin (Wester and Maibach, 1983). Thus, Wormuth *et al.* (2006) assumed that adult human skin is 7-fold less permeable and infant skin 2-fold less permeable than rodent skin. We did not make any such adjustments because the permeability of human skin varies by anatomic site, and rodent skin may be an adequate model for neonatal skin because neonatal skin is more permeable than adult human skin (Wester and Maibach, 1983).

We used the fraction of applied dose per hour to estimate percutaneous absorption, which is similar to the method used by Wormuth *et al.* (2006). Although this method frequently is used for exposure assessment, it can underestimate percutaneous exposure. Percutaneous absorption rates were obtained from animal studies in which PEs were applied at 5 to 8 mg/cm² (Elsisi *et al.*, 1989). In contrast, for personal care products, such as soap and shampoo, we estimate that DEP contacts the skin at a rate of only 20 to 60 µg/cm². Thus, the dose rate in the animal study was 100-fold greater than the equivalent human exposure. The efficiency of absorption (percentage of the applied dose absorbed) may be greater at lower applied doses (Wester and Maibach, 1983). If the dose rate in the animal study was sufficiently high to saturate the absorption kinetics, then the percutaneous absorption in humans could be greatly underestimated (Kissel, 2011). The only way to assess this would be to obtain dose response data for percutaneous absorption of PEs.

4.1.3 Specific Exposure Scenarios

4.1.3.1 Diet

Two studies were considered for food concentration data (Page and Lacroix, 1995; Bradley, 2011). The Bradley study is the most recent available data, and it is of high quality. Although it represents exposures in the United Kingdom, it is still relevant to U.S. phthalate exposure. The Page and Lacroix study was conducted in Canada between 1985 and 1989. Although it may be more relevant to the United States, it is now decades old and does not include all the PEs of interest; Page and Lacroix did not measure DINP, DIDP, and DNOP.

Established methods are available for estimating dietary exposures from food contaminants. The simplest scheme was selected to categorize food residues (EPA, 2007) because it reduces the occurrence of categories for which no residue data are available. Thus, the simplest scheme provides exposure estimates that are more stable, that is, less sensitive to the choice of food categories (Carlson and Patton, 2012, at Appendix E3). This approach is limited for estimating infant exposure, however, in that it does not include categories for infant formula, baby food, or breast milk. Nevertheless, comparable exposure estimates were derived from other studies with more detailed food categories (Wormuth *et al.*, 2006; Clark *et al.*, 2011; Carlson and Patton, 2012).

A sensitivity analysis for dietary exposures was also performed (Carlson and Patton, 2012). We calculated dietary PE exposures using two data sets (Page and Lacroix, 1995; Bradley, 2011), three sets of food categories and consumption estimates (Wormuth *et al.*, 2006; EPA, 2007;

Clark *et al.*, 2011), and varying assumptions for bioavailability. Generally, the results agreed within a factor of three (Carlson and Patton, 2012).

4.1.3.2 Environmental Media

Quality data were available on PE levels in environmental media, such as indoor and outdoor air, house dust, and soil. However, the best data on soil residues were from a European study (Vikelsøe *et al.*, 1999). The best U.S. data were from a study that measured only DBP and BBP (Morgan *et al.*, 2004). The DBP and BBP levels in the U.S. study were higher than the corresponding levels in the European study. It is possible that the soil exposures estimated here are underestimates for the United States. The data on environmental media are somewhat limited in that several studies did not include all of the PEs of interest, especially DIBP, DNOP, DINP, and DIDP.

4.1.3.3 Mouthing of Teethers and Toys

The method for measuring plasticizer migration into simulated saliva was specifically developed and validated for the purpose of estimating children's exposure to phthalates from mouthing PVC articles (Simoneau *et al.*, 2001; CPSC, 2002; Babich *et al.*, 2004). The laboratory method was compared to a study with adult volunteers who mouthed PVC disks. Saliva was collected and analyzed to measure the PE migration rate *in vivo*. Migration data were available for only two PEs: DINP and DEHP (Chen, 2002). Exposures resulting from mouthing products containing DIDP, DNOP, and other PEs could not be evaluated.

Mouthing durations are from an observational study of children's mouthing activity (Greene, 2002). Mouthing duration depends on the child's age and the type of object mouthed. The category "all soft plastic articles except pacifiers" was used to estimate children's exposure from mouthing PVC articles. This category includes articles such as teethers, toys, rattles, cups, and spoons. Pacifiers are not included in this category because they are generally made with natural rubber or silicone (CPSC, 2002).

Products in the "all soft plastic articles except pacifiers" category are not necessarily made with PVC. About 35% of the soft plastic toys and less than 10% of the soft plastic child care articles tested by the CPSC contained PVC (Table E1-3). Toys and child care articles are also made from other plastics, wood, textiles, and metal. Because six PEs are currently prohibited from use in toys and child care articles, the use of mouthing durations for the category "all soft plastic articles except pacifiers" may be considered a reasonable upper bound estimate for children's exposure to PEs from mouthing PVC children's products.

4.1.3.4 Drugs and Dietary Supplements

Data on prescription drugs containing DEP were provided by the U.S. FDA (Jacobs, 2011). From these data, it was estimated that less than 5% of the population uses prescription drugs containing DEP. The highly skewed nature of the exposure distribution suggests that the mean exposure estimate (population mean) overestimates the typical (median) exposure. On the other hand, users can have very high DEP exposures. We estimate the maximum individual exposure from prescription drugs to be about 1,800 µg/kg-d in women and 5,000 µg/kg-d in toddlers. It should

be noted that DEP does not induce the same developmental and reproductive effects in animals as some PEs, although the effects in humans are uncertain (reviewed in the CHAP report).

Adequate information on PE exposure from nonprescription drugs and dietary supplements was not available. However, DEP and other PEs are known to be present in some of these products (Hauser *et al.*, 2004; Hernandez-Diaz *et al.*, 2009; Kelley *et al.*, 2012). Maximum PE exposures from these products are as high as 16.8 mg DEP and 48 mg DBP (Kelley *et al.*, 2012), or about 220 µg/kg-d DEP and 640 µg/kg-d DBP in adults. The lack of exposure estimates for nonprescription drugs and dietary supplements may be a significant data gap.

4.1.3.5 Dermal Contact with PVC Products

Consumers regularly come into direct dermal contact with PVC products, such as wall coverings, flooring, vinyl upholstery, protective gloves, child care products (play pens, changing pads), toys, shower curtains, and rain wear. Adequate data on the presence of PEs in consumer products and a validated methodology for estimating these exposures are not available. Not all products in these categories are made with PVC or PEs. We estimated exposure from these scenarios, as described in Wormuth *et al.* (2006). Wormuth's method was based on a study in which a PVC film containing 40% ¹⁴C-DEHP was placed on the backs of rats and percutaneous absorption of the DEHP was measured (Deisinger *et al.*, 1998). This method is limited in that DEHP migration/absorption was measured at only one DEHP concentration; thus, it does not account for differences in migration due to different PE concentrations. To adjust for the lack of data for other PEs, Wormuth multiplied the DEHP migration/absorption rate by the ratio of the percutaneous absorption rate of the other PEs to that of DEHP (equation 5). This adjustment only accounts for differences in percutaneous absorption between PEs, not for differences in migration from the PVC film.

Wormuth applied this approach to protective gloves. A similar approach was used in this report for other products, including toys (dermal exposure), child care articles, and vinyl upholstery. This was done to satisfy the mandate for the CHAP report to include toys and child care articles, and all routes of exposure. This required a number of assumptions, such as the skin surface area in contact with the PVC product, the contact duration, and frequency of contact. It was observed that, depending on the assumptions chosen and the number of products included, estimated exposures from these scenarios could equal or exceed the modeled exposures from food and total exposures estimated from biomonitoring studies. Because biomonitoring studies are considered the most reliable estimates of total PE exposure, it was concluded that the approach for assessing exposures from contact with PVC products likely results in overestimates of dermal exposure.

There are several possible reasons Wormuth's method might overestimate exposure. Deisinger *et al.* (1998) measured the average percutaneous absorption of DEHP from a vinyl film over a period of seven days. Consumer contact with PVC products tends to be brief and episodic. The efficiency of PE transfer during brief exposures is unknown. Percutaneous absorption generally has a lag time on the order of an hour before steady-state absorption kinetics is achieved. Vinyl flooring may be covered with a wear layer of inorganic oxides and a polyurethane layer for shine. These layers may limit the migration of PEs from vinyl flooring. Also, percutaneous absorption through the sole of the foot, which has thick skin, may be limited.

We conclude that this scenario (dermal contact with PVC products) provides highly uncertain exposure estimates. It was included to satisfy the CHAP's mandate to include toys and child care articles, and all relevant routes and sources of exposure. Data on PE use in consumer products and an improved methodology are needed to improve estimates for this scenario.

4.1.3.6 Aerosol Paints

Data on consumer use of aerosol paints by the general population were not available. The available data on PE concentrations in these products (NLM, 2012) suggest that few of these contain PEs. The average (population average) exposure estimates presented here may overestimate the average exposure. However, the potential exposure to users of these products and others present in the home is high. We estimate a maximum individual exposure of about 100 µg/kg-d for frequent aerosol paint users.

4.1.3.7 Adult Toys

This scenario was included because of its relevance to women of reproductive age and because the fetus is probably the most sensitive life stage for potential adverse effects from phthalate exposure. Thus, the CHAP is concerned about PE exposures to women of reproductive age. Data for estimating exposure are available from one study (Nilsson *et al.*, 2006), but validated methodologies are not available. We assumed conservatively that 100% of PE migrating from the product would be absorbed through the vaginal (or rectal) epithelium. Therefore, the exposure estimates for this scenario are highly uncertain. Although estimated average exposures were minimal, the use of these products with an oil-based lubricant led to higher migration rates and consequently larger exposures (Nilsson *et al.*, 2006). A maximum exposure of 27 µg/kg-d DEHP (highest migration rate and frequency of use) was estimated for this scenario.

4.2 Comparison with Other Studies

Overall, the exposure estimates in this study are in general agreement (within an order of magnitude) of the exposure estimates from two other studies (Wormuth *et al.*, 2006; Clark *et al.*, 2011). This is noteworthy, considering the differences in methodologies among these three studies. Wormuth included a number of consumer scenarios, including mouthing toys and personal care product use. Wormuth also included a detailed assessment of dietary exposures. The primary limitation of the Wormuth study for the present purpose is that it presents exposure estimates specific to Europe. Clark included a detailed assessment of dietary and environmental exposures, but did not include consumer products. The present study attempted to include a number of household sources, including toys, PVC products, personal care products, and prescription drugs. A more simplified scheme for assessing dietary exposures was used.

The present study also agreed quite well with total exposure estimates from human biomonitoring studies. This is encouraging because biomonitoring probably provides the most reliable estimates of total exposure. However, the appearance of concordance could also be due to compensating overestimates and underestimates in the present study.

The general agreement among the three modeling studies and two biomonitoring studies tends to increase overall confidence in the conclusions of this study.

4.3 Regulatory Considerations

Considering PE sources by jurisdiction, most exposures are from sources under the purview of the FDA: food, prescription drugs, and personal care products (cosmetics). Food packaging and processing materials are suspected of being the major sources of PEs in food (Rudel *et al.*, 2011). However, food can come into contact with PEs at any point between the farm and dinner table. The relative importance of food contact articles and other sources has not been elucidated.

DEP and DEHP are found in certain prescription drugs and medical devices, respectively. Exposure from these sources affects a small population with overriding medical concerns. The situation regarding nonprescription drugs and dietary supplements is less clear. FDA has issued a draft guidance document on limiting the use of PEs in drugs (FDA, 2012).

The use of DEP and other PEs in personal care products has declined over time due to voluntary reformulation by manufacturers (compare Hubinger and Havery, 2006; with Hubinger, 2010).

The U.S. Environmental Protection Agency (EPA) has jurisdiction over production and importation of chemical substances. EPA is in the process of assessing cumulative health risks from PE exposure.

The CPSC has jurisdiction over teething and toys, child care articles, and other consumer products, such as home furnishings, air fresheners, and aerosol paints. The CPSIA permanently prohibits the use of DBP, BBP, and DEHP in child care articles and toys, and prohibits the use of DNOP, DINP, and DIDP on an interim basis in child care articles and toys that can be placed in a child's mouth. The CHAP on phthalates and phthalate substitutes was convened to advise the CPSC on whether any additional phthalates or phthalate substitutes should be prohibited in toys and child care articles.

4.4 Data Gaps

Modeling exposures to PEs is a data-intensive process. Although recent, high-quality data on PE levels in food are available from the United Kingdom, data on the U.S. food supply, including data on infant formula, baby food, and breast milk, are lacking. Similarly, data on environmental sources of PEs are generally more abundant in Europe. Studies of environmental media do not always include DIBP, DNOP, DINP, and DIDP. Except for mouthing of teething and toys, there is a general lack of data on PE levels in consumer products and child care articles. Standardized methodologies for assessing exposures from many consumer products are also lacking. Some of the methods used here, for example, dermal contact with PVC articles, have not been validated, by comparison with more direct exposure measures. Additional data on percutaneous absorption are needed to estimate dermal exposure accurately.

4.5 Conclusions

Diet is the primary source of exposure to DIBP, BBP, DNOP, DEHP, DINP, and DIDP. Personal care products are the primary sources of DEP and DBP exposure, while air fresheners and certain prescription drugs contribute to total DEP exposure. Exposures to DIBP, BBP, and DNOP may also arise from a variety of sources, including diet, the environment, and consumer products.

In infants, mouthing and handling toys, and contact with child care articles, contributes to the total exposure to higher molecular weight PEs. The mouthing of soft plastic products accounts for up to 11% of total DINP exposure in this population. Dermal contact with toys and child care articles may contribute up to an additional 18%. In infants, about 65% of DINP and more than 90% of DIDP are estimated to be from the diet.

5 Supplemental Data

Table E1-S1 Estimated phthalate ester (PE) exposure ($\mu\text{g}/\text{kg}\text{-d}$) by individual exposure scenario for women.

Source	DEP		DBP		DIBP		BBP		DNOP		DEHP		DINP		DIDP	
	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95
Total	1.8 E+01	4.0 E+02	2.9 E-01	5.7 E+00	1.5 E-01	5.0 E-01	1.1 E+00	2.6 E+00	1.7 E-01	2.1 E+01	1.6 E+00	5.6 E+00	5.1 E+00	3.3 E+01	3.2 E+00	1.2 E+01
Diet	9.3 E-02	3.6 E-01	7.8 E-02	2.3 E-01	1.3 E-01	4.6 E-01	1.6 E-01	2.5 E-01	1.3 E-01	3.6 E-01	1.4 E+00	4.9 E+00	4.8 E+00	1.5 E+01	3.2 E+00	9.3 E+00
Drugs^a	1.4 E+01	3.7 E+02														
Personal care, dermal																
Shampoo	1.2 E-02	6.5 E-02														
Soap / body wash	2.3 E-02	4.1 E-02														
Lotion	5.0 E-02	1.8 E-01														
Deodorant	7.4 E-01	1.9 E+01														
Perfume	2.8 E+00	6.2 E+00														
Nail polish	3.4 E-03	1.5 E-02	1.7 E-01	5.4 E+00												
Hair spray	4.7 E-02	1.4 E-01														
Personal care, inhalation^b																
Deodorant	5.1 E-02	1.3 E+00														
Perfume	2.0 E-01	4.2 E-01														

Source	DEP		DBP		DIBP		BBP		DNOP		DEHP		DINP		DIDP	
	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95
Hair spray	6.2 E-03	1.8 E-02														
Dermal, PVC^c																
Toys^d									8.0 E-03	8.0 E-03	8.0 E-03	8.0 E-03	6.7 E-03	6.7 E-03	1.1 E-03	1.1 E-03
Furniture^e									0.0 E+00	2.0 E+01			0.0 E+00	1.7 E+01	0.0 E+00	2.9 E+00
Gloves							2.3 E-01	2.3 E-01	3.3 E-02	3.3 E-02	3.3 E-02	3.3 E-02	2.8 E-02	2.8 E-02	4.7 E-03	4.7 E-03
Household-dermal^e																
Paint/lacquer							5.4 E-04	1.5 E-03					2.5 E-05	0.0 E+00		
Adhesive							1.0 E-03	3.6 E-03								
Household, inhalation^f																
Air freshener, spray^b	1.1 E-01	3.6 E-01	1.6 E-05	2.0 E-05												
Air freshener, liquid	1.5 E-02	4.0 E-02	9.2 E-06	2.4 E-05	6.8 E-06	9.8 E-06										
Paint, spray^b							6.6 E-01	2.0 E+00					1.5 E-01	3.1 E-01		
Indirect ingestion																
Dust	3.4 E-03	4.3 E-03	1.1 E-02	1.8 E-02	1.2 E-03	2.0 E-03	5.0 E-02	1.1 E-01			2.0 E-01	3.4 E-01	5.2 E-02	4.0 E-01	1.4 E-02	4.4 E-02
Soil			9.3 E-06	4.3 E-05			1.6 E-06	6.9 E-06	3.5 E-06	1.1 E-05	7.2 E-05	3.1 E-04	2.1 E-05	8.1 E-05		

Source	DEP		DBP		DIBP		BBP		DNOP		DEHP		DINP		DIDP	
	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95
Inhalation, air																
Indoor air	9.5 E-02	2.4 E-01	3.3 E-02	7.4 E-02	1.8 E-02	4.4 E-02	3.8 E-03	8.9 E-03	5.9 E-05	5.9 E-05	1.5 E-02	2.9 E-02				
Outdoor air	1.4 E-03	3.8 E-03	8.4 E-05	3.6 E-04	8.6 E-05	2.6 E-04	7.2 E-05	1.2 E-04	8.4 E-06	8.4 E-06	4.8 E-04	2.9 E-03				
Adult toys^g									3.8 E-04	8.0 E-02	1.9 E-04	2.6 E-01				

^aAverage exposure is the population average. 95th percentile is the average user.

^bIncludes exposure from the breathing zone during application and subsequent exposure to room air.

^c95th percentile estimate not available.

^dExposure is conditional on the presence of phthalates in toys. Six phthalates are currently prohibited.

^ePrevalence of vinyl-covered or imitation leather furniture is unknown. Assume average user is not exposed; upper bound is exposed.

^fUse information is available for “users” only. 95th percentile PE concentration is 0; 95th % for frequency of use was used to estimate 95th percentile exposure.

^gUpper bound DEHP exposure is with an oil-based lubricant.

Table E1-S2 Estimated phthalate ester exposure (µg/kg-d) by individual exposure scenario for infants.

Source	DEP		DBP		DIBP		BBP		DNOP		DEHP		DINP		DIDP	
	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95
Total	3.1 E+00	1.5 E+01	5.1 E-01	1.2 E+00	4.8 E-01	1.5 E+00	1.8 E+00	4.0 E+00	4.4 E+00	9.6 E+00	1.2 E+01	3.4 E+01	2.1 E+01	5.7 E+01	1.0 E+01	2.6 E+01
Diet	3.0 E-01	1.2 E+00	2.0 E-01	5.3 E-01	3.5 E-01	1.2 E+00	5.5 E-01	6.7 E-01	3.8 E-01	9.8 E-01	5.0 E+00	1.8 E+01	1.4 E+01	3.6 E+01	9.3 E+00	2.5 E+01
Drugs^a	0.0 E+00	0.0 E+00	0.0 E+00	0.0 E+00	0.0 E+00	0.0 E+00	0.0 E+00	0.0 E+00	0.0 E+00	0.0 E+00	0.0 E+00	0.0 E+00	0.0 E+00	0.0 E+00	0.0 E+00	0.0 E+00
Teethers & toys^b																
Mouthing^c											7.3 E-01	2.9 E+00	2.3 E+00	9.2 E+00		
Dermal											4.0 E-01	4.0 E-01	3.3 E-01	3.3 E-01		
Personal care, dermal																
Body wash/ shampoo	8.8 E-03	4.8 E-02														
Lotion	1.5 E+00	8.2 E+00														
Personal care, inhalation^d																
Perfume	4.8 E-02	1.0 E-01														
Deodorant	1.1 E-01	2.9 E+00														
Hair spray	3.6 E-01	3.6 E-01														
Dermal, PVC^b																
Changing pad									1.7 E+00	1.7 E+00	1.7 E+00	1.7 E+00	1.4 E+00	1.4 E+00	2.4 E-01	2.4 E-01
Play pen									2.4 E+00	7.0 E+00	2.4 E+00	7.0 E+00	2.0 E+00	5.9 E+00	3.4 E-01	9.9 E-01

Source	DEP		DBP		DIBP		BBP		DNOP		DEHP		DINP		DIDP	
	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95
Indirect ingestion																
Dust	3.3 E-02	4.2 E-02	1.1 E-01	1.7 E-01	1.1 E-02	1.9 E-02	4.8 E-01	1.1 E+00			1.9 E+00	3.3 E+00	5.0 E-01	3.8 E+00	1.3 E-01	4.2 E-01
Soil			1.3 E-04	6.3 E-04			2.3 E-05	1.0 E-04	5.0 E-05	1.6 E-04	1.0 E-03	4.4 E-03	3.0 E-04	1.2 E-03		
Inhalation																
Indoor air	6.0 E-01	1.5 E+00	2.1 E-01	4.7 E-01	1.1 E-01	2.8 E-01	2.4 E-02	5.6 E-02	3.7 E-04	3.7 E-04	9.4 E-02	1.8 E-01				
Outdoor air	2.8 E-03	7.4 E-03	1.6 E-04	6.9 E-04	1.7 E-04	5.1 E-04	1.4 E-04	2.2 E-04	1.6 E-05	1.6 E-05	9.2 E-04	5.5 E-03				
Air freshener, spray^d	1.0 E-01	3.2 E-01	6.4 E-05	8.0 E-05												
Air freshener, liquid^d	5.9 E-02	1.6 E-01	3.6 E-05	9.5 E-05	2.7 E-05	3.9 E-05										
Paint, spray^{d,e}							7.3 E-01	2.2 E+00					3.0 E-01	8.9 E-01		

^a Drugs were not included for infants because data specific for children 0 to 1 year old were not available.

^b Assumes that phthalate esters are present in these products. Currently six phthalates are prohibited.

^c 95th percentile exposure is based on the 95th percentile mouthing duration.

^d Incidental exposure from product use by others in the home.

^e Prevalence of phthalate esters in these products is unknown but is believed to be low. Consumer use information is available for users only.

Assumes that the average exposure is zero; upper bound exposure is for the average user.

Table E1-S3 Estimated phthalate ester exposure (µg/kg-d) by individual exposure scenario for toddlers.

Source	DEP		DBP		DIBP		BBP		DNOP		DEHP		DINP		DIDP	
	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95
Total	2.8 E+00	2.2 E+03	6.9 E-01	1.6 E+00	8.6 E-01	3.0 E+00	2.4 E+00	5.8 E+00	5.4 E+00	1.6 E+01	1.6 E+01	4.7 E+01	3.1 E+01	9.3 E+01	1.7 E+01	4.8 E+01
Diet	6.7 E-01	2.7 E+00	3.6 E-01	9.8 E-01	7. 3E-01	2.7 E+00	6.4 E-01	1.1 E+00	6.1 E-01	1.6 E+00	7.6 E+00	2.6 E+01	2.4 E+01	6.9 E+01	1.6 E+01	4.5 E+01
Drugs^a	5.3 E-01	2.2 E+03														
Teethers & toys^b																
Mouthing^c											4.2 E-01	1.7 E+00	1.3 E+00	5.2 E+00		
Dermal											4.0 E-01	4.0 E-01	3.3 E-01	3.3 E-01		
Personal care, dermal																
Shampoo	7.2 E-05	3.9 E-04														
Soap	1.1 E-02	2.1 E-02														
Lotion	9.1 E-02	5.0 E-01														
Personal care, inhalation^d																
Perfume	4.4 E-01	9.5 E-01														
Deodorant	1.1 E-01	3.0 E+00														
Hair spray	3.8 E-02	1.1 E-01														
Dermal, PVC^b																
Changing pad									1.3 E+00	1.3 E+00	1.3 E+00	1.3 E+00	1.1 E+00	1.1 E+00	1.8 E-01	1.8 E-01

Source	DEP		DBP		DIBP		BBP		DNOP		DEHP		DINP		DIDP	
	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95
Play pen									3.6 E+00	1.3 E+01	3.6 E+00	1.3 E+01	3.0 E+00	1.1 E+01	5.1 E-01	1.9 E+00
Indirect ingestion																
Dust	4.1 E-02	5.2 E-02	1.3 E-01	2.1 E-01	1.4 E-02	2.4 E-02	6.0 E-01	1.3 E+00			2.4 E+00	4.1 E+00	6.2 E-01	4.8 E+00	1.6 E-01	5.3 E-01
Soil			1.4 E-04	6.6 E-04			2.4 E-05	1.1 E-04	5.2 E-05	1.7 E-04	1.1 E-03	4.6 E-03	3.1 E-04	1.2 E-03		
Inhalation																
Indoor air	5.8 E-01	1.4 E+00	2.0 E-01	4.5 E-01	1.1 E-01	2.7 E-01	2.3 E-02	5.4 E-02	3.6 E-04	3.6 E-04	9.0 E-02	1.7 E-01				
Outdoor air	2.7 E-03	7.1 E-03	1.6 E-04	6.7 E-04	1.6 E-04	4.9 E-04	1.3 E-04	2.1 E-04	1.6 E-05	1.6 E-05	8.9 E-04	5.3 E-03				
Air freshener, spray^d	1.5 E-01	4.9 E-01	9.9 E-05	1.2 E-04												
Air freshener, liquid^d	9.1 E-02	2.5 E-01	5.6 E-05	1.5 E-04	4.1 E-05	6.0 E-05										
Paint, spray^{d,e}							1.1 E+00	3.4 E+00					4.6 E-01	1.4 E+00		

^a Drugs were not included for infants because data specific for children 0 to 1 year old were not available.

^b Assumes that phthalate esters are present in these products. Currently six phthalates are prohibited.

^c 95th percentile exposure is based on the 95th percentile mouthing duration.

^d Incidental exposure from product use by others in the home.

^e Prevalence of phthalate esters in these products is unknown but is believed to be low. Consumer use information is available for users only. Assumes that the average exposure is zero; upper bound exposure is for the average user.

Table E1-S4 Estimated phthalate ester exposure (µg/kg-d) by individual exposure scenario for children.

Source	DEP		DBP		DIBP		BBP		DNOP		DEHP		DINP		DIDP	
	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95
Total	2.8 E+00	1.1 E+03	5.5 E-01	7.4 E+00	4.5 E-01	1.6 E+00	1.1 E+00	2.4 E+00	5.2 E-01	1.5 E+01	5.4 E+00	1.7 E+01	1.4 E+01	5.5 E+01	9.1 E+00	2.8 E+01
Diet	3.4 E-01	1.4 E+00	2.1 E-01	5.8 E-01	4.1 E-01	1.5 E+00	3.9 E-01	6.4 E-01	3.5 E-01	9.2 E-01	4.2 E+00	1.5 E+01	1.4 E+01	4.0 E+01	9.0 E+00	2.6 E+01
Drugs^a	1.4 E+00	1.1 E+03														
Personal care, dermal																
Shampoo	2.8 E-03	1.5 E-02														
Soap	5.6 E-03	1.0 E-02														
Lotion/cream	1.2 E-02	4.4 E-02														
Deodorant	1.8 E-01	4.7 E+00														
Perfume	2.7 E-01	6.0 E-01														
Nail polish	4.1 E-04	1.8 E-03	2.1 E-01	6.6 E+00												
Hair spray	5.7 E-03	1.7 E-02														
Personal care, inhalation^b																
Deodorant	7.0 E-02	7.0 E-02														
Perfume	1.3 E-01	2.9 E-01														
Hair spray	5.8 E-03	1.7 E-02														
Dermal, PVC^c																

Source	DEP		DBP		DIBP		BBP		DNOP		DEHP		DINP		DIDP	
	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95	ave.	0.95
Toys^d									1.6 E-01	1.6 E-01	1.6 E-01	1.6 E-01	1.4 E-01	1.4 E-01	2.3 E-02	2.3 E-02
Furniture^e									0.0 E+00	1.4 E+01			0.0 E+00	1.2 E+01	0.0 E+00	2.0 E+00
Indirect ingestion																
Dust	1.7 E-02	2.1 E-02	5.3 E-02	8.6 E-02	5.7 E-03	9.8 E-03	2.4 E-01	5.4 E-01			9.9 E-01	1.7 E+00	2.5 E-01	2.0 E+00	6.6 E-02	2.2 E-01
Soil			9.8 E-06	4.2 E-05			4.4 E-04	1.9 E-03	2.1 E-05	6.9 E-05	4.4 E-04	1.9 E-03	1.3 E-04	5.0 E-04		
Inhalation																
Indoor air	2.1 E-01	5.3 E-01	7.4 E-02	1.7 E-01	4.1 E-02	9.9 E-02	8.5 E-03	2.0 E-02	1.3 E-04	1.3 E-04	3.4 E-02	6.5 E-02				
Outdoor air	2.1 E-03	5.5 E-03	1.2 E-04	5.2 E-04	1.2 E-04	3.8 E-04	1.0 E-04	1.7 E-04	1.2 E-05	1.2 E-05	6.9 E-04	4.1 E-03				
Air freshener, spray^b	5.7 E-02	1.8 E-01	3.7 E-05	4.6 E-05												
Air freshener, liquid^b	3.4 E-02	9.1 E-02	2.1 E-05	5.4 E-05	1.5 E-05	2.2 E-05										
Paint, spray^{b,f}							4.2 E-01	1.2 E+00					1.7 E-01	5.1 E-01		

^aAverage exposure is the population average. 95th percentile is the average user.

^bIncludes exposure from the breathing zone during application and subsequent exposure to room air.

^c95th percentile estimate not available.

^dExposure is conditional on the presence of phthalates in toys. Six phthalates are currently prohibited.

^ePrevalence of vinyl-covered or imitation leather furniture is unknown. Assumes average user is not exposed; upper bound is exposed.

^fUse information is available for “users” only. 95th percentile PE concentration is 0; 95th percent for frequency of use was used to estimate 95th percentile exposure.

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