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Supplemental Material

Erratum: "Identifying and Prioritizing Chemicals with Uncertain Burden of Exposure: Opportunities for Biomonitoring and Health-Related Research"

Edo D. Pellizzari, Tracey J. Woodruff, Rebecca R. Boyles, Kurunthachalam Kannan, Paloma I. Beamer, Jessie P. Buckley, Aolin Wang, Yeyi Zhu, and Deborah H. Bennett (Environmental influences on Child Health Outcomes)

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Abbreviations

Acronym	Name	Acronym	Name
µg	Micrograms	MOCA	4,4´-Methylenebis(2-chloroaniline)
2NPPD	2-Nitro-1,4-phenylenediamine	MOXA	Metolachlor oxanilic acid
3,5-DCA	3,5-dichloroaniline	ND	Not detected
AFR	Alternate Flame Retardant	ng	nanogram
AhR	Aryl hydrocarbon receptor	NOEL	No-observable-effect-level
AMPA	Aminomethylphosphonic acid	OA	Oxanilic acid
ATBC	Acetyl tributyl citrate	ODA	4,4'-Oxydianiline
ATMAC C18	N, N, N-Trimethyloctadecan-1-aminium chloride (stearyltrimethylammonium, or	OTC	Over-the-counter
ATSDR	Toxic Substances and Disease Registry	OTSA	o-Toluene sulfonamide
BAC C16	Benzylhexadecyldimethylammonium chloride	PBBz	Pentabromobenzene
BAC C18	N, N-Dimethyl-N-benzyl-N-octadecylammonium chloride	PBEB	Pentabromoethylbenzene
BADGE	Bisphenol A diglycidyl ether	PBO	Piperonyl butoxide
BATE	2-Bromoallyl 2,4,6-tribromophenyl ether	PBT	Pentabromotoluene
BBOEP	bis(2-butoxyethyl) phosphate	PFBA	Perfluorobutanoic acid
BEHP	Bis(2-ethylhexyl) phosphate	PFHpS	Perfluoroheptane sulfonic acid
BEH-TEBP	Bis(2-ethylhexyl) tetrabromophthalate	PFHxA	Perfluorohexanoic acid
BHT	2,6-di-tert-butyl-4-methylphenol,	PFHxDA	Perfluorohexadecanoic acid
BPA	Bisphenol A	PFODA	Perfluorooctadecanoic acid
BPAF	Bisphenol AF	PFPeA	Perfluoropentanoic acid
BTBPE	1,2-Bis(2,4,6-tribromophenoxy) ethane	PFPeS	Perfluoropentane sulfonic acid
bw	Body weight	pg	Picograms
CALEPA	California Environmental Protection Agency	pTBX	2,3,5,6-Tetrabromo-*p*-xylene
CASRN	Chemical Abstract Services Registry Number	PTMS/PTMSA	2-Aminotoluene-5-methylbenzenesulphonic acid
CRT	Creatinine	QSAR	Quantitative Structure Activity Relationship
DBA	Di-butyl adipate	RXR	retinoid X receptor
DBDPE	Decabromobiphenyl ethane	SHBG	hormone-binding globulin
DBS	Dibromostyrene	SVOC	Semi-volatile organic chemicals
DBS	Di-butyl sebacate	TBA	2,4,6-Tribromoaniline
DDMAC	Didecyldimethylammonium chloride	TBBA	2,3,4,5,-Tetrabromo benzoic acid
DEHA	Bis(2-ethylhexyl) adipate	TBBP-A	3,3’,5,5’-Tetrabromobisphenol A
DEHT	Bis(2-ethylhexyl)-1, 4-terephthalate	TBCT	Tetrabromo-*o*-chlorotoluene
DHP	Dimethyl hydrogen phosphite	TBE	1,2-Bis(2,4,6-tribromophenoxy) ethane
DMPP	Dimethyl propyl phosphonate	TBOEP	Tris(2-butoxyethyl) phosphate
DMPT	N,N-Dimethyl-p-toluidine or	TBP	Tris (2,3-dibromopropyl) phosphate
DNMPP	Dimethyl N-methylolphosphonopropionamide	TBPA	2,3,4,5-tetrabromo phthalic acid
DOS	Dioctyl succinate	TBPA	Tetrabromophthalic anhydride

Acronym	Name	Acronym	Name
DOTP	Dioctyl terephthalate	TBPD	Tetrabromophthalate diol
ECHA	European Chemicals Agency	TBP-DBPE	2,3-Dibromopropyl 2,4,6-tribromophenyl ether
ECHO	Environmental influences on Children Health Outcomes	TBZ	Thiabendazole
EHDPP	2-Ethylhexyl diphenyl phosphate	TCEP	Tris(2-chloroethyl) phosphate
ESA	Ethane sulfonic acid	TCIPP	Tris(2-chloro-iso-propyl) phosphate
ETBP	Ethylene bis(tetrabromo) phthalmide	TDCnPP	Tris(2,3-dichloropropyl) phosphate
FDA	Food and Drug Administration	TDCnPP	Diquanidine hydrogen phosphate (DHP).
g	Gram	TEB-COOH	Carboxyl tebuconazole
GM	Geometric mean	TEB-OH	Hydroxyl tebuconazole
HBBz	Hexabromobenzene	TEHP	Tris(2-ethylhexyl) phosphate
HBCD	Hexabromocyclododecane	TEP	Triethyl phosphate
HTP	High-throughput	TETM	Tri-2-ethylhexyl trimellitate
IPCS-INCHEM	International Programme on Chemical Safety – In Chemistry	THPI	Tetrahydrophthalimide
kg	Kilogram	THPI	Tetrahydrophthalimide
LOD	Limit of detection	TraTBA	2,2’,6,6’-Tetrachlorobisphenol A
m^3	Cubic meter	TrCBA	3,3’,5-Trichlorobisphenol A
MBC	Carbendazim	TTBNPP	Tris-(tribromoneopentyl) phosphate
MBOT	4,4’-Methylenedi-o-toluidine or	TTCA	Thiazolidine-2-thione-4-carboxylic acid
MDA	Methylenedianiline	TXIB	2,2,4-Trimethyl 1,3-pentanediol monoisobutyrate
MEHA	Mono-2-ethylhexyl adipate	USDA	United States Department of Agriculture
MEHHA	mono-2-ethylhydroxyhexyl adipate	USEPA	United States Environmental Protection Agency
MEOHA	mono-2-ethyloxohexyl adipate	V6	2,2-Bis(chloromethyl) propane-1,3-diyltetrakis(2-chloroethyl) bisphosphate
MESA	Metolachlor ethane sulfonic acid	WHO	World Health Organization
mg	Milligram	α-DBE-DBCH	*alpha*-Tetrabromoethylcyclohexane
mL	Milliliter	β-DBE-DBCH	*beta*-Tetrabromoethylcyclohexane
MMA	2-Methoxy-5-methylaniline		

Table S1. Consumer Product Categories Surveyed in USEPA CPCat (U.S. EPA 2014)

CPCat Term	No. of entries with/without displayed chemical structures	Percent with displayed structures
Air fresheners	142/70	67
Anti-wrinkle	19/8	70
Arts crafts	619/270	70
Baby use	249/134	65
Bath	450/120	79
Bedding	43/17	72
Beverage	520/474	52
Body	298/342	47
Carpet	93/50	65
Child use	81/19	81
Childcare	897/638	58
Conditioner	93/229	29
Consumer use	3301/10739	24
Cleaning, washing	4911/4488	46
Cooking oils	82/59	58
Cosmetics	3941/9297	30
Cream	203/369	35
Deodorant	130/68	66
Detergent	625/1140	35
Eye	133/346	28
Face	547/3813	13
Lotion	65/35	65
Mouthing	3/1	75
Personal use	6051/14719	41
Shampoo	152/383	28
Skin	113/53	68
Soap	544/1059	34
Antimicrobial	751/367	67
Antifungal	35/7	83
Bactericide	31/3	91
Biocide	1017/729	58
Disinfection by-products	99/1	99
Flame retardant	90/477	16
Fungicide	122/51	71
Herbicide	138/34	19
Insecticide	49/35	58
PAHs	18/0	100
Parabens	6/0	100
PBDEs	7/2	78
PCBs	93/7	93
PCNs	4/6	40
Pesticide	161/339	32
Pet	276/1493	16
Phthalates	39/7	85
Rodenticide	13/8	62
Totals:	**27,261 / 52,506**	**36**

Table S2. Literature Search Terms

Search Terms for Identifying Chemicals in Environmental Media
[SVOC] AND [air]; [SVOC] AND [indoor dust]; [SVOC] AND [drinking water]; [SVOC] AND [ground water]; [SVOC] AND [food]; [chemical class] AND [air]; [chemical class] AND [house dust]; [chemical class] AND [drinking water] OR [ground water]; [chemical class] AND [food]. (SVOC is an acronym for semi-volatile organic compound.) Chemical classes included: pesticides, environmental phenols, parabens, perfluoroalkyl substances, phthalates, polynuclear aromatic hydrocarbons, volatile organic compounds, polychlorinated biphenyls, polybrominated diphenyl ethers, and metals.
Search Terms for Environmental and Biomonitoring Data[a]
[chemical name] OR [CASRN] OR [“chemical’s acronym”] AND [human urine]; [chemical name] OR [CASRN] OR [“chemical’s acronym”] AND [human serum]; [chemical name] OR [CASRN] OR [“chemical’s acronym”] AND [breast milk]; [chemical name] OR [CASRN] OR [“chemical’s acronym”] AND [air]; [chemical name] OR [CASRN] OR [“chemical’s acronym”] AND [dust]; [chemical name] OR [CASRN] OR [“chemical’s acronym”] AND [water]; [chemical name] OR [CASRN] OR [“chemical’s acronym”] AND [food]
Search Terms for Health-effects and Toxicity Data[a]
[chemical name] OR [CASRN] AND [toxicity]; [chemical name] OR [CASRN] AND [endocrine disruption]; [chemical name] OR [CASRN] AND [developmental toxicity]; [chemical name] OR [CASRN] AND [reproductive toxicity]; [chemical name] OR [CASRN] AND [neurotoxicity].

[a]These search terms were applied to the 155 chemicals prioritized and listed in Tables 4,5, and 6.

Table S3. Chemicals recommended for biomonitoring

In this table we present summaries for each chemical that were recommended for biomonitoring in ECHO. We were conservative in assigning priorities, e.g., predictive modeling results and isolated *in vitro* toxicity studies required corroborating evidence such as positive *in vivo* or HTP *in vitro* assay results. The prioritization criteria are presented in the main text.

Alternative Flame Retardants
Bis(2-ethylhexyl) tetrabromophthalate (BEH-TEBP). ***Exposure.*** BEH-TEBP was quantified in serum and breast milk at detection frequencies of 17% and 32%, respectively (Zhou et al. 2014). BEH-TEBP occurred in air, house dust, and in foods. In meta-analysis studies, the reported geometric mean (GM) level in house dust was ~300 ng/g (Mitro et al. 2016) and the range of medians was 12-650 ng/g (Larsson and Berglund 2018). Inhalation and dermal contact were the likely routes of exposure. EPA predicted exposure was in the 10^{-6} mg/kg-bw/day range (See Excel Table S2). ***Toxicity.*** Effects of BEH-TEBP on thyroid hormone deiodinase and sulfotransferase in human bioassays were reported (Smythe et al. 2017); nevertheless, there was a paucity of other human health effects reported for BEH-TEBP. Predictive modeling suggested that BEH-TEBP was a developmental toxicant (See Excel Table S10). ***Biomarker.*** Measurement of BEH-TEBP in serum and breast milk serves as a biomarker of exposure. 2,3,4,5,-Tetrabromo benzoic acid (TBBA), a metabolite of BEH-TEBP, was used as a biomarker of exposure (Hoffman et al. 2014). In addition to TBBA, a second metabolite, 2,3,4,5-tetrabromo phthalic acid (TBPA), was measured in rat urine (Silva et al. 2016).
Hexabromobenzene (HBBz). ***Exposure.*** HBBz was detected in 48% of Norwegian serum samples, with a median value of 0.03 ng/g lipid (Cequier et al. 2015a). HBBz was detected in >50% of ambient air samples but at a low level, with an average of 4.6 ± 1.0 pg/m^3 (Venier et al. 2012), in indoor air (Liagkouridis et al. 2017; Newton et al. 2015), and in >80% of house dust samples at levels of 10-100s of ng/g (Brown et al. 2014; Cristale et al. 2016; Shoeib et al. 2012). EPA predicted exposure was in the 10^{-7} mg/kg-bw/day range (See Excel Table S2). ***Toxicity.*** EPA reported that HBBz induced serum carboxylesterase activity in a rat dietary study (U.S. EPA 1985, 1987a). *In vitro* activation of aryl hydrocarbon receptor (AhR) was reported, suggesting developmental disruption potential of this compound (Brown et al. 2004). ***Biomarker.*** HBBz was metabolized (debrominated) to penta, tetra, and tribromo species in rats, with conjugated forms produced (Koss et al. 1982; Yamaguchi et al. 1988). HBBz bioaccumulated in tissues (See Excel Table S10). HBBz was measured in human serum and hair as a biomarker for exposure (Cequier et al. 2015a; Li et al. 2018).
Hexabromocyclododecane (HBCD). ***Exposure.*** Alpha, beta, and gamma-isomers of HBCD were detected in ambient and indoor air, house dust, and food (Eljarrat et al. 2009; Mitro et al. 2016; Newton et al. 2015; Sahlstrom et al. 2015). The ubiquitous presence of HBCD in house dust and air suggested that inhalation and dermal contact were major routes of continual human exposure, while exposure through food was likely a more episodic event, since this chemical was not found in all foods. HBCD was found in nearly 100% of house dust samples, in the hundreds of ng/g (Larsson and Berglund 2018; Mitro et al. 2016). In one study, HBCD was found in all human milk samples (Carignan et al. 2012), and the nursing infant dietary intake for HBCD was approximately 175 ng/kg of body weight/day (Eljarrat et al. 2009). HBCD was measured in human adipose tissue (Johnson-Restrepo et al. 2008). ***Toxicity.*** HBCD was suggested as an endocrine disruptor in humans (Johnson et al. 2013). Administration of HBCD in several animal studies suggested the occurrence of reproductive, endocrine, developmental, and neurotoxic effects (Ema et al. 2008; Koike et al. 2016; Miller-Rhodes et al. 2014; Tung et al. 2017; van der Ven et al. 2006; van der Ven et al. 2009). Results of *in vitro* studies also suggested reproductive effects (Hamers et al. 2006; Krivoshiev et al. 2016). ***Biomarker.*** The parent compound was measured as a biomarker of exposure in human breast milk (Carignan et al. 2012). HBCD was shown to exist as three diastereoisomers, of which the *alpha* form was more slowly debrominated than the *beta* and *gamma* isomers (Hakk 2016).

Melamine.

Exposure. Melamine, a nitrogen-containing AFR, was reported in indoor dust in the U.S. (mean 19 µg/g) as well as 11 other countries (Zhu and Kannan 2018b). Seventy-six percentof 492 urine samples from the general U.S. population had measurable levels; the geometric mean was 2.37 ng/mL, the 95th percentile was 12 ng/mL, and the range was from less than the limit of detection (LOD) to 161 ng/mL of urine (Panuwet et al. 2012). The high frequency of detection and broad concentration range suggested that the U.S. population was exposed to either melamine and/or the pesticide cyromazine, found in foods and which degrades to melamine. In contrast to the deliberate addition of melamine to food that occurred in China, it still was detected in infant formulas, dairy, and bread products in the U.S. (Hilts and Pelletier 2009; Zhu and Kannan 2018a). Results reported by the FDA for melamine and related analogues in 1,428 food samples revealed a range from <LOQ to 1,817 mg/kg, and cyanuric acid levels ranging from <LOQ to 3.62 mg/kg (Hilts and Pelletier 2009). EPA predicted exposure was in the 10^{-5} mg/kg-bw/day range (See Excel Table S2). Melamine was reported as a high-volume chemical, with a production of 100-250 X 10^{6} pounds per year (U.S. EPA 2016b).

Toxicity. NSF International determined that melamine produces urolithiasis based on a 13-week study in rats (U.S. EPA 2010a). Melamine was implicated as a reproductive and developmental toxicant in mice and rats (Bandele et al. 2014; Bhat et al. 2010; Chang 2014; Chu et al. 2017; Dai et al. 2015; Stine et al. 2014; Yin et al. 2013). Endocrine disruption and neurotoxicity were also reported for melamine (An et al. 2011; Bolden et al. 2017; Yang et al. 2012). Predictive modeling suggested that it may be a developmental toxicant (See Excel Table S10). Melamine in combination with cyanuric acid was a potent renal toxicant.

Biomarker. The parent compound was measured in human urine as a biomarker for exposure (Lin et al. 2013; Panuwet et al. 2012).

Alternative Plasticizers

Bis(2-ethylhexyl) adipate (DEHA).

Exposure. DEHA metabolites were measured in human urine and fingernails (Alves et al. 2017). Mean levels of 80 µg/g and 49 ng/m^3 were measured in house dust and indoor air, respectively (Fromme et al. 2015). In a study of meta-analysis, the median house dust concentration ranged from 5-173 ng/g (Larsson and Berglund 2018). DEHA was frequently detected in surface water, groundwater, and drinking water in the U.S., at levels of a few parts per billion (ng/mL). DEHA was measured over 3 decades ago at levels of 0.2-1 ng/mL in water samples collected from 5 of 23 U.S. sites (Fan 2003). It was quantified in duplicate diets for infants, with a daily intake at the 95th percentile of 4.66 ug/kg-bw (Fromme et al. 2013). The principal route of exposure to DEHA was through food and water ingestion. EPA predicted exposure was in the 10^{-4} mg/kg-bw/day range, which was in the top 15 ranking of exposure amongst modelled chemicals (See Excel Table S2).

Toxicity. Based on rat studies, DEHA exhibited developmental toxicity (U.S. EPA 1989b). The International Agency for Research on Cancer (IARC) and the EPA evaluated the oral carcinogenicity data for DEHA and classified it as a C — possible human carcinogen. The cancer weight-of-evidence classification was based on all routes of exposure (U.S. EPA 1998c). Predictive modeling suggested that DEHA was a reproductive toxicant (See Excel Table S10).

Biomarker. Mono-2-ethylhexyl adipate (MEHA), a DEHA-specific biomarker; mono-2-ethylhydroxyhexyl adipate (MEHHA); and mono-2-ethyloxohexyl adipate (MEOHA) were identified using human liver microsomes (Silva et al. 2013). Metabolites as biomarkers were monitored in human urine and fingernails; the relative proportion of the secondary metabolites was higher in urine (Alves et al. 2017). The occurrence of DEHA in several environmental media likely gives rise to continuous integrated exposure that may reach levels that potentially elicit health effects.

Bis(2-ethylhexyl)-1, 4-benzenedicarboxylate [Bis(2-ethylhexyl)-1, 4-terephthalate (DEHT)].

Exposure. DEHT was measured in indoor dust (mean 74 µg/g), indoor air (mean 31 ng/m^3) (Fromme et al. 2015), and at a maximum level of 440 µg/g in house dust (Nagorka et al. 2011). Two isomeric forms of DEHT, the primary metabolite, were found with a 97% detection frequency in urine (Alves et al. 2017). EPA predicted exposure was in the 10^{-5} mg/kg-bw/day range (See Excel Table S2).

Toxicity. NSF International evaluated the noncancer oral toxicity data for DEHT and performed a risk assessment based on the degeneration of the outer nuclear layer of the retina observed in female rats. Data indicated that DEHT did not produce antiandrogenic effects (Gray Jr et al. 2000). Results for potential

endocrine-disrupting activity of DEHT against human sex hormone-binding globulin (SHBG) using *in silico* approaches suggested that it has potential to engage residues of SHBG and thus interfere with its steroid homeostatic function (Sheikh et al. 2016). No evidence of male reproductive effects characteristic of certain ortho-phthalates was observed with DEHT (ITER 2018a). Predictive QSAR modeling results suggested that DEHT was a developmental toxicant (See Excel Table S10).

Biomarker. Two isomeric forms of the DEHT primary metabolite were used as biomarkers in human urine; research is needed to determine whether one or both forms best represent the biomarker of exposure (Alves et al. 2017).

Aromatic Amines

2-Methoxyaniline (Anisidine).

Exposure. The production estimate in 2006 was 0.5-1 million pounds (U.S. EPA 2007). This compound was measured in paper napkins (Yavuz et al. 2016), cigarette smoke, and wastewater from oil refineries (Desai 2014). This compound was excreted in urine, and a general population biomonitoring study in Germany reported the occurrence of this compound in urine (Kutting et al. 2009). An EPA predicted median exposure value was in the 10^{-6} mg/kg-bw/day range (See Excel Table S2).

Toxicity. 2-Methoxyaniline was a bladder carcinogen in laboratory animals (Naiman et al. 2012). The IARC classified this compound as a group 2B, possible human carcinogen, and it is on the top 100 hazardous substances list (SaferChemicals 2017). One HTP *in vitro* assay activated an endocrine process (See Excel Table S9). Predictive QSAR modeling was positive for developmental toxicity (See Excel Table S10).

Biomarker. This compound was oxidized to an n-hydroxy derivative, which interacted with heme in hemoglobin, leading to the formation of methemoglobin. Biomonitoring was demonstrated in human urine (Kutting et al. 2009).

2-Methylaniline (also known as o-Toluidine).

Exposure. In 2006, U.S. production was reported to be 10-50 million pounds (U.S. EPA 2007). This compound was also measured in urine from the general population in Germany at sub-µg/L concentrations (Kutting et al. 2009) and was reported in breast milk (DeBruin et al. 1999). 2-Methylaniline was reported to be present in tobacco smoke and in a variety of foods, including kale, celery, carrots, peas, and cabbage at parts-per-million levels (Neurath et al. 1977). Occurrence of this compound in hair dyes, paper products, and indoor air was documented (Akyüz and Ata 2008; Yavuz et al. 2016). An EPA predicted median exposure value was in the 10^{-5} mg/kg-bw/day range (See Excel Table S2).

Toxicity. 2-Methylaniline was reported as highly toxic to humans (U.S. EPA 2016b). Acute, short-term exposure in humans caused methemoglobinemia, with clinical signs of central nervous system depression. This compound also formed DNA and hemoglobin adducts (Richter 2015). Chronic long-term exposure to this compound affected anemia, anorexia, weight loss, skin lesions, and cyanosis. Increased risk of bladder cancer was observed in occupationally exposed populations, and this compound was a carcinogen in rats and mice. The EPA classified this compound as a probable human carcinogen, and it was on the top 100 hazardous substances list (SaferChemicals 2017). This compound activated 2 endocrine and 2 obesity HTP *in vitro* assays (See Excel Table S9).

Biomarker. Besides excreted unchanged, 4-amino-3-methylphenol was found in urine of exposed rats. Also, it was measured in urine of an occupationally exposed German population (Teass et al. 1993).

3,4-Dichloroaniline.

Exposure. Other isomers, such as 3,5-dichloroaniline and 2,5-dichloroaniline, were reported in the literature. This chemical is also an environmental degradation product of phenyl urea and phenyl carbamate pesticides, especially diuron and propanil (Tasca and Fletcher 2018). Population-based biomonitoring studies were reported with urinary levels at several hundreds of parts-per-trillion (ng/L) in a German population (Kutting et al. 2009). Contamination of rice with soil-bound 3,4-dichloroaniline was reported (Still et al. 1980). This compound was found in surface water and tap water at ng/L concentrations (Birch et al. 2015; Jurado-Sanchez et al. 2012; Kussmaul 1978; Wegman and Dekorte 1981). An EPA predicted median exposure value was in the 10^{-7} mg/kg-bw/day range (See Excel Table S2).

Toxicity. This compound has estrogenic properties (Tasca and Fletcher 2018). Several ecotoxicological studies exist on the occurrence and toxicity of the compound (Carbajal-Hernandez et al. 2017; Horie et al. 2017; Xiao et al. 2016). This compound produced methemoglobinemia in rats and mice (Gosselin et al. 1984).

Hemoglobin adducts of this compound were identified in farmers exposed to propanil 4 months after the application of the herbicide.

Biomarker. Urine samples collected from farmers had measurable levels of 3,4-dichloroaniline (Pastorelli et al. 1998). Exposure to 3,4-dichloroaniline yielded 5-amino-2,3-dichlorophenol in laboratory animals (Goodwin 1976). 3,5-Dichloroaniline was a biomarker for exposure to fungicides such as vinclozolin, procymidone, iprodione, and chlozolinate, and 3,4-dichloroaniline was a biomarker of exposure to herbicides such as diuron, linuron, neburon, and propanil (Wittke et al. 2001). A method for the analysis of this compound in human urine was published (Turci et al. 2006). 3,4-Dichloroaniline was measured in urine of a German population (Kutting et al. 2009). This compound was demonstrated as a biomarker in urine from an occupationally exposed population (El Marbouh et al. 2002).

Environmental Phenols

Bisphenol A diglycidyl ether (BADGE).

Exposure. BADGE was detected in urine, plasma, adipose fat, and cord blood (Asimakopoulos et al. 2014; Chang et al. 2014; Wang et al. 2012b; Wang, L et al. 2015; Xue et al. 2015). In the largest study of 127 samples from the U.S. and China, BADGE was detected in 100% of urine samples (Wang et al. 2012b). Among the U.S. samples, the GM concentration was 3 ng/mL; notably, concentrations of BADGE were 3-4 times higher than concentrations of bisphenol A (BPA) (Wang et al. 2012b). BADGE was measured in indoor air for several building environments with a mean of 0.70 ng/m^3 with a detection frequency of 86% (Xue et al. 2016). BADGE was detected in 78% of 40 U.S. indoor dust samples (Wang et al. 2012a), at a median of 2.4 ng/g (Larsson and Berglund 2018), and also detected in a variety of food items, including canned energy drinks (Gallo et al. 2017) and canned mushrooms (Alabi et al. 2014). Notably, BADGE was found in all 21 samples of canned liquid infant formula analyzed, with concentrations ranging from 2.4-262 ng/g (Cao et al. 2009). EPA predicted exposure was in the 10^{-5} mg/kg-bw/day range (See Excel Table S2).

Toxicity. Developmental effects in male rat pups have been shown; findings suggested that BADGE has developmental effects consistent with it being an endocrine disruptor (Hyoung et al. 2007). *In vitro* studies have shown adipogenic differentiation in stem cells (Chamorro-Garcia et al. 2012), neurobehavior (Prakash and Kumar 2014), and developmental toxicity (Ahn et al. 2008). Predictive modeling suggests that BADGE was a developmental toxicant (See Excel Table S10).

Biomarker. BADGE was biomonitored in human urine, plasma, and cord blood. Exposure and biomarkers of BADGE were recently reviewed (Andra et al. 2015).

Bisphenol AF (BPAF).

Exposure. In a convenience sample of U.S. adults during 2000-2014, urinary BPAF concentrations were detected in <3% of 616 samples (Ye et al. 2015). BPAF was not detected in plasma or cord blood in a study in the Czech Republic (Kolatorova et al. 2018). In a U.S. study (Liao and Kannan 2013), BPAF was found in 10% of 267 food items tested, including items in all 9 food group types. BPAF was not detected in indoor dust in the U.S., but was detected in indoor dust samples for 9% of samples in Japan and 76% of samples in Korea (Liao et al. 2012). EPA predicted exposure was in the 10^{-8} mg/kg-bw/day range (See Excel Table S2).

Toxicity. Effects on the endocrine system and numerous developmental systems were reported in rats (Umano et al. 2012). Decreased cognitive memory and neurological and behavioral effects were observed in both sexes of mice exposed to BPAF (Gong et al. 2017). *In vitro* studies have shown estrogenic effects (Cao et al. 2017), neurotoxicity (Lee et al. 2013), and endocrine disruption (Bhhatarai et al. 2016; Cao et al. 2017). HTP *in vitro* assay results indicated that BPAF was a neurotoxicant, obesogenic, and an endocrine disruptor (See Excel Table S9). Predictive modeling results suggested that BPAF was an endocrine disruptor (See Excel Table S11).

Biomarker. Exposure and biomarkers of BPAF were recently reviewed (Andra et al. 2015; Chen et al. 2016). The parent compound was measured in human urine, plasma, and breast milk (Kolatorova et al. 2018; Niu et al. 2017; Ye et al. 2015).

Bisphenol B.

Exposure. Bisphenol B concentrations were not detected in urine samples in Australia (Heffernan et al. 2016; Yang et al. 2014), but were detected in 2 of 20 (10%) urine samples in a Spanish study (Cunha and Fernandes 2010). An Italian study detected bisphenol B in serum collected from 16 of 58 women with endometriosis and 0 of 11 controls (Cobellis et al. 2009). Bisphenol B was not detected in indoor dust in the U.S., Japan, or Korea

(Liao et al. 2012). Bisphenol B was detected in dairy products (3.5% of 29 products), cereal products (12.5% of 48 products) (Liao and Kannan 2013), commercial milk (9% of 68 samples) (Grumetto et al. 2008), canned green beans, tripe, and cockles (Alabi et al. 2014; Grumetto et al. 2008). EPA predicted exposure was in the 10^{-7} mg/kg-bw/day range (See Excel Table S2).

Toxicity. We did not find human or animal toxicity studies. However, HTP *in vitro* testing activated the calcium ion channel indicating neurotoxicity, 3 endocrine, and 5 obesity assays (See Excel Table S9). Also, predictive modeling results suggest that bisphenol B is a developmental and reproductive toxicant (See Excel Table S10), and an endocrine disruptor (See Excel Table S11).

Biomarker. Biomarkers of bisphenol B were recently reviewed (Andra et al. 2015).

A method was available for measuring urinary bisphenol B in humans (NIEHS 2018).

3,3',5,5'-Tetrabromobisphenol A (TBBP-A).

Exposure. TBBP-A was measured in ambient and indoor air and in many house dust samples (Mitro et al. 2016). TBBP-A levels occurred in the hundreds of ng/g of house dust (Larsson and Berglund 2018; Mitro et al. 2016; Wang, W et al. 2015), suggesting that inhalation and dermal contact were the primary routes of exposure. TBBP-A was detected in 44% of human breast milk samples, ranging from 0.06-37 ng/g, in 30% of maternal and cord serum samples, with an average of 154 and 199 pg/g, respectively, but not detected in adipose tissue (Cariou et al. 2008). However, in a different study, TBBP-A was detected in human adipose tissue (Johnson-Restrepo et al. 2008). EPA predicted exposure was in the 10^{-5} mg/kg-bw/day range (See Excel Table S2).

Toxicity. TBBP-A was shown to produce uterine endometrial atypical hyperplasia (Wikoff et al. 2015). There was evidence that TBBP-A may weakly affect thyroid activity based on a mother-infant paired study (Kim and Oh 2014). Disruption of thyroid homeostasis was proposed as the primary toxic effect of TBBP-A (Birnbaum and Staskal 2004). Neurobehavioral effects, endocrine disruption, and immunological changes in animals were reported (Hall et al. 2017; Kitamura et al. 2002; Koike et al. 2016; Liu et al. 2018; Mariussen and Fonnum 2003; Pollock et al. 2017; Szychowski and Wojtowicz 2016; Szymanska 1995; Watanabe et al. 2010). One study suggested that TBBP-A did not exhibit reproductive and developmental effects (Stuer-Lauriden 2007). Two neurotoxicity, 4 endocrine, and 5 obesity *in vitro* assays were activated by TBBP-A (See Excel Table S9).

Biomarker. TBBP-A was measured in serum, breast milk, and cord blood serum to assess exposure (Cariou et al. 2008). Also, TBBP-A was measured in human adipose tissue (Johnson-Restrepo et al. 2008). The toxicokinetics for TBBP-A in humans and rats were studied, and the half-life in rats was 13 h. In humans, it was rapidly conjugated as glucuronide and, to a lesser extent, as sulfate (Schauer et al. 2006).

2,2',6,6'-Tetrachlorobisphenol A (TraTBA).

Exposure. TraTBA was detected in 4 of 10 urine samples from anonymous donors in France, with concentrations ranging from <LOQ to 1.50 ng/mL (Venisse et al. 2014), but not detected in two other pilot studies in Spain (Rodriguez-Gomez et al. 2014; Vela-Soria et al. 2014). EPA predicted exposure was in the 10^{-7} mg/kg-bw/day range (See Excel Table S2).

Toxicity. Agonistic activity to human retinoid X receptor (RXR) was suggested by a two-hybrid yeast assay (Li et al. 2016). TraTBA increased the level of the estrogen-regulated proteins, progesterone receptor, and pS2 (Olsen et al. 2003). Predictive modeling suggested that TraTBA was a developmental and reproductive toxicant (See Excel Table S10).

Biomarker. Biomarkers of chlorinated bisphenols were recently reviewed (Andra et al. 2015). Methods were published to measure TraTBA in human urine (Grignon et al. 2016; Liao and Kannan 2012), breast milk (Rodriguez-Gomez et al. 2014), and plasma (del Olmo et al. 2005).

4-n-Nonylphenol.

Exposure. 4-n-Nonylphenol was detected in environmental media, including air (Rudel et al. 2003), house dust (Mitro et al. 2016; Rudel et al. 2003), food (Guenther et al. 2002; Ying et al. 2002), and drinking water (Kolpin et al. 2002; Kuch and Ballschmiter 2001; Watanabe et al. 2007). In one study, 4-n-nonylphenol was detected only rarely in food, beverages, indoor air, outdoor air, house dust, soil, hand surfaces, and urine [detection frequencies (DFs) <11%] (Wilson et al. 2007). Another study suggested that 4-n-nonylphenol is rarely detected because it is not used commercially (Rudel et al. 2010). Concentrations of 4-n-nonylphenol have been measured in human urine (Calafat et al. 2005; Kuklenyik et al. 2003; Tang et al. 2013; Wilson et al. 2007), blood (Chen et al. 2008), and breast milk (Ademollo et al. 2008). Notably, a study of urinary concentrations

measured in a reference population of U.S. adults found detectable concentrations of 4-n-nonylphenol in 51% of 371 samples (Calafat et al. 2005).

Toxicity. Data showed that p-nonylphenol induced an estrogen-like response in first-trimester human placenta by increasing trophoblast differentiation and apoptosis (Bechi et al. 2006). An *in vitro* culture study of chorionic villus explants from human placenta found that an unbalanced cytokine network at the maternal-fetal interface resulted in implantation failure, pregnancy loss, or other complications (Wang et al. 2013). Developmental and reproductive effects were observed in rats (Chapin et al. 1999; Flynn 1999; Han et al. 2004; Hossaini et al. 2001; Jie et al. 2016). HTP *in vitro* testing results indicated that p-nonylphenol was neurotoxic, affected the thyroid, and activated 4 of 6 obesity assays (See Excel Table S9). Predictive modeling suggested that it was a developmental and reproductive toxicant (See Excel Table S10).

Biomarker. Although 4-n-nonylphenol was measured in human samples, it was not a specific biomarker of the parent compound, as it incorporated exposure to 4-nonylphenol, which was also metabolized to 4-n-nonylphenol (Calafat et al. 2005).

Organophosphorus-based Flame Retardants

2,2-Bis(chloromethyl) propane-1,3-diyltetrakis(2-chloroethyl) bisphosphate (V6).

Exposure. V6, an emerging flame retardant, was detected in almost 90% of car dust samples at a mean of 676 ng/g (Christia et al. 2018), in house dust with a median of 11 ng/g (Larsson and Berglund 2018), and in house dust and baby products in a different study (Fang et al. 2013). The levels were considerably higher in car dust than house dust. Tris(2-chloroethyl) phosphate (TCEP) was reported to co-occur with V6 (Fang et al. 2013). In a Norwegian cohort study, V6 was detected in 12% of fingernails, but its three metabolites were not detected in urine (Alves et al. 2017).

Toxicity. V6 exhibited developmental and reproductive toxicity in rats (van der Veen and de Boer 2012). QSAR predictive modeling suggested that this compound was a reproductive toxicant.

Biomarker. Biomonitoring is feasible since V6 was measured in human fingernails (Alves et al. 2017); however, a biomarker in urine has not been reported but likely could be developed.

2-Ethylhexyl diphenyl phosphate (EHDPP).

Exposure. In a meta-analysis study, EHDPP was prevalent in house dust with a median of medians of 696 ng/g (Larsson and Berglund 2018). In other studies EHDPP was measured in house dust at low µg/g levels (Brommer and Harrad 2015; Xu et al. 2016) and also found in some foods in a Swedish and U.S. food market basket survey (Darnerud et al. 2016; Poma et al. 2017; Wang and Kannan 2018). Low correlations between house dust and urine levels were reported (Dodson et al. 2014). Exposure was demonstrated by measuring the parent compound in hair (Kucharska et al. 2015) and its metabolite, diphenyl phosphate (DiPP), in urine (Fromme et al. 2014). EPA predicted exposure was relatively high at 10^{-5} mg/kg-bw/day range (See Excel Table S2). The production volume was 1-10 X 10^{6} pounds per year in 2016 (U.S. EPA 2016b).

Toxicity. Neurotoxicity was reported in the medaka fish (Jarema et al. 2015). It was implicated as a developmental toxicant and in human sphingolipid homeostasis (Zhao et al. 2016). EHDPP activated the calcium ion channel assay and 4 obesity HTP *in vitro* assays (See Excel Table S9). Our QSAR modeling results suggest that EHDPP was a reproductive toxicant (See Excel Table S10).

Biomarker. EHDPP has been measured in hair, and its metabolite, DiPP, has been measured in human urine (Fromme et al. 2014; Kucharska et al. 2015; Wang et al. 2019).

Bis(2-ethylhexyl) phosphate (BEHP).

Exposure. This compound is a flame retardant and an alternative plasticizer. BEHP was reported in urine with a geometric mean value of 13.4 pg/mL (specific gravity adjusted) (Wang et al. 2019). It was quantified in indoor air of two studies (0.31-2.4 µg/m^3, with a mean of 276 ng/m^3) (Fromme et al. 2015; Takeuchi, S et al. 2014). It was also quantified in house dust in Germany, with a mean of 1,973 µg/g (Fromme et al. 2015) and in foodstuffs in the U.S. (Wang and Kannan 2018). The production volume reported for the U.S. in 2016 was 1-10 X 10^{6} pounds per year (U.S. EPA 2016b).

Toxicity. No publications were found for toxicity. BEHP activated 5 obesity assays in HTP *in vitro* testing (See Excel Table S9). QSAR predictive modeling results suggested that this chemical was a developmental and reproductive toxicant (See Excel Table S10).

Biomarker. This chemical was measured in human urine (Sun et al. 2018; Wang et al. 2019). However, bis(2-ethylhexyl) phosphate is a metabolite of tris(2-ethylhexyl) phosphate. Thus, it is a biomarker of exposure representing two chemicals.

Tris(2-butoxyethyl) phosphate (TBOEP).

Exposure. TBEOP was detected in indoor dust and occurred at a high detection frequency, with a range of 0.050-27 µg/g (Ali et al. 2012; Marklund et al. 2003). The median of medians reported in a meta-analysis on house dust was 12,700 ng/g (Larsson and Berglund 2018). TBOEP was reported in ≥90% of 150 surface and drinking water samples (Kim and Kannan 2018). Continuous exposure to TBOEP likely occurs via inhalation, dermal contact, and ingestion routes. TBOEP was not found in 12 analyzed food categories (Poma et al. 2017) but was detected in meat and seafood collected from Albany, New York (Wang and Kannan 2018). TBOEP has been measured in breast milk (He et al. 2018; Sundkvist et al. 2010), hair (Kucharska et al. 2015), and as its metabolite, bis(2-butoxyethyl) phosphate (BBOEP), in urine (Cequier et al. 2015b; He et al. 2018; Wang et al. 2019). BBOEP was measured in urine in a mother-child pair cohort study (Cequier et al. 2015b). EPA predicted exposure was high in the 10^{-4} mg/kg-bw/day range (See Excel Table S2). The production volume was 1-10 X 10^{6} pounds per year in 2016 (U.S. EPA 2016b).

Toxicity. The Agency for Toxic Substances and Disease Registry (ATSDR) and NSF International have evaluated oral toxicity data for TBOEP in a dietary exposure study of rats that showed hepatocyte vacuolation (Reyna and Thake 1987). Other health effects were reported, including asthma and allergies (Araki et al. 2014), endocrine disruption (Kwon et al. 2016; Ma et al. 2016), developmental (Egloff et al. 2014; Liu, R et al. 2017), reproductive (Han et al. 2014), and neurotoxicity (Carrington et al. 1990; Sun et al. 2016).

Biomarker. The parent compound was measured in breast milk and hair to assess exposure, and its metabolite, bis(2-butoxyethyl) phosphate (BBOEP), was measured in urine (Dodson et al. 2014; He et al. 2018; Wang et al. 2019).

Tris(2-ethylhexyl) phosphate (TEHP).

Exposure. This compound was detected at low levels in ambient air (García 2014), indoor air (up to 3.4 ng/m^3) (Hartmann et al. 2004), and dust (0.06-13 µg/g) (Larsson and Berglund 2018; Marklund et al. 2003). It was not detected in food samples (Poma et al. 2017). In a study of 400 urine samples, the detection frequency of its

metabolite, bis(2-ethylhexyl) phosphate, was about 20%; whereas in hair it was about 80% (He et al. 2018). In another study, the detection frequency and median concentration in hair were 93% and 10 ng/g, respectively (Kucharska et al. 2015). Its reported production volume in 2016 was 1-10 X 106 pounds per year (U.S. EPA 2016b).

Toxicity. *In vitro* results suggested that this chemical increased basal progesterone production significantly (Schang et al. 2016). HTP *in vitro* testing showed activation of 4 obesity assays (See Excel Table S9). Predictive modeling suggested that it was a developmental toxicant (See Excel Table S10).

Biomarker. Biomonitoring of tris(2-ethylhexyl) phosphate as the parent compound in human hair and as the metabolite BEHP in human urine was reported (He et al. 2018; Kucharska et al. 2015; Wang et al. 2019).

Perfluoroalkyl Substances

Perfluorobutanoic acid (PFBA).

Exposure. PFBA was detected infrequently in raw water (Post et al. 2013) and house dust (0.7 ng/g in Belgium) (D'Hollander et al. 2010). However, in a recent study, PFBA was detected in 92% and 58% in tap and bottled water samples, respectively, with a median value of 1.3 ng/L (Kabore et al. 2018). In food, PFBA was detected at a frequency of 8% with a mean concentration of 1,093 pg/g in cereals (Perez et al. 2014). In a meta-analysis study of house dust levels, the median of medians was 0.2 ng/g (Larsson and Berglund 2018).

Toxicity. PFBA was associated with hypertension (Bao et al. 2017). *In vivo* studies suggested that PFBA delayed eye opening and onset of puberty in mice (Das et al. 2008) and pupillary reflex in adult rats (Butenhoff et al. 2012). PFBA was shown to affect the thyroid, with reduced serum thyroxine (Butenhoff et al. 2012).

Biomarker. PFBA is one of Biomonitoring California's Designated Chemicals (Biomonitoring California 2015). Valid biomarkers are available in serum (Department of Toxic Substances Control [DTSC] 2017), and another study suggested potential biomarkers for PFBA in human urine and hair (Perez et al. 2012). PFBA was measured in human serum in a Chinese population (Bao et al. 2017).

Perfluorohexanoic acid (PFHxA).

Exposure. PFHxA was detected in 63% (average 47 pg/g) of 30 hair samples collected in Belgium (Alves et al. 2015) and in human milk in the U.S., Sweden, Germany (mean 0.820, 0.230, and 0.150 ng/mL, respectively) (Kubwabo et al. 2013; Tao et al. 2008), and Korea (median 0.047 ng/mL) (Kang et al. 2016). Studies in North America and Europe reported detection in drinking water (Zafeiraki et al. 2015), in a food basket survey conducted in Sweden (fruits and vegetables, LOD to 6.4 pg/g) (Gebbink et al. 2015; Vestergren et al. 2012), house dust (medians 0.3 and 0.9 ng/g in two separate studies in Belgium) (D'Hollander et al. 2010; Karaskova et al. 2016), and in indoor air (Padilla-Sanchez et al. 2017). PFHxA was detected in >20% of 30 potable water systems water samples in New Jersey (Post et al. 2013) and in 64% and 50% in tap and bottled water samples, respectively (Kabore et al. 2018). In a meta-analysis study of house dust, the median of medians was 4.8 ng/g (Larsson and Berglund 2018).

Toxicity. Studies suggested that PFHxA was associated with changes in testosterone (Zhao et al. 2016) and thyroid hormone (Li et al. 2017) levels. Decreased survival (Klaunig et al. 2015), reduced body weight and weight gain (Loveless et al. 2009), and neurotocixity (Chengelis et al. 2009) was reported in rats. Moreover, predictive modeling suggested that PFHxA was an endocrine disruptor (See Excel Table S11).

Biomarker. PFHxA is included in Biomonitoring California's Designated Chemicals list (Biomonitoring California 2015). PFHxA was measured in human serum in a Chinese population (Bao et al. 2017). Methods are available for measuring PFHxA in human serum (DTSC 2017) and hair (Alves et al. 2015).

Perfluoropentanoic acid (PFPeA).

Exposure. PFPeA was detected at low levels in human blood samples in the U.S. (Olsen et al. 2012). PFPeA was also detected in 82% of breast milk samples, with a mean of 0.058 ng/mL in Korea (Kang et al. 2016). It was detected in cereal, milk, olive oil, fish, and pork at a frequency of about 2-5% (Perez et al. 2012). PFPeA was detected in >20% of 30 potable water system water samples in New Jersey (Post et al. 2013) and in 68% and 32% in tap and bottled water samples, respectively (Kabore et al. 2018). The median of medians for house dust was 0.37 ng/g (Larsson and Berglund 2018).

Toxicity. Prenatal PFPeA was positively associated with T4 thyroid hormone changes in cord blood (Ha et al. 2016; Shah-Kulkarni et al. 2016). In another study, PFPeA was significantly positively correlated with the thyroid hormones TGAb and TMAb in the general population (Li et al. 2017). PFPeA was shown to activate

human peroxisome proliferator-activated receptor-alpha (PPARα) in an *in vitro* cell model (Wolf et al. 2012) and was a probable endocrine disruptor (See Excel Table S11).

Biomarker. It is included in Biomonitoring California's Designated Chemicals list (Biomonitoring California 2015). PFPeA was measured in blood serum in U.S. and Chinese populations (Liu, Y et al. 2017; Olsen et al. 2012), in Korean children (Kim and Oh 2014), and in breast milk (Kang et al. 2016).

Perfluorotridecanoic acid (PFTrDA).

Exposure. PFTrDA was detected among maternal or cord plasma samples collected from 2003-2011 in Japan (DF for maternal, 97-100%, median level: 0.24-0.41 ng/mL) (Okada et al. 2013). One study that included 521 samples from Japan, Korea, and Vietnam found the detection frequency and median levels of PFTrDA ranged from 96-100% and 0.31-0.60 ng/mL, respectively (Harada et al. 2011). However, in a study of Swedish women, all serum samples had values below the method detection limit (MDL) (Glynn et al. 2012). PFTrDA was also found in food (fish and eggs; 43 and 5.3 pg/g/d, respectively) (Gebbink et al. 2015) and vacuum cleaner dust (Liu et al. 2011). Examples of its use in products include awning textiles, leather, ski wax, and gloves (Kotthoff et al. 2015). The median of medians for house dust was 1.1 ng/g (Larsson and Berglund 2018).

Toxicity. PFTrDA may interfere with human T_4 thyroid hormone (Ji et al. 2012; Kim et al. 2011). Lower prenatal exposure to PFTrDA may decrease the risk of developing eczema in early childhood, only in female infants (Okada et al. 2014). Results from another study (Okada et al. 2014) suggested that PFTrDA may have an immunosuppressive effect on allergic diseases in 4-year-old children. Predictive modeling suggested that PFTrDA is an endocrine disruptor (Table S11).

Biomarker. PFTrDA was included in Biomonitoring California's Designated Chemicals list (Biomonitoring California 2015) and the candidate list of the REACH system in the European Union (KEMI 2015). It was measured in serum of Swedish women (Glynn et al. 2012) and in sera of Chinese, Korean, and Vietnamese populations (Bao et al. 2017; Harada et al. 2011; Kim et al. 2011).

Pesticides

Azoxystrobin.

***Exposure*.** Azoxystrobin, a fungicide, has been measured in ambient air (Murphy and Haith 2007), surface water (Battaglin et al. 2011), groundwater, finished and unfinished drinking water, and in food in the United States (Lentza-Rizos et al. 2006; USDA 2017). In 2013, this fungicide was detected in 52% of drinking water samples with a concentration range of 1.3-99 ng/L (USDA 2017) and in 45% of 103 sampled U.S. streams (Battaglin et al. 2011). Azoxystrobin was reported to occur in fruits and vegetables (USDA 2017). In spinach samples collected in 2011, azoxystrobin occurred at a mean of 616 ng/g (USDA 2017). This compound was detected in groundwater at a frequency of 17% with a median of 0.08 ng/L and in surface water at a 58% detection frequency with a median of 30.6 ng/L (Reilly et al. 2012). It has been detected in 23% of hair samples from farm workers in Belgium (Schummer et al. 2012) and in urine of pregnant French women (Jamin et al. 2014). EPA predicted exposure was in the 10^{-8} mg/kg-bw/day range (See Excel Table S2).

***Toxicity*.** Developmental effects have been reported (EFSA 2010a; FAO 2008). HTP *in vitro* results were positive in 3 neurotoxicity and 2 obesity assays (See Excel Table S9). Predictive modeling suggested azoxystrobin is a developmental toxicant (See Excel Table S10).

***Biomarker*.** In rats given a single oral dose of radiolabeled azoxystrobin, 73-89% of the administered dose was recovered in feces and 9-18% in urine after 7 days. Between 82% and 96% of the administered dose was excreted within the first 48 hours (FAO 2008). In addition, in hair and urine, azoxystrobin has been measured in serum in a Chinese population (Chang et al. 2017; Jamin et al. 2014; Schummer et al. 2012).

Recommendation. Based on the measured developmental effects, along with the availability of a biomarker, azoxystrobin is prioritized for monitoring in ECHO. Prior to testing in ECHO, measurements in a U.S. population needs to be conducted to confirm anticipated exposure.

Benomyl.

***Exposure*.** Benomyl, a systemic fungicide, has been reported in ambient air and U.S. surface water; however, the papers were published in the 1990s and may not reflect current levels (Baker et al. 1996; Senseman et al. 1997). Benomyl has been reported to occur in grapes with a 37% detection frequency, and a mean of 41 ng/g (FDA 2004-2005a, 2011; Roy et al. 1997; Yess et al. 1991). It has also been detected at lower frequencies in other fruits. Even though the registration for this fungicide was cancelled in 2001, it has been recently reported in house dust (Ouyang et al. 2017). EPA predicted exposure was in the 10^{-7} mg/kg-bw/day range (See

Excel Table S2).

Toxicity. Benomyl has been listed as a developmental and reproductive toxicant (OEHHA 1991). Developmental toxicity and teratogenic effects in mice were suggested in the 1980s (Kavlock et al. 1982; U.S. EPA 1987c). HTP *in vitro* result was positive in the TPO thyroid assay (See Excel Table S9). Predicted modeling results suggested benomyl was a developmental and reproductive toxicant (See Excel Table S10).

Biomarker. The metabolite methyl-5-hydroxy-2-benzimidazole carbamate in urine has been measured as a biomarker of exposure to benomyl in nursery workers (Hoekstra et al. 1996). The biological half-life of benomyl is <24 hours (PubChem 2018a).

Recommendation: Based on the continual appearance of benomyl in food and house dust, concern for its toxicity, and availability of a biomarker in urine, it is recommended for biomonitoring.

Captan.

***Exposure*.** Captan, a fungicide, was detected in groundwater, drinking water, food, and indoor and personal air (Briggins and Moerman 1995; FDA 2004-2005a, 2011; Gartrell et al. 1986; Kool et al. 1982; Lewis et al. 1988; USDA 2017). Captan was detected in 100% of strawberries tested (FDA 2004-2005a, 2011) and in peaches at a frequency of 6.9% and a mean of 288 ng/g in 707 samples collected in 2014, and other fruits (USDA 2017). It was measured as tetrahydrophthalimide in maternal and cord blood plasma at detection frequencies of 50% and 44%, respectively (Whyatt et al. 2003), and in urine (Galea et al. 2015). EPA predicted exposure was in the 10^{-7} mg/kg-bw/day range (See Excel Table S2).

Toxicity. Captan was reported as a developmental toxicant in rats (U.S. EPA 1989a, 2004). It was shown to increase thyroid weight, and an *in vitro* study exhibited antiestrogenic activity (Okubo et al. 2004) and androgen activity (Judson et al. 2015). HTP *in vitro* result was positive in an androgen antagonist assay (See Excel Table S9). Predicted modeling results suggested captan was a developmental toxicant (See Excel Table S10).

Biomarker. The toxicokinetics of captan are well known (Berthet et al. 2012; Heredia-Ortiz and Bouchard 2012; Krieger and Thongsinthusak 1993). The biological half-life (feces, urine, or metabolism) of tetrahydrophthalimide (THPI) and the urine clearance rate are 13 and 21 hours, respectively (Heredia-Ortiz and Bouchard 2012). Its metabolites, THPI and thiazolidine-2-thione-4-carboxylic acid (TTCA), have been measured in human urine (de Cock et al. 1995; Galea et al. 2015; Heredia-Ortiz and Bouchard 2012; Krieger and Thongsinthusak 1993; van Welie et al. 1991). More than 90% of an oral dose of captan was excreted in feces and urine within 24 hours (PubChem 2018d). Captan has been measured as the parent compound in urine (Galea et al. 2015). THPI has also been measured in mother and cord blood plasma (Whyatt et al. 2003).

Recommendation: Based on the developmental toxicity in rats, the prevalence of exposure in the U.S. population, and the existence of biomarkers, biomonitoring of captan is recommended.

Chlorpropham.

Exposure. Chlorpropham, a carbamate herbicide and plant growth regulator, has been found in groundwater but not in drinking water (Mehnert et al. 1995). Low levels of chlorpropham have been reported in ambient air (Coscolla et al. 2011). It has been quantified in food (FDA 2004-2005a, 2011; Juhler et al. 1999; Ripley et al. 2000; USDA 2017) and has been detected in potatoes collected in 2015 at a frequency of 98.6% and a mean of 1,782 ng/g (USDA 2017). In the Total Diet Study it was detected at a frequency of 72% in potato chips, with a mean of 745 ng/g (FDA 2004-2005a, 2011). An untargeted analysis in urine of farm workers showed the presence of this compound (Jamin et al. 2014). EPA predicted exposure was in the 10^{-8} mg/kg-bw/day range (See Excel Table S2).

Toxicity. Dogs exposed to chlorpropham showed developmental affects (U.S. EPA 1996, 1998f). Teratogenic effects have also been observed in rabbits (U.S. EPA 1998f). There likely was an overlap between *in vitro* assay activity and predicted exposures (Wetmore et al. 2012), specifically with the 1 assay associated with *in vivo* thyroid activity. HTP *in vitro* assay results indicated neurotoxicity (See Excel Table S9). Predictive modeling results suggested chlorpropham was a developmental and reproductive toxicant (See Excel Table S10).

Biomarker. The metabolism of chlorpropham to m-chloroaniline has been reported in rats (Balaji et al. 2006). It has a tissue half-life in the rat ranging from 3.8-11.6 hours. Chlorpropham has been measured in human blood serum from a Chinese population (Chang et al. 2017).

Recommendation: Based on developmental effects, prevalence in food, and the existence of a biomarker, biomonitoring of chlorpropham is recommended.

Cyprodinil.

Exposure. Cyprodinil, a fungicide, has been detected in ambient air (Coscolla et al. 2011), groundwater (Meffe and de Bustamante 2014), and food (Esteve-Turrillas et al. 2016; Lorenzin 2007; USDA 2017). In 2014, cyprodinil was detected in 688 samples of blueberries at a frequency of 27% and mean of 110 ng/g and was present in several other fruits and vegetables (USDA 2017). Cyprodinil was measured at a frequency of 23% with a maximum concentration of 1.2 µg/g (Schummer et al. 2012). EPA predicted exposure was in the 10^{-9} mg/kg-bw/day range (See Excel Table S2).

Toxicity. The EPA has identified developmental effects in rats (U.S. EPA 1998b). *In vitro* results suggested cyprodinil was an AhR-agonist (Medjakovic et al. 2014) and may have antiandrogen and androgen effects in one assay (Orton et al. 2011) but not in another (Judson et al. 2015). There likely was an overlap between *in vitro* assay activity and predicted exposures (Wetmore et al. 2012), specifically for 7 assays associated with *in vivo* liver and kidney effects. The fungicide had hits on 9 of the tested HTP *in vitro* assays (See Excel Table S9). Predictive modeling suggested that cyprodinil binds to an androgen receptor (See Excel Table S11).

Biomarker. In rats, approximately 48-68% of the administered dose was eliminated within 48 hours in urine (PubChem 2018f). The parent compound served as the biomarker measured in hair (Schummer et al. 2012).

Recommendation: Based on the measured developmental effects, prevalence in food, and the availability of a biomarker, biomonitoring of cyprodinil is recommended.

Dicloran.

Exposure. Dicloran, a fungicide, has been detected in maternal plasma at a frequency of 77% and a range of limit of detection (LOD) to 28 pg/g creatinine (CRT), and in cord blood plasma at a frequency of 83% and a range of LOD to 32 pg/g CRT (Whyatt et al. 2003). It has been detected in surface water, groundwater (Maddy et al. 1982), and food (FDA 2004-2005a, 2011; Gartrell et al. 1986; Gotz et al. 1998; Readman et al. 1997; USDA 2017). Dicloran was detected in 553 samples of sweet potatoes collected in 2010 at a frequency of 46.1% and a mean of 273 ng/g (USDA 2017). EPA predicted exposure was in the 10^{-6} mg/kg-bw/day range (See Excel Table S2).

Toxicity. Dicloran is a developmental toxicant, but no reproductive effects were noted in exposed dogs (IPCS-INCHEM 1974; U.S. EPA 2006a). There likely is an overlap between *in vitro* assay activity and predicted exposures (Wetmore et al. 2012), specifically with the 1 assay associated with *in vivo* neuropathology and liver, kidney, and spleen effects. The fungicide is potentially a neurotoxicant based on HTP *in vitro* assays (See Excel Table S9). HTP *in vitro* assay results were positive for thyroid effects, neurotoxicity, and obesity (See Excel Table S9). Predictive modeling results suggested dicloran was a developmental toxicant (See Excel Table S10).

Biomarker. Dicloran is measurable as a biomarker in plasma (Whyatt et al. 2003). Urinary excretion in rats accounted for approximately 90% of the administered dose, which was recovered within 48 hours; up to 85% was recovered within 8 hours of treatment. The remainder was present in feces. The major metabolites are 2,6-dichloro-4-hydroxyaniline sulfate (22-63%) and 2,6-dichloro-4-hydroxyaniline glucuronide (16-29%) in rat urine, but 2,6-dichloro-4-hydroxyaniline was not present in goat urine (Moase 1998).

Recommendation: Based on dicloran detection in maternal plasma, cord blood plasma, and food; its classification as a developmental toxicant; and the availability of a biomarker, biomonitoring is recommended.

Glyphosate.

Exposure. Glyphosate, an herbicide, has been measured in urine with a mean of 1 ng/mL and a maximum of 4.2 ng/mL in a Danish sample of 13 mothers and 14 children (Knudsen et al. 2017) and in another study in adult urine (John and Liu 2018). The results are similar to those reported in a German study "Urinale 2015", which found glyphosate in 99.6% of the spot urine samples from 2011 participants (Knudsen et al. 2017). The youngest group of participants in the German study — ranging from 0 to 9 years and 10 to 19 years — had the highest concentrations of glyphosate in the urine (Knudsen et al. 2017). It has been measured in urine of children and adults in an agricultural area (Curwin et al. 2007) and in a small study of German adults using an ELISA assay (mean 9 ng/mL, range of 0.1-71.3 ng/mL) (Knudsen et al. 2017). Sixty percent of farmers had detectable levels in their urine on the day of the application, while their spouses had 4% detectable levels (Acquavella et al. 2004). Detectable levels of glyphosate were found in horticulturalists (Connolly et al. 2017). In one study, it was measured in human urine samples from 1993-2016; for the 2014-2016 period, the mean

was 0.314 ng/mL for glyphosate and 0.2285 ng/mL for its metabolite, aminomethylphosphonic acid (AMPA) (Mills et al. 2017). Ninety percent of pregnant women in Indiana hade detectable levels in their urine, which was associated with shorter gestational length (Parvez et al. 2018). It was found in very low levels in lake water (John and Liu 2018) and in water at levels in the 5-50 ng/mL range (U.S. EPA 2016a, b). Glyphosate was detected in food — e.g., in 300 samples of soybeans collected in 2011, glyphosate was found at a frequency of 90% with a mean of 1,937 ng/g (USDA 2017).

Toxicity. Glyphosate is developmental toxicant (U.S. EPA 2017a). A report in 2017 that reviewed the toxicity of glyphosate concluded that it does not have estrogen, androgen, thyroid, and steroidogenesis-mediated endocrine-disrupting properties (EFSA 2017a). The carcinogenicity of glyphosate was reviewed (Williams et al. 2016). Predictive modeling suggested that glyphosate was a developmental toxicant (See Excel Table S10).

Biomarker. Glyphosate and its metabolite AMPA were measured in urine of older adults (Mills et al. 2017). Glyphosate slowly metabolizes to AMPA (Anadon et al. 2009). The elimination half-life of AMPA is 15 hours after oral administration of glyphosate (Anadon et al. 2009; PubChem 2018b). Also, because many detergents degrade to AMPA, it is not a specific biomarker of exposure for glyphosate. Therefore, measurement of glyphosate in urine is recommended.

Recommendation: Based on its prevalence in biofluids and foods and its potential as a developmental toxicant, biomonitoring of glyphosate is recommended.

Iprodione.

Exposure. Iprodione, a fungicide, has been measured in ambient air, food, and urine (Bradman et al. 2007; Coscolla et al. 2011; FDA 2004-2005a, 2011; Magnér et al. 2015; USDA 2017). In a longitudinal study, its metabolite, 3,5-dichloroaniline (3,5-DCA) was measured in urine at a concentration range of 1.4-69 µg/g CRT with a median value of 13 ug/g CRT (Magnér et al. 2015). In 362 samples of peaches collected in 2015, it was detected at a 46% frequency with a mean of 672 ng/g (USDA 2017). It was also found in other fruits and in vegetables. It was not detected in house dust, including urban and farm homes (Quiros-Alcala et al. 2011). EPA predicted exposure was in the 10^{-8} mg/kg-bw/day range (See Excel Table S2).

Toxicity. A review of endocrine disruptor potency suggests that iprodione exhibits weak effects on aromatase activity and estrogen production (Stoker and Kavlock 2010). Reproductive and hematologic effects in dogs were observed (U.S. EPA 1988b). The U.S. EPA did not consider iprodione a developmental toxicant (U.S. EPA 1988b). HTP *in vitro* assay results indicated the potential for steroidogenic toxicity and was positive in 4 obesity assays (See Excel Table S9).

Biomarker. Dicarboximide fungicides (diuron, vinclozolin, and iprodione) contain the common moiety 3,5-DCA that was measured as a biomarker of exposure in human urine collected from an occupationally exposed population (Lindh et al. 2007; Turci et al. 2006).

Recommendation: Based on its prevalence in food, the potential for endocrine disruption, and reproductive toxicity, biomonitoring of iprodione is recommended using 3,5-DCA as a valid biomarker of exposure in a non-occupationally exposed population.

Metalaxyl.

Exposure. Metalaxyl, a fungicide, was measured in surface waters and food (Battaglin et al. 2011; USDA 2017). It was detected in 27% of 103 samples of surface waters (Battaglin et al. 2011; USDA 2017). In 232 finished drinking water samples, it was detected at a frequency of 45% at a concentration range of 2-25 ng/L (USDA 2017). In 744 samples of cucumbers, metalaxyl was detected at a frequency of 31%, with a mean of 47 ng/g (USDA 2017). Metalaxyl was quantified in ambient air with a mean of 45 pg/m^3 (Coscolla et al. 2011). Biomonitoring of metalaxyl in maternal and cord blood plasma revealed detection frequencies of 10% and 18%, respectively, at a concentration range of "not detected" (ND) to 28 pg/g CRT and ND to 250 pg/g CRT, respectively (Whyatt et al. 2003). EPA predicted exposure was in the 10^{-7} mg/kg-bw/day range (See Excel Table S2).

Toxicity. Liver toxicity and anemia in dogs (EFSA 2015) and developmental effects in rats were reported (EFSA 2015). Predictive modeling suggested metalaxyl was a developmental toxicant (See Excel Table S10). EFSA concluded that endocrine effects were unlikely (EFSA 2015). HTP *in vitro* results were positive in 4 obesity assays (See Excel Table S9). Predictive modeling results suggested metalaxyl was a developmental toxicant (See Excel Table S10).

Biomarker. Metalaxyl has been measured in serum, plasma, and its metabolites have been measured in urine

(Chang et al. 2017; Headley et al. 1996; Whyatt et al. 2003). Its metabolism in human liver has been shown to yield 2 hydroxymetalaxyl metabolite derivatives; N-(2-hydroxmethyl-6-methylphenyl)-N-(methoxyacetyl)alanine methyl ester and/or N-(2,6-dimethyl-5-hydroxphenyl)-N-(methoxyacetyl)alanine methyl ester (Abass et al. 2007). In a rat study, female rats eliminated 55-60% of the dose in urine, whereas males eliminated 60-70% in feces (PubChem 2018h).

Recommendation: Based on its prevalence in drinking water and food, measured developmental toxicity, and a biomarker of exposure, biomonitoring of metalaxyl is recommended.

Propiconazole.

Exposure. Propiconazole, a fungicide, was measured in food commodities (USDA 2017). In 285 samples of peaches collected nationally, it was detected at a frequency of 46%, with a mean of 180 ng/g (USDA 2017). In another study it was detected in 17% of water samples from 29 streams in 13 states in the United States (Battaglin et al. 2011). Reports of its occurrence in other environmental and exposure media were not found. EPA predicted exposure was in the 10^{-8} mg/kg-bw/day range (See Excel Table S2).

Toxicity. The European Chemicals Agency (ECHA) proposed classifying propiconazole as a category 18 reproductive toxicant, supported by its effects on the endocrine system (EFSA 2017b), which were consistent with the results of predictive modeling for endocrine disruption. In chronic toxicity studies in rats and mice, developmental toxicity was observed (EFSA 2017b). Traditional studies did not show evidence of neurotoxicity, and the European Food Safety Authority (EFSA) identified a data gap in endocrine disruption on male reproduction (EFSA 2017b). HTP *in vitro* testing showed activities in 11 different assays (See Excel Table S9). Predictive modeling suggested that propiconazole binds to the androgen receptor (See Excel Table S11).

Biomarker. Biomonitoring of propiconazole was performed in serum in a Chinese population (Chang et al. 2017). A comparison of propiconazole in hair with levels in plasma and urine in a rat study exhibited a linear relationship (Hardy et al. 2015). It was measured in mothers' milk (Yildizdas et al. 2018).

Recommendation: Based on the likelihood of exposure from ingesting food, classification as a reproductive toxicant, its effect on the endocrine system, and availability of a biomarker, biomonitoring of propiconazole is recommended.

Pyrimethanil.

Exposure. Pyrimethanil, a fungicide, was detected in several fruits and vegetables. For example, in a national sample of 705 pear samples, it was detected at a frequency of 76%, with a mean of 869 ng/g (USDA 2017). It has been measured in grapes (USDA 2017) and wine (Esteve-Turrillas et al. 2016). Using a passive wrist sample, pyrimethanil has been detected among farm workers (Aerts et al. 2018), indicating potential dermal exposure. EPA predicted exposure was in the 10^{-9} mg/kg-bw/day range (See Excel Table S2).

Toxicity. Developmental toxicity has been observed in rats and rabbits (EFSA 2006b), which was in contrast to the World Health Organization (WHO) report that concluded no adverse effects (WHO 2007a). A mechanistic study showed an effect on thyroid hormone balance due to increased hormone clearance from chronic thyroid stimulation (EFSA 2006b). *In vitro* results suggested AhR-agonist activity (Medjakovic et al. 2014) and antiandrogen and androgen effects (Orton et al. 2011). HTP *in vitro* results were positive in 3 obesity assays (See Excel Table S9).

Biomarker. Pyrimethanil has been measured in serum in a Chinese population (Chang et al. 2017).

Recommendation: Based on its prevalence in fruits and vegetables, measured developmental toxicity, potential effects on the thyroid hormone balance, and availability of a biomarker, biomonitoring is recommended.

Tebuconazole.

Exposure. Tebuconazole, a fungicide, was measured in hair, surface waters, and food commodities. Tebuconazole has also been detected in surface waters and foods (Battaglin et al. 2011; USDA 2017). It was detected in 6% of 103 surface water samples collected from streams in 13 states in the United States (Battaglin et al. 2011). In a national sample of 232 samples of cherries, it was detected at a frequency of 30%, with a mean of 557 ng/g (USDA 2017). It was also found in other fruits and vegetables. It was detected at a frequency of 14.5% in farmers' hair (Schummer et al. 2012). Using a passive wrist sample, tebuconazole was detected on farm workers (Aerts et al. 2018), suggesting potential dermal exposure. EPA predicted exposure was in the 10^{-8} mg/kg-bw/day range (See Excel Table S2).

Toxicity. Canada Health has reviewed the toxicity of tebuconazole (Canada 2016). Studies with rats, mice, and rabbits did not exhibit adverse effects on reproductive parameters (Canada 2016), and traditional *in vivo* studies did not show evidence of neurotoxicity (IPCS-INCHEM 2006). *In vitro* results indicated weak antiandrogen effects (Judson et al. 2015; Orton et al. 2011). This fungicide was activated in 9 HTP *in vitro* assays (See Excel Table S9). Predictive modeling suggested that tebuconazole was a developmental and reproductive toxicant (See Excel Table S9) and binds to the androgen receptor (See Excel Table S11).
Biomarker. In a study of agricultural workers, the metabolites identified in urine were TEB-OH, TEB-COOH, and their glucuronide conjugates, with TEB-OH being the most abundant (molar fraction: 0.67) (Brumby 2018; Fustinoni et al. 2014). These were also the metabolites of penconazole. In animals, 91-98% of tebuconazole was excreted after 72 hours. A difference in sex-related excretion in test animals was noted, with males having a urine/feces ratio of 16/78 in contrast to the females, which excreted at a ratio of 30/62 (PubChem 2018g). The measurement of tebuconazole in hair was validated (Polledri et al. 2018).
Recommendation: Based on the prevalence of tebuconazole in food, activation of numerous *in vitro* assays, and the availability of a biomarker, biomonitoring of tebuconazole is recommended.

Table S4. Chemicals Deferred Pending Further Data

For chemicals in the deferred group, they are divided into 4 categories based on the information available for exposure, toxicity, and availability of a biomarker. As such, opportunities exist for conducting research to fill existing knowledge gaps.

Category	Detected in Biospecimens?	Detected in Environmental Media?	Toxicity Concern?	Biomarker Exists?	Additional Research Needed
A	Insufficient Information	Insufficient Information	Yes	Yes	Chemicals should be measured in biospecimens of a U.S. non-occupationally exposed population to determine if there is exposure.
B	Insufficient Information	Yes	Yes	No	No biomarker exists; develop one and test it in a U.S. non-occupationally exposed human biospecimens to confirm anticipated exposure.
C	Insufficient Information	Insufficient Information	Yes	No	Need data on exposure levels for U.S. non-occupationally exposed population and develop a biomarker
D	Insufficient Information	Insufficient Information	Insufficient Information	Yes/No	Needs information on exposure, toxicity and perhaps biomarker development.

Alternative Flame Retardants

Category D:

2-Bromoallyl 2,4,6-tribromophenyl ether (BATE).

Exposure. The levels of BATE in house dust was detected close to the detection limit (Brown et al. 2014; Larsson and Berglund 2018). Also, it was reported in fish samples (Sahlstrom et al. 2015).
Toxicity. Docking and *in vitro* study results suggest that BATE is a neuronal and endocrine disruptor and an androgen receptor antagonist (Kharlyngdoh et al. 2015). Predictive QSAR modeling suggests that it may be a

developmental toxicant (See Excel Table S10). ***Biomarker.*** No information on a biomarker was found. ***Recommendation.*** There are insufficient data on toxicity and exposure. If concerns for toxicity are discovered, then a biomarker should be developed, or other exposure data measured to determine the exposure prevalence in a non-occupationally exposed U.S. population.
alpha*-Tetrabromoethylcyclohexane (α-DBE-DBCH).** ***Exposure. α-DBE-BDCH is commonly found in indoor and outdoor samples (Newton et al. 2015). The levels of *alpha*-DBE-DBCH in house dust were close to the detection limit in one study (Sahlstrom et al. 2015) and were at a median of 1.2 ng/g in another (Larsson and Berglund 2018). ***Toxicity.*** We did not find any *in vivo* or *in vitro* studies on the health effects of α-DBE-BDCH. Modeling results indicate that it may be a developmental toxicant (See Excel Table S10). ***Biomarkers.*** We found no information on biomarkers. ***Recommendation.*** Category D. Because there was insufficient information indicating toxicity, i*n vivo* animal toxicological research should determine whether there are concerns for endocrine disruption, developmental, reproductive, and neurological effects of α-DBE-BDCH. Also, HTP *in vitro* assays should be conducted. If concerns for toxicity are discovered, then a biomarker should be developed and α-DBE-BDCH measured in a non-occupationally exposed U.S. population to assess the extent of human exposure.
beta*-Tetrabromoethylcyclohexane (β-DBE-DBCH).** ***Exposure. Low levels of β-DBE-DBCH have been reported (medians ranging from 0.6-3.4 ng/g) in house dust (Larsson and Berglund 2018; Liagkouridis et al. 2017). No reports were found on exposure to β-DBE-DBCH. ***Toxicity.*** β-DBE-DBCH effects on thyroid hormone deiodinase and sulfotransferase have been reported using *in vitro* assays (Smythe et al. 2017); however, no other studies were uncovered. ***Biomarkers.*** No publications were found on a biomarker for β-DBE-DBCH. ***Recommendation.*** Category D. Because there was insufficient information indicating toxicity, β-DBE-DBCH should be tested in HTP *in vitro* assays for endocrine, developmental, reproductive, or neurotoxic effects, and if concerns for toxicity are discovered, then develop a suitable biomarker of exposure. Subsequently, it should be measured in a non-occupationally exposed U.S. population to determine the extent of exposure.
Decabromobiphenyl ethane (DBDPE). ***Exposure.*** DBDPE is present in ambient (Newton et al. 2015) and indoor air, and house dust (geometric mean, 94 µg/g) (Mitro et al. 2016). In a mega-analysis study on house dust, the median of reported medians was 143 ng/g (Larsson and Berglund 2018). A production volume of 50 to 100 X 10^6 was reported in the United States in 2016 (U.S. EPA 2016b). ***Toxicity.*** In an *in vitro* study using human tissues, effects of DBDPE on thyroid hormone deiodinase and sulfotransferase were reported (Smythe et al. 2017); however, studies were not found for other endpoints of interest to ECHO. Predictive QSAR modeling suggests it may be a developmental toxicant (See Excel Table S10). ***Biomarker.*** No information on biomarkers was found. ***Recommendation.*** Category D. Because there was insufficient information indicating toxicity, DBDPE should be further tested in HTP *in vitro* assays for endocrine, developmental, reproductive, or neurotoxic effects and if concerns for toxicity are discovered, then develop a suitable biomarker of exposure. Subsequently, it should be measured in a non-occupationally exposed U.S. population to determine the extent of exposure.
Dimethyl hydrogen phosphite (DHP). ***Exposure.*** No human exposure or environmental measurement studies were found for DHP. DHP production volume in the U.S. is very low at 0.5 -1.0 X 10^6 pounds per year (U.S. EPA 2016b). Besides its usage as a flame retardant, it is also used to produce insecticides and herbicides. ***Toxicity.*** DHP was shown to produce lung and forestomach tumors in rats (Dunnick et al. 1986; NTP 1985), but no endocrine, developmental, reproductive, and neurotoxicity studies have been reported. Modeling results suggest that DHP is a developmental toxicant (See Excel Table S10). ***Biomarker.*** The *in vivo* metabolite in rats is monomethyl hydrogen phosphite (Nomeir and Uraih 1988). However, its suitability as a biomarker of exposure has not been established. ***Recommendation.*** Category D. Investigations into the cited endpoints of toxicity using HTP *in vitro* assays are recommended, and if concerns for toxicity are discovered, then a biomarker should be developed and

DHP measured in a non-occupationally exposed U.S. population to assess the extent of exposure.
Pentabromobenzene (PBBz). ***Exposure.*** PBBz was quantified in >50% of ambient air samples at low pg/m^3 levels (Venier et al. 2012). It was detected in all fish samples tested and at quantifiable levels in nearly 80% of house dust samples (mean 8.8 ng/g and median 0.65 ng/g) (Larsson and Berglund 2018; Sahlstrom et al. 2015). Exposure likely occurs continuously through inhalation, dermal contact, and sporadically from food. ***Toxicity.*** No studies on PBBz toxicity were found. QSAR predictive modeling results suggest it may be a developmental toxicant (See Excel Table S10). ***Biomarker.*** A biomarker for PBBz in hair was reported in a Chinese study (Li et al. 2018). ***Recommendation.*** Category D. Because of the scarcity of research papers on human exposure and toxicity/health effects, we recommend testing in HTP *in vitro* assays to allow an assessment of PBBz health effects, and if concerns for toxicity are discovered, then apply the biomarker to determine prevalence of exposure in a non-occupationally exposed U.S. population.
Pentabromoethylbenzene (PBEB). ***Exposure.*** PBEB was found in the atmosphere, indoor air, house dust, and food (Bjorklund et al. 2012; Brown et al. 2014; Carignan et al. 2013; Eljarrat et al. 2009; Hoh and Hites 2005; Larsson and Berglund 2018; Liagkouridis et al. 2017; Sahlstrom et al. 2015). In one study it was not detected in breast milk. ***Toxicity.*** No toxicity data were found. QSAR predictive modeling suggests it may be a developmental toxicant (See Excel Table S10). ***Biomarker.*** PBEB was measured as the parent compound, but since it was not detected in breast milk (Muller et al. 2016), its suitability is unknown. No other biomarker data were uncovered in the literature search. ***Recommendation.*** Category D. Its prevalence in environmental media suggests that more research is needed to determine whether human exposure occurs and whether testing in HTP *in vitro* assays indicate toxicity/health effects before it is warranted for study in ECHO. Verification of the parent compound as a biomarker for exposure is recommended
Pentabromotoluene (PBT). ***Exposure.*** The levels of PBT in house dust were measured close to the detection limit (Brown et al. 2014; Larsson and Berglund 2018), and at levels at 10-100 ng/g in other studies (Brown et al. 2014; Cristale et al. 2016; Newton et al. 2015; Shoeib et al. 2012); it was also measured at very low levels in the ambient air (Venier et al. 2012) and food (Sahlstrom et al. 2015). PBT was monitored in breast milk; however, it was not detected (Muller et al. 2016). ***Toxicity.*** Toxicity publications were scarce. In an *in vitro* study, PBT activated AhR and, thus, is a possible endocrine disruptor (Brown et al. 2004). QSAR modeling results suggest PBT may be a developmental toxicant (See Excel Table S10). ***Biomarker.*** Since PBT was not detected in breast milk, it should be determined whether it is a valid biomarker. ***Recommendation.*** Category D. Validation of a biomarker of exposure for PBT in biofluids is recommended. Testing in HTP *in vitro* assays is recommended to determine whether PBT has endocrine, developmental, reproductive, neurotoxic, or obesity effects, and if concerns for toxicity are discovered, then a biomarker should be developed and the prevalence in a non-occupationally exposed U.S. population should be determined.
Tetrabromo-*o*-chlorotoluene (TBCT). ***Exposure.*** TBCT was measured in house dust at a detection frequency of 16% with a mean of 0.60 ng/g (Larsson and Berglund 2018; Shoeib et al. 2012); however, no other papers were found that monitored it in other environmental media or in human biofluids. Production volume for TBCT has not been reported. ***Toxicity and Biomarker.*** No papers were found on toxicity/health effects studies for TBCT or on suggested biomarkers. ***Recommendation.*** Category D. Additional research is needed to establish whether exposure to TBCT occurs in humans and to determine whether TBCT toxicity is relevant to ECHO using HTP *in vitro* assays for endocrine, developmental, neurotoxic, and obesity effects, and if concerns for toxicity are discovered, then a biomarker should be developed and it should be measured in a non-occupationally exposed U.S.

population to determine exposure prevalence.

Tetrabromophthalic anhydride (TBPA).

Exposure. No studies were found on human exposure or environmental levels for TBPA.

Toxicity. An *in vitro* study suggests that TBPA is an endocrine disruptor by activating AhR (Brown et al. 2004).

Biomarker. No information.

Recommendation. Category D. We recommend testing in HTP *in vitro* assays for endocrine, developmental, neurotoxic, and obesity effects, and if concerns for toxicity are discovered, then a biomarker should be developed and it should be measured in a non-occupationally exposed U.S. population to determine exposure prevalence.

Dibromostyrene (DBS); Dimethyl propyl phosphonate (DMPP); Dimethyl N-methylolphosphonopropionamide (DNMPP); Ethylene bis(tetrabromo) phthalmide (ETBP); 1,2-Bis(2,4,6-tribromophenoxy) ethane (TBE); and Tetrabromophthalate diol (TBPD).

We did not find research publications on exposure, environmental media levels, toxicity/health effects, or biomarkers for DMPP, ETBP, DBS, and TBPD. Modeling results suggest that DMPP, and ETBP are developmental toxicants (See Excel Table S10).

Recommendation. Category D. We recommend that they are tested in HTP *in vitro* assays for endocrine, developmental, neurotoxic, and obesity effects, and if concerns for toxicity are discovered, then a biomarker should be developed, and exposure prevalence measured in a non-occupationally exposed U.S. population.

Alternate Plasticizers

Category B:

2,2,4-Trimethyl 1,3-pentanediol monoisobutyrate (TXIB).

Exposure. TXIB levels have been reported in the indoor air of homes (1.2 to 20.8 µg/m^3) (Takeuchi, S et al. 2014) and commercial buildings (DT 100%, geometric mean 1.09 µg/m^3) (Wu et al. 2011). An EPA predicted median exposure value in the 10^{-4} mg/kg-bw/day range was reported (See Excel Table S2), ranking in the top 10 of chemicals with predicted exposure.

Toxicity. One school study found an association between the indoor air concentration of TXIB and asthmatic symptoms in school children (Naimi 2013). Agonistic affects ER1, ER2, and antagonistic effects TRβγ were observed *in silico* (Simon et al. 2016). This compound activated 4 *in vitro* obesity assays (See Excel Table S9). QSAR modeling results suggest TXIB is a developmental toxicant (See Excel Table S10).

Biomarker. No information on biomarkers were found.

Recommendation. Category B. Given the initial report of an effect of TXIB on airways, and its reported effects on the endocrine system, a biomarker should be developed and its exposure prevalence in a non-occupationally exposed U.S. population should be determined.

Acetyl tributyl citrate (ATBC).

Exposure. Primary and secondary ATBC metabolites have been reported in urine and in fingernails, with DF ranging from 46-95% (Alves et al. 2017). ATBC levels in dust have been reported (mean 80 µg/g dust, detected in 40% of samples) and in indoor air (mean 19 ng/m^3 and in 27% of samples) (Fromme et al. 2016). In a mega-analysis of house dust, the median of reported medians was 106 ng/g (Larsson and Berglund 2018). Levels of ATBC found in cheese wrapped in ATBC-containing wrapping were 2-8 mg/kg (Castle et al. 1988). ATBC was detected in bottled sake samples (3-7 ug/g), possibly migrating from the gasket of the bottle cap. It was low in several food samples, with their no-observable-effect-level (NOEL) below the guideline levels as food additives (Tsumura et al. 2002). An EPA predicted median exposure value in the 10^{-4} mg/kg-bw/day range was reported (See Excel Table S2), ranking in the top 100 chemicals with predicted exposures.

Toxicity. Adult CD-1 females (n=7-8 per treatment) were dosed orally with 5 or 10 mg kg^{-1} day^{-1} ATBC, daily for 15 days, and then bred. ATBC exposure did not alter body weights, estrous cyclicity, or gestational and litter parameters. ATBC at 10 mg kg^{-1} day^{-1} exhibited endocrine disruption (Rasmussen et al. 2017). ATBC activated 4 obesity assays in HTP *in vitro* testing (See Excel Table S9).

Biomarker. Primary and secondary ATBC metabolites have been measured in fingernails and urine (Alves et al. 2017).

Recommendation. Category B. Biomonitoring is deferred pending the determination of whether the primary ATBC metabolite best serves as a biomarker of exposure. Its measurement in a non-occupationally exposed U.S. population is recommended to determine exposure prevalence.

Tri-2-ethylhexyl trimellitate (TETM).

Exposure. TETM was measured in house dust (7-107 µg/g with a 63% detection frequency) and not detected in indoor air (Fromme et al. 2015). No exposure data were discovered. An EPA predicted median exposure value in the 10^{-5} mg/kg-bw/day range was reported (See Excel Table S2).

Toxicity. Potential endocrine disrupting activity of TETM against human sex hormone-binding globulin (SHBG) using *in silico* approaches was reported; these results suggest that it has potential to engage the residues of SHBG and thus interfere in its steroid homeostatic function (Sheikh et al. 2016). HTP *in vitro* assays indicated this compound is an estrogen agonist (See Excel Table S9). QSAR modeling results suggest TETM is a developmental toxicant (See Excel Table S10).

Biomarker. No reports on biomarkers of TETM were found.

Recommendation. Category B. Since there is an indication of endocrine disruption and developmental toxicity by TETM, biomonitoring is deferred pending additional development of a biomarker and measurement in biospecimens of a non-occupationally exposed U.S. population to determine the extent of exposure.

Category C:

o-Toluene sulfonamide (OTSA).

Exposure. Low OTSA intakes for drinking water, meat, milk, and air have been reported (2E-6 mg/kg/d, 2E-11 mg/kg/d, 3E-10 mg/kg/d, and 1E-10 mg/kg/d, respectively) (Stuer-Lauridsen et al. 2001).

Toxicity. Doses of 0-250 mg/kg throughout gestation and lactation yielded a dose-response for bladder calculi in 21-day-old pups and 105-day-old rats. In another study, OTSA was reported to be teratogenic (Stuer-Lauridsen et al. 2001). Predictive QSAR modeling results suggest that OTSA is a developmental and reproductive toxicant (See Excel Table S10).

Biomarker. When dosed orally at 0.2-0.4 mg/kg-bw, OTSA was excreted more slowly in humans than in rats, with 50% excreted after 24 hours, 80% within 48 hours, and <1% found in the feces. The main urine metabolites reported are 2-sulfamoylbenzyl alcohol and its sulfates and glucuronic conjugates (35%), saccharin (35%), 2-sulfamoylbenzoic acid (4%), and N-acetyltouluene-2-sulphonamide (2%) (Stuer-Lauridsen et al. 2001).

Recommendation. Category C. There is concern for toxicity; a biomarker for exposure should be developed and OTSA measured in a non-occupationally exposed U.S. population to determine its exposure prevalence.

Category D:

Dioctyl terephthalate (DOTP).

Exposure. No studies were found on exposure or environmental levels.

Toxicity. Timed-pregnant rats were administered DOTP (750 mg/kg-bw/2.5 mL vehicle) from gestational day 14 (sperm positive = GD1) through postnatal day 3. Adult males exposed perinatally to DOTP were unaffected. (Gray Jr et al. 2000). QSAR modeling results suggest DOTP is a developmental toxicant (See Excel Table S10).

Biomarker. There was no information on biomarkers.

Recommendation. Category D. Biomonitoring is deferred pending additional data on exposure. We recommended testing in HTP in vitro assays for endocrine, developmental, neurotoxic, and obesity effects, and if concerns for toxicity are discovered, then a biomarker should be developed and DOTP should be measured in a non-occupationally exposed U.S. population to establish exposure prevalence.

Aromatic Amines

Category A:

2,4-Diaminotoluene (2,4-toluenediamine).

Exposure. The production estimate in 2002 was between 10,000 and 500,000 pounds in the USA (U.S. EPA 2012). Studies have reported the occurrence of this compound in textiles, polyurethane toy samples, and air (Abe et al. 2016; Kowalska and Jezewska 2017). Migration of 2,4-diaminotoluene from polyimide kitchenware was documented (Sanllorente et al. 2016). An EPA predicted median exposure value in the 10^{-6} mg/kg-bw/day range was reported (See Excel Table S2).

Toxicity. Rats exposed to 2,4-diaminotoulene developed a variety of tumors in the liver, mammary glands, lungs, and leukemia. In HTP *in vitro* assay screening, this compound activated an endocrine assay (See Excel Table S9). It is on the Hazardous 100+ List of Chemicals of High Concern as a carcinogen (SaferChemicals 2017).

Biomarker. A urinary elimination rate of <0.01-5.7 µg/hr was reported for 2,4-diaminotoluene in workers exposed to polyurethane foam and glue (Dalene et al. 1997). A major urinary metabolite of the compound was 2,4-diamino-5-hydroxytoluene (IARC 1978), (Zhang et al. 2013), (De Palma et al. 2012), and one study reported a method to measure urinary concentrations of this compound.

Recommendation. Category A. A biomarker exists, and we recommend it be measured in a non-occupationally exposed U.S. population to establish exposure prevalence.

4,4'-Diaminodiphenylmethane [methylenedianiline (MDA)].

Exposure. MDA is a high-production-volume chemical (4 million metric tons/year) (Carvajal-Diaz 2015). This compound is present in food-contact utensils (Trier et al. 2010). Occurrence of this compound was reported in indoor air (Mirmohammadi et al. 2009). An EPA predicted median exposure value in the 10^{-6} mg/kg-bw/day range was reported (See Excel Table S2).

Toxicity. Occupational exposures have been linked to liver damage (Giouleme et al. 2011). This compound was identified as IARC class 2B—a possible human carcinogen (Zhang et al. 2006). In the HTP assays, MDA activated 2 endocrine and 2 obesity assays (See Excel Table S9). Predictive modeling suggests that it is an endocrine disruptor (See Excel Table S11) and developmental toxicant (See Excel Table S10). It is on the Hazardous 100+ List of Chemicals of High Concern as a carcinogen (SaferChemicals 2017).

Biomarker. While there are no reports of environmental exposure measurements, this compound was measured in urine of occupationally exposed populations (Weiss et al. 2011). Biomarkers of exposure to MDA have been reviewed (Sabbioni et al. 2007). Urine samples collected at the end of a work shift from workers exposed to MDA contained concentrations ranging from 43-236 µg/g creatinine (Dalene et al. 1995; IARC 1986; Kusters 1992). While the parent compound was measured in urine, several metabolites have also been reported to occur in urine (Schutze et al. 1995). This compound was reported to form albumin and hemoglobin adducts, which have been measured in urine (Sepai et al. 1995b).

Recommendation. Category A. There is evidence that MDA occurs in indoor environments, has potential sources of human exposure, and there are concerns for its toxicity. A biomarker is available, and it is recommended that MDA be measured in non-occupationally exposed U.S. population to determine if exposure is prevalent.

Category B:

2-Methoxy-5-methylaniline (MMA or p-Cresidine).

Exposure. Microbial transformation of Sudan III, a food colorant, resulted in the formation of MMA (Zanoni et al. 2013). This compound was reported to occur in commercial food red colorant (Takeda et al. 1993). Studies have reported the occurrence of MMA in textile-dyeing industry sludge and wastewater (Ning et al. 2015; van der Zee and Villaverde 2005). An EPA predicted median exposure value in the 10^{-6} mg/kg-bw/day range was reported (See Excel Table S2).

Toxicity. Laboratory animal exposure to MMA produced bladder and liver carcinomas (Delker et al. 2000; Hursting et al. 2009; Sasaki et al. 1998). There is sufficient evidence of carcinogenicity in animal exposure studies, and this compound is classified by IARC as group 2B—a possible carcinogen (IARC 1993). Using HTP *in vitro* testing, 2 endocrine assays were activated (See Excel Table S9).

Biomarker. No biomarker information was found.

Recommendation. Category B. Biomonitoring is deferred pending development of a biomarker and measuring the prevalence of exposure in a non-occupationally exposed U.S. population.

4,4'-Methylenebis(2-methylaniline) (also known as 4,4'-Methylenedi-o-toluidine or MBOT).

Exposure. This compound is also referred to as dimethyldiaminophenyl methane or 4,4'-diamino-3,3'-dimethylphenylmethane. Several isomeric forms of this compound were in production. MBOT was reported to occur in paper napkins and textiles (Yang et al. 2016; Yavuz et al. 2016). An EPA predicted median exposure value in the 10^{-6} mg/kg-bw/day range was reported (See Excel Table S2).

Toxicity. This compound induced hepatocellular carcinomas in rats and dogs and neoplasms of the lung, mammary gland, and skin of rats (IARC 1974; Stula et al. 1975). IARC classified this compound as a group 2B, possible carcinogen (IARC 1993). HTP *in vitro* testing results indicate that this compound activated neurotoxic, 3 endocrine, and 3 obesity assays (See Excel Table S9). Predictive QSAR modeling results suggest MBOT is an endocrine disruptor (See Excel Table S9) and a developmental toxicant (See Excel Table S10). It is on the Hazardous 100+ List of Chemicals of High Concern as a carcinogen (SaferChemicals 2017).

Biomarker. No biomarker information was found.

Recommendation. Category B. There are concerns for its toxicity. Biomonitoring is deferred pending the development of a biomarker and its measurement in non-occupationally exposed U.S. population to establish exposure prevalence.

4,4'-Oxydianiline (ODA) [4,4'-oxybis-benzenamine or diaminodiphenyl ether].

Exposure. This is a high-production-volume chemical (1-10 million pounds in 2002). Occurrence of ODA in food packaging, tobacco smoke, and hair dye was shown (Lizier and Boldrin Zanoni 2012; Schubert et al. 2011; Yang et al. 2016). The EPA predicted median exposure is high at 10^{-5} mg/kg-bw/day range (See Excel Table S2).

Toxicity. ODA is a possible human carcinogen, and enough evidence exists on the carcinogenicity of this compound in animal studies. Suppression of thyroid and pituitary gland activity was reported in rats (Murthy et al. 1985). Two endocrine and 2 obesity assays were activated in HTP screening studies (See Excel Table S9). QSAR predictive modeling suggests that ODA may be a reproductive toxicant (See Excel Table S10). It is on the Hazardous 100+ List of Chemicals of High Concern as a carcinogen (SaferChemicals 2017).

Biomarker. Reports of biomarkers of exposure to this chemical in the general population are not available.

Recommendation. Category B. Considering its widespread production and usage, and concern for its toxicity, studies are needed to develop the methods for the analysis of this compound. Also, we recommend its measurement in a non-occupationally exposed U.S. population to determine its exposure prevalence.

Category C:

2-Amino-5-azotoluene.

Exposure. Exposure and environmental measurements have not been reported for this compound in the U.S. population. An EPA predicted median exposure value in the 10^{-5} mg/kg-bw/day range was reported (See Excel Table S2).

Toxicity. This compound is reported as a hepatocarcinogen (Furihata et al. 2016; Liu et al. 1969). IARC classified this compound as a possible human carcinogen (group 2B) (IARC 1993). This compound activated neurotoxicity, 3 endocrine, and 6 obesity HTP *in vitro* assays (See Excel Table S9). QSAR predictive modeling suggests that it is an endocrine disruptor (See Excel Table S11).

Biomarkers. No methods are available for monitoring. Toxicokinetics and metabolism of this compound are not well understood.

Recommendation. Category C. There are concerns about its toxicity based on results from *in vitro* tests. Even though the exposure prediction is high, we recommend that a biomarker be developed, and it should be measured in a non-occupationally exposed U.S. population to determine prevalence of exposure.

2,3-Dichloroaniline.

Exposure. Whereas 3,4-, and 2,5-dichloroanilines have been widely studied, this isomer is not well studied in terms of human exposures. 2,3-Dichloroaniline is widely used in the manufacture of dyes, pesticides, and pharmaceuticals. This compound is also an impurity in 1,2-dichlorobenzene production. The herbicide propanil is rapidly hydrolyzed to yield 2,3-dichloroaniline. This compound was detected in surface waters in the Netherlands (Wegman and Dekorte 1981).

Toxicity. Dichloroanilines elicit methemoglobinemia in exposed rodents and are also nephrotoxic (Lo et al. 1990). Predictive modeling suggests that it is a developmental toxicant (See Excel Table S10).

Biomarker. Biomarker(s) have not been established.
Recommendation. Category C. There is concern for toxicity; a biomarker should be developed and the compound measured in a non-occupationally exposed U.S. population to demonstrate the prevalence of exposure.

2,5-Dichloroaniline.

Exposure. The exposure pattern and toxicity of 2,5-dichloroaniline is expected to be like those reported for 3,4-dichloroaniline (in main text). There is a heavy global demand for this compound. Degradation of tattoo pigments (Red 22 and Red 9) was shown to yield 2,5-dichloroaniline (Engel et al. 2006). 2,5-Dichloroaniline is reported to occur in rivers (Wegman and Dekorte 1981) at concentrations of several tens of ng/L (Bester et al. 1998). The general population may be exposed to 2,5-dichloroaniline via drinking water (Fattore et al. 1998) and dermal contact through dye products. No biomonitoring studies have reported exposures. An EPA predicted median exposure value in the 10^{-7} mg/kg-bw/day range was reported (See Excel Table S2).
Toxicity. This compound causes nephrotoxicity in rats (Lo et al. 1990). QSAR predictive modeling suggests that it is a developmental toxicant (See Excel Table S10).
Biomarker. 2,5-Dichloroaniline is also a metabolic product of vinclozolin and pentachloronitrobenzene (fungicides) (Tas et al. 2006; Zober et al. 1995), which will make establishment of a specific biomarker more difficult.
Recommendation. Category C. There is enough concern for toxicity. A method should be developed for monitoring exposure to this compound, and the exposure prevalence in a non-occupationally exposed U.S. population should be determined.

2-Nitro-1,4-phenylenediamine (2NPPD) (also known as 2,5-diaminonitrobenzene).

Exposure. This compound is closely related p-phenylenediamine and 4-nitro-o-phenylenediamine. The general population may be exposed to this compound through hair color products. Furthermore, occupational exposure of hair dressers to this compound was suggested. This is a high-production-volume chemical (10,000-500,000 pounds) (U.S. EPA 2002b). An EPA predicted median exposure value in the 10^{-8} mg/kg-bw/day range was reported (See Excel Table S2).
Toxicity. The IARC classified this compound as a group 3 carcinogen (IARC 1993). This compound was reported to affect the cornea of neonates following exposure of pregnant rats (Erbagci et al. 2010). Predictive modeling suggests that it may be a developmental toxicant (See Excel Table S10).
Biomarker. Urinary metabolites of this compound following exposures in rats were: unchanged 2NPPD, acetylated 2NPPD, and conjugates of 2NPPD (Yourick and Bronaugh 2000).
Recommendation. Category C. The is concern for developmental toxicity; however, there is insufficient exposure data and a biomarker needs to be developed. 2NPPD should be measured in a non-occupationally exposed U.S. population to determine exposure prevalence.

Category D:

1,2,4-Benzenetriamine, N'-phenyl (also known as 2,4-diaminodiphenylamine).

Exposure. This compound is used in hair dyes.
Toxicity. No information on the endpoints of toxicity relevant to ECHO was found for this compound.
Biomarker. No information on a biomarker was found.
Recommendation. Category D. We recommend it be deferred for additional data from testing in HTP *in vitro* assays for endocrine, developmental, neurotoxic, and obesity effects; and if concerns for toxicity are discovered, then a biomarker should be developed; and it should be measured in a non-occupationally exposed U.S. population to assess the extent of exposure.

1,3-Benzodioxol-5-amine.

Exposure. This is also known as methylenedioxyaniline. No data exist regarding their production, usage, environmental occurrence, and human exposures.
Toxicity. No *in vivo* or *in vitro* toxicity data were found. Predictive modeling suggests this compound is a carcinogen (See Excel Table S10).
Recommendation. Category D. We recommend it be deferred for additional data from testing in HTP *in vitro* assays for endocrine, developmental, neurotoxic, and obesity effects; and if concerns for toxicity are discovered, then development of a biomarker and measurement in a non-occupationally exposed U.S.

population to assess the extent of exposure should be undertaken.

2,4,6-Tribromoaniline (TBA).

Exposure. TBA was reported to occur in colorants such as D&C Red Nos. 21, 21L, and 22 (Eosin Y) at several hundreds of parts per million (Weisz et al. 2004). A study reported the occurrence of this compound in river sediments in Germany (Schwarzbauer et al. 2001).

Toxicity. Characterization of toxicity effects of interest to ECHO was not reported.

Biomarker. Monobromoanilines have been measured in urine of rats (Duckett et al. 2015; Nicholson et al. 2000).

Recommendation. Category D. There is insufficient toxicity information to judge whether it warrants study in ECHO. We recommend HTP *in vitro* testing for endocrine, developmental, neurotoxic, and obesity effects, and if concerns for toxicity are discovered, then we recommend measuring this compound in a non-occupationally exposed U.S. population to demonstrate the prevalence of exposure.

2,6-Toluenediamine (2,6-diaminotoluene).

Exposure. Exposure patterns of this compound may be like that of 2,4-diaminotoluene, as reported above. Production was reported to be 10,000-500,000 pounds (U.S. EPA 2002b). The literature contains reports of the analysis of 2,4- and 2,6-toluenediamines in air (Dalene et al. 1988; Geens et al. 2012), hair dyes (Johansson et al. 1981), polyurethane foams (Hirayama et al. 1985), and in body fluids (Sepai et al. 1995a; Skarping et al. 1994). Occupational exposure of hair dressers to this compound was reported (Geens et al. 2016; Goebel et al. 2018; Lind et al. 1997). Exposure from polyurethane breast implants was studied (Sepai et al. 1995a). An EPA predicted median exposure value in the 10^{-7} mg/kg-bw/day range was reported (See Excel Table S2).

Toxicity. This compound was shown to be carcinogenic and mutagenic. No other toxicological endpoints have been reported.

Biomarker. 2,4-Toluenediamine and 2,6-toluenediamine have been identified in urine of persons exposed to toluene diisocyanates (TDI) used in the polyurethane industry (Collins 2002; Dalene et al. 1997). The CDC was developing methods for the analysis of this compound (Bhandari et al. 2016; Sennbro et al. 2003).

Recommendation. Category D. Biomonitoring is deferred pending additional data from HTP *in vitro* assays for endocrine, developmental, neurotoxic, and obesity effects. If concerns for toxicity are discovered, then, a biomarker exists, and the extent of exposure in a non-occupationally exposed U.S. population should be determined.

2-Aminotoluene-5-methylbenzenesulphonic acid (PTMS/PTMSA).

Exposure. A closely related compound is 6-aminotoluene-3-sulfonic acid. No data exist on the environmental or human exposures. An EPA predicted median exposure value in the 10^{-6} mg/kg-bw/day range was reported (See Excel Table S2).

Toxicity. No toxicity data were found. QSAR predictive modeling suggests this compound is a developmental toxicant (See Excel Table S10).

Biomarker. Use of the HPLC method for analysis of this compound was reported (Perez-Gonzalez et al. 2015). No biomarker was established.

Recommendation. Category D. We recommend HTP in vitro assays for endocrine, developmental, neurotoxic, and obesity effects. If concerns for toxicity are discovered, then a biomarker should be developed, and this compound should be measured in a non-occupationally exposed U.S. population to demonstrate the prevalence of exposure.

2-Biphenylamine (2-aminobiphenyl).

Exposure. The production of 4-aminobiphenyl was ceased in the USA in the 1950s, but it is probable that 2-biphenylamine is still being produced. Exposure to 2-aminobiphenyl arises through contact with chemical dyes and inhalation of cigarette smoke (Seyler and Bernert 2011). 2-Aminobiphenyl was found at concentrations of up to 310 ng/g in hair dyes (Turesky et al. 2003). Because this compound can be present in hair dyes and tobacco smoke, exposure of the general population is inevitable. An EPA predicted median exposure value in the 10^{-7} mg/kg-bw/day range was reported (See Excel Table S2).

Toxicity. A closely related compound, 4-biphenylamine (also known as 4-phenylaniline), is a well-studied compound and is a known carcinogen. Another closely related compound, 4,4-diaminobiphenyl (benzidine), is a known human carcinogen. Although 4-aminobiphenyl was a known carcinogen (especially bladder

cancer), little research has been done on 2-aminobiphenyl. The little evidence suggests that this compound is non-carcinogenic (Chung and Adris 2003), although a few studies suggest that this compound can cause DNA damage (Chen et al. 2003; Wang et al. 2006). QSAR predictive modeling suggests that it is a developmental toxicant (See Excel Table S10).

Biomarker. Methods for the analysis of 2-aminobiphenyl in urine exist in the literature (Seyler and Bernert 2011).

Recommendation. Category D. Even though there is evidence of exposure, biomonitoring is deferred pending test results from HTP *in vitro* assays for endocrine, developmental, neurotoxic, and obesity effects. If concerns for toxicity are discovered, then the prevalence of exposure in a non-occupationally exposed U.S. population should be determined.

2-Bromo-4,6-dinitroaniline.

Exposure. This compound was reported to occur in sediment, as a breakdown product of azo dyes (Maguire 1992). Recent studies have shown that this compound is present in textiles at concentrations of up to 400 µg/g (Luongo et al. 2016). An EPA predicted median exposure value in the 10^{-7} mg/kg-bw/day range was reported (See Excel Table S2).

Toxicity. Toxicity information for this compound is sparse. This compound was shown to be mutagenic in the Ames test (Kawai et al. 1986).

Biomarker. This compound was reported to be excreted in urine as sulfate conjugates, with a half-life of <10 hours in rats.

Recommendation. Category D. There is insufficient toxicity information to judge whether it warrants study in ECHO. We recommend HTP *in vitro* testing for endocrine, developmental, neurotoxic, and obesity effects, and if concerns for toxicity are discovered, then we recommend development of a biomarker and measuring this compound in a non-occupationally exposed U.S. population to demonstrate the prevalence of exposure.

2-Chloro-1,4-diaminobenzene sulfate (also known as 2-chloro-1,4-phenylenediamine sulfate).

Exposure. This compound is a derivative of p-phenylenediamine (PPD), a compound frequently used in hair dye formulation. This compound is also a derivative of 1,4-diaminobenzene. The general population may be exposed to this compound by dermal contact, using hair dyes containing 1,4-benzenediamine. An EPA predicted median exposure value in the 10^{-7} mg/kg-bw/day range was reported (See Excel Table S2).

Toxicity. This compound is a skin sensitizer and allergenic; forms methemoglobin. Many related PPDs are genotoxic (Chye et al. 2008).

Biomarker. No biomarker information was found.

Recommendation. Category D. There is insufficient toxicity information to judge whether it warrants study in ECHO. We recommend HTP in vitro testing for endocrine, developmental, neurotoxic, and obesity effects, and if concerns for toxicity are discovered, then we recommend developing a biomarker and measuring this compound in a non-occupationally exposed U.S. population to demonstrate the prevalence of exposure.

2-Chloro-4,6-dinitroaniline.

Exposure. No data exist about the production, usage, environmental occurrence, and human exposures.

Toxicity. No toxicity data were found. Predictive modeling suggests this compound is a developmental toxicant (See Excel Table S10).

Recommendation. Category D. We recommend it be deferred for additional data from testing in HTP *in vitro* assays for endocrine, developmental, neurotoxic, and obesity effects; and if concerns for toxicity are discovered, then we recommend development of a biomarker and measurement in a non-occupationally exposed U.S. population to assess the extent of exposure.

2-Naphthylamine.

Exposure. An isomer of this compound, 1-naphthylamine is commercially produced but is reported to be less toxic than 2-naphthylamine. 2-Naphthylamine was found in cigarette smoke (0.02 µg/cigarette) and coal tar (Stabbert et al. 2003; Takemura 1965). 2-Naphtylamine was reported to occur in the urine of smokers and non-smokers (Grimmer et al. 2000; Riffelmann et al. 1995). An EPA predicted median exposure value in the 10^{-6} mg/kg-bw/day range was reported (See Excel Table S2).

Toxicity. Evidence of carcinogenicity of 2-naphthylamine exists, and this compound is classified as a group 1 carcinogen. Individuals involved in the purification of 2-naphtylamine in the dye industry developed bladder

cancer (Klaassen 2001). A number of animal studies clearly point to liver and bladder carcinogenicity of this compound. Acute exposure to this compound results in methemoglobinemia and hemorrhagic cystitis. It is on the Hazardous 100+ List of Chemicals of High Concern as a carcinogen (SaferChemicals 2017).
Biomarker. 2-Naphtylamine metabolism occurs through oxidation to 2-amino-1-naphthol. At least 10 different metabolites have been identified in the urine of rats and rabbits exposed to 2-naphthylamine. Although 2-naphthylamine is no longer produced in the United States, exposure from several sources, including tobacco smoke, is expected.
Recommendation. Category D. Obtaining data from HTP *in vitro* assays for endocrine, developmental, neurotoxic, and obesity effects is recommended; and if concerns for toxicity are discovered, then research is needed on a biomarker(s) to select appropriate metabolite(s) for biomonitoring in humans and measuring in a non-occupationally exposed U.S. population.

3-Nitroaniline.
Exposure. A related compound, 4-nitroaniline, is widely produced and used in the United States (in 2002, 1-10 million pounds). An EPA predicted median exposure value in the 10^{-7} mg/Kg-bw/day range was reported (See Excel Table S2).
Toxicity. Acute exposure to this compound can cause methemoglobinemia and cyanosis. Chronic exposure to this compound can induce liver damage.
Biomarker. Exposure to 3-nitroaniline yields 2-amino-4-nitrophenol, m-phenylenediamine, and 4-amino-2-nitrophenol in rabbits (Goodwin 1976). 3-Nitroaniline was reported as a rat urinary metabolite of 1,3-dinitrobenzene (Nystrom and Rickert 1987).
Recommendation. Category D. Testing results from HTP *in vitro* assays for endocrine, developmental, neurotoxic, and obesity effects is needed. Although no biomonitoring studies exist on the occurrence of these chemicals in human populations, the potential for exposures is high, and if concerns for toxicity are discovered, then methods need to be developed to assess exposures in a non-occupationally exposed U.S. population.

4,4´-Methylenebis(2-chloroaniline) (MOCA).
Exposure. In 2002, the U.S. production was estimated to be between 1 and 10 million pounds (U.S. EPA 2006c). Studies have reported the occurrence of MOCA in air (Brede et al. 2003; Oostdyk et al. 1994). The EPA predicted median exposure value is high at 10^{-5} mg/kg-bw/day range (See Excel Table S2).
Toxicity. This compound was reported to be carcinogenic in laboratory animal models (Cheung et al. 1996; Reid et al. 1998; Wang et al. 2017). IARC considers MOCA as a group 1 carcinogen. MOCA is on the Hazardous 100+ List of Chemicals of Concern as a carcinogen (SaferChemicals 2017). The California Occupational Safety and Health Administration's reference value for MOCA in urine was 100 µg/L (Liu et al. 2005). Predictive QSAR modeling suggests MOCA is an endocrine disruptor (See Excel Table S11) and a developmental toxicant (See Excel Table S10).
Biomarker. MOCA is excreted in urine, with a half-life of 23 hours in humans. Methods for the analysis of MOCA in urine have been proposed (Robert et al. 1999; Shih et al. 2007; Takeuchi, A et al. 2014), and biomonitoring studies have been performed among polyurethane foam industry workers (Keen et al. 2012). Urinary MOCA levels as high as 15,700 µg/g creatinine have been reported in a bladder cancer patient of a polyurethane factory.
Recommendation. Category D. There is insufficient toxicity information on the endpoints of concern in ECHO. Test results from HTP *in vitro* assays for endocrine, developmental, neurotoxic, and obesity effects is needed. A biomarker of exposure exists, and if concerns for toxicity are discovered, then we recommend that measurement of MOCA in a non-occupationally exposed U.S. population be conducted to determine its exposure prevalence.

4-Chloro-2-nitroaniline.
Exposure. This compound was reported to be produced at 10,000-500,000 tons (U.S. EPA 2002b). A closely related compound is 2-chloro-4-nitroaniline, and another related compound, 2,6-dichloro-4-nitroaniline, was detected in textiles at concentrations of 1-576 µg/g (Luongo et al. 2016). An EPA predicted median exposure value in the 10^{-7} mg/kg-bw/day range was reported (See Excel Table S2). Potential for human and environmental exposure exists.
Toxicity and Biomarker. Toxicity and biomarker(s) have not been established.

Recommendation. Category D. Additional toxicity information is needed to judge whether it warrants study in ECHO. We recommend HTP *in vitro* assay testing for endocrine, developmental, neurotoxic, and obesity effects; and if concerns for toxicity are discovered, then we recommend developing a biomarker and measuring this compound in a non-occupationally exposed U.S. population to demonstrate the prevalence of exposure.

Aniline (benzenamine or aminobenzene).

Exposure. Aniline is a very high-production-volume chemical (1.9 billion pounds) (U.S. EPA 2002b). Aniline was detected in drinking and surface waters at ng/L concentrations (Jurado-Sanchez et al. 2012; Wegman and Dekorte 1981). Aniline was measured in urine of a general population in Germany at µg/L levels (Kutting et al. 2009). An EPA predicted median exposure value in the 10^{-5} mg/kg-bw/day range was reported (See Excel Table S2).

Toxicity. Aniline was reported as a probable human carcinogen. Excess bladder cancer among rubber industry workers was linked to aniline exposure (Ward et al. 1996). Predictive QSAR modeling suggests aniline is a developmental toxicant (See Excel Table S10). It is on the Hazardous 100+ List of Chemicals of High Concern as a carcinogen (SaferChemicals 2017).

Biomarker. Aniline is excreted as is and as a metabolite, aminophenol, in urine. Furthermore, acetanilide and acetaminophen were identified as metabolites of aniline (Dierkes et al. 2014). Aniline is excreted in urine and was used as a biomarker of exposure in the general population of Germany (Kutting et al. 2009) and in occupationally exposed rubber industry workers (Ward et al. 1996).

Recommendation. Category D. Findings suggest that aniline exposure may occur and there is a biomarker of exposure. It is recommended that it be deferred pending additional data that include testing with HTP *in vitro* assays for endocrine, developmental, neurotoxic, and obesity effects, and if concerns for toxicity are discovered, then we recommend measuring aniline in a non-occupationally exposed U.S. population to determine exposure prevalence.

N,N,4-Trimethylaniline (also known as N,N-Dimethyl-p-toluidine or DMPT).

Exposure. This compound is used as a polymerization accelerator in the manufacture of bone cements (e.g., artificial fingernail) and dental material. It is a high-production-volume chemical (500,000-1 million pounds in 2014) (U.S. EPA 2016b). A closely related isomer of this compound is N,N-dimethylaniline. Potential for widespread human exposure through its use in dental materials and bone cements (hip and knee joints) exists. An EPA predicted median exposure value in the 10^{-8} mg/kg-bw/day range was reported (See Excel Table S2).

Toxicity. Human exposure to DMPT has resulted in methemoglobinemia and allergic responses (Ghanbari K. 2004). Significantly increased incidences of nephrotoxicity and renal failure have been reported in rodents from exposure to this compound (Dix et al. 2007; National Toxicology Program 2012). Studies of the NTP showed an evidence for carcinogenicity, especially hepatocellular carcinoma (Dunnick et al. 2016). This compound also forms hemoglobin adducts (Birner 1988).

Biomarker. Rats exposed to this compound produced several urinary metabolites, including p-(N-acetylhydroxyamino)hippuric acid, N-methyl-p-toluidine, and the parent compound DMPT. DMPT metabolism is similar to that of N,N-dimethylaniline (Kim et al. 2007). Methods for the analysis of trimethylanilines exist (DeBruin et al. 1998).

Recommendation. Category D. We recommend it be deferred for additional data from testing in HTP *in vitro* assays for endocrine, developmental, neurotoxic, and obesity effects; and if concerns for toxicity are discovered, then development of a biomarker and measurement in a non-occupationally exposed U.S. population to assess the extent of exposure.

P-chloroaniline (4-chloroaniline).

Exposure. P-chloroaniline is a widespread soil contaminant as a degradation product resulting from the use of phenylurea herbicide. 4-Chloroaniline is an ingredient of chlorhexide, which is an antimicrobial disinfectant for soft contact lenses and mouthwashes (Below et al. 2017). This compound was measured in urine of the general population in Germany (Kutting et al. 2009). 4-Chloroaniline was reported to occur in drinking water, surface water, and in food packaging (Jurado-Sanchez et al. 2012; OuYang et al. 2014; Wegman and Dekorte 1981). An EPA predicted median exposure value in the 10^{-6} mg/kg-bw/day range was reported (See Excel Table S2).

Toxicity. The U.S. EPA and IARC classified this compound as a group B2 probable human carcinogen. This compound produces methemoglobin in animal models (Pizon et al. 2009). In exposed rats, this compound produced spleen tumors (IPCS 2005). Further, nephrotoxic and hepatotoxic potential of this compound was documented in animal models (IPCS 2005). It is on the Hazardous 100+ List of Chemicals of High Concern as a carcinogen (SaferChemicals 2017).

Biomarker. Following exposure, both the parent compound and the metabolites p-aminophenol, 2-amino-5-chlorophenol, and 4-chlorooxanilic acid were found in urine (IARC 1993; Yoshida et al. 1993). Biomonitoring studies have reported the occurrence of this compound in urine of firefighters and chemical industry workers (Bader et al. 2014).

Recommendation. Category D. Biomarkers for exposure exist, and it was found in urine of the German population (Kutting et al. 2009). However, we recommend it be deferred for additional data from testing in HTP *in vitro* assays for endocrine, developmental, neurotoxic, and obesity effects, and if concerns for toxicity are discovered, then measuring its exposure prevalence in a non-occupationally exposed U.S. population.

Environmental Phenols

Category A:

3,3',5-Trichlorobisphenol A (TrCBA).

Exposure. The detection of TrCBA in human samples varies across studies. In two small French studies it was detected in all of 21 colostrum samples (Migeot et al. 2013) and in 1 of 3 breast milk samples (0.68 ng/mL) (Cariot et al. 2012), but not detected in breast milk (Rodriguez-Gomez et al. 2014) or in urine (Vela-Soria et al. 2014) in two small Spanish studies. In the only U.S. study, TrCBA was not detected in 14 serum samples but was detected in 6 of 31 urine samples (Liao and Kannan 2012). TrCBA was reported in waste and surface water but not in other environmental samples (Andra et al. 2015).

Toxicity. The toxicity of TrCBA was reviewed, and there is evidence that it is an endocrine disruptor (Andra et al. 2015). In vitro binding to mouse PPARα was observed (Hein et al. 2018).

Biomarkers. TrCBA was measured in breast milk and urine. A biomarker of TRCBA was reviewed ((Andra et al. 2015).

Recommendation. Category A. There is concern that TrCBA is an endocrine disruptor. A biomarker exists; however, evidence for exposure is sparse. Thus, we recommend TrCBA be deferred pending additional data on exposure measurements in a non-occupationally exposed U.S. population.

Category B:

2,6-Di-Tert-butylphenol.

Exposure. The presence of 2,6,-di-tert-butylphenol was reported in 3.5% of 85 water samples in the United States (Kolpin et al. 2002). The production volume of 2,6,-di-tert-butylphenol in the United States is 100,000,000-250,000,000 lbs (U.S. EPA 2016b). An EPA predicted median exposure value in the 10^{-7} mg/kg-bw/day range was reported (See Excel Table S2).

Toxicity. In mice, a single inter-perineal dose of butylated hydroxytoluene (2,6-di-tert-butyl-4-methylphenol, BHT) produces a proliferation of alveolar cells and an accompanying increase in lung weight (Mizutani et al. 1982). HTP *in vitro* testing results indicated the potential for thyroid effects, and the compound activated 2 obesity assays (See Excel Table S9). QSAR predictive modeling suggests it may be a developmental toxicant (See Excel Table S10).

Biomarker. Studies were not found that measured 2,6,-di-tert-butylphenol in biological samples.

Recommendation. Category B. There is concern for its toxicity. Based on low-level occurrence in environmental media, a biomarker should be developed and it should be measured in a non-occupationally exposed U.S. population to determine if biomonitoring is warranted in ECHO.

4-Nonylphenol diethoxylate.

Exposure. 4-Nonylphenol diethoxylate was widely detected in house dust samples as well as indoor air (Mitro et al. 2016; Rudel et al. 2003) and water (Kolpin et al. 2002). In a meta-analysis of 3 studies, the pooled geometric mean concentration of a mixture of nonylphenol diethoxylates in indoor dust was 6972 ng/g (95% CI: 3722, 13060), the fourth-highest concentration of 45 chemicals studied (Mitro et al. 2016). The production volume (branched and linear combined) in the United States is 270-370 million pounds (U.S.

EPA 2010b).

Toxicity. Reproductive and developmental toxicity in rats and dogs were demonstrated (ECHC 2001) The action of alkylphenols is mediated by the estrogen receptor, as their effects depended on its presence and were blocked by estrogen antagonists. OP, 4-nonylphenol, and 4-nonylphenoxycarboxylic acid appear to possess intrinsic estrogenic activity; they compete for binding to the estrogen receptor (White et al. 1994). Predictive QSAR modeling suggests it may be a developmental toxicant (See Excel Table S10).

Biomarker. No studies were identified that measured 4-nonylphenol diethoxylate in biological samples.

Recommendation. Category B. There are concerns about its toxicity. Biomarker development is needed to measure it in a non-occupationally exposed U.S. population to determine whether exposure prevalence warrants its inclusion in ECHO.

Dibutylated hydroxytoluene (BHT, butylated hydroxytoluene).

Exposure. BHT is commonly used as an oxidant additive for foods, cosmetics, and petroleum products. BHT is also on the Environmental Working Group's (EWG's) list of food additives to avoid. General Mills announced in 2015 the discontinued use of BHT in breakfast cereals. It is found in plastic materials such as food wrappings and found to migrate into food. BHT was reported in dust in 12 countries (Hasegawa et al. 2005; Liu, R et al. 2017; Wang et al. 2016). BHT was reported at low levels in river streams around the United States (Kolpin et al. 2002). The EPA predicted median exposure value was high at 10^{-5} mg/kg-bw/day range (See Excel Table S2).

Toxicity. Five percent of BHT-treated offspring mice showed increased exploration, decreased sleeping, decreased self-grooming, slower learning, and a decreased orientation reflex. BHT-treated offspring showed decreased sleeping, increased social and isolation-induced aggression, and a severe deficit in learning (Stokes and Scudder 1974). Diet at dose levels of 0 (control), 0.015, 0.045, 0.135, and 0.405% BHT showed little adverse effect on reproductive and neurobehavioral parameters on mice (Toyohito et al. 1993). BHT 500 mg/kg-bw/day given in the diet to F_0-rats resulted in developmental effects (Meyer and Hansen 1980). QSAR modeling results classify BHT as a developmental toxicant (See Excel Table S10).

Biomarker. BHT was measured in plasma and tissue homogenates by HPLC and electrochemical detection; data demonstrate that in humans, at the current level of exposure to dietary antioxidants, significant amounts of BHT are accumulated in the omentum (Bianchi et al. 1997).

Recommendation. Category B. BHT appears to occur in environmental media and exhibits health effects. We recommend measuring BHT in a non-occupationally exposed U.S. population to assess exposure prevalence.

Phenol.

Exposure. Phenol was measured in air (Borhan et al. 2012; Morville et al. 2006; Sturaro et al. 2010), drinking water (ATSDR 2008; Kolpin et al. 2002), and food (ATSDR 2008). It has also been detected in human urine samples in studies measuring phenol as a biomarker of occupational exposure to industrial solvents (e.g., benzene) (Bieniek 1994; D'Andrea and Reddy 2016, 2018; Schettgen et al. 2015). The EPA predicted median exposure was high at the 10^{-4} mg/kg-bw/day range (See Excel Table S2), which ranks 48th amongst all chemicals with predicted exposures.

Toxicity. EPA derived a reference dose (RfD) of 0.3 mg/kg-day based on decreased maternal weight gain observed in a developmental toxicity study in rats (U.S. EPA 2002a). Clinical maternal developmental toxicity was evidenced by dose-related alterations in weight gain and slight developmental toxicity in litters and for malformations (kink tail) for phenol (Narotsky and Kavlock 1995). RIVM derived a tolerable daily intake (TDI) of 4E-2 mg/kg-day based on a NOEL of 40 mg/kg-day. Two studies of women exposed to phenol and other chemicals during pregnancy did not provide evidence of birth defects (ATSDR 2008). QSAR predictive modeling suggests it is a developmental toxicant (See Excel Table S10).

Biomarker. Occupational studies have measured urinary phenol concentrations as a biomarker of exposure to benzene or other industrial solvents (Bieniek 1994; D'Andrea and Reddy 2016, 2018; Schettgen et al. 2015), but has not been demonstrated as a valid biomarker for a non-occupationally exposed population.

Recommendation. Category B. Based on occurrence in environmental media and concerns for its toxicity, a biomarker should be developed and tested in a non-occupationally exposed U.S. population.

4-Nonylphenol monoethoxylate.

Exposure. 4-Nonylphenol monoethoxylate was widely detected in house dust and indoor air (Mitro et al.

2016; Rudel et al. 2003), as well as water (Kolpin et al. 2002). In a meta-analysis of three studies, the pooled geometric mean concentration of a mixture of nonylphenol monoethoxylates in indoor dust was 3517 ng/g (95% CI: 2023, 6111), the eighth-highest concentration of 45 chemicals studied (Mitro et al. 2016).

Toxicity. Reproductive and developmental toxicants in rats and dogs were reported (ECHC 2001). The treatment with 100 µg/L NP (measured 29 µg/L) induced gonadal intersex in >80% of exposed males, mixed secondary sex characteristics in >40% of exposed fish, and suppression of the development of papillae on the anal fin of 100% of males (Balch and Metcalfe 2006). QSAR predictive modeling suggests it may be a developmental and reproductive toxicant (See Excel Table S10).

Biomarker. More research is needed, since studies were not found that measured 4-nonylphenol monoethoxylate in biological samples.

Recommendation. Category B. There is enough concern for toxicity. Development of a biomarker and its measurement in a non-occupationally exposed U.S. population to assess extent of exposure should be undertaken.

Category D:

4-Nonylphenol ethoxycarboxylate.

Exposure. 4-Nonylphenol ethoxycarboxylate was detected in the indoor air of 7% of 30 homes (range: <RL to 18 ng/m^3) and in 93% of house dust samples from 30 homes (range: <RL to 9.45 µg/g) (Rudel et al. 2003).

Toxicity. NOEL levels for reproductive and developmental toxicity in rats and dogs are generally >1000 mg/kg-bw/day (ECHC 2001). The action of alkylphenols is mediated by the estrogen receptor, as their effects depended on its presence and were blocked by estrogen antagonists (White et al. 1994). Given the reported environmental concentrations and bioconcentration factors of APE products, the potential for these compounds to produce estrogenic effects in the environment appears low.

Biomarker. Lacking studies that identified 4-nonylphenol ethoxycarboxylate in biological samples, more research is needed.

Recommendation. Category D. We recommend testing in HTP *in vitro* toxicity assays; testing for endocrine, developmental, neurotoxic, and obesity effects; and if concerns for toxicity are discovered, then development of a biomarker and measurement of exposure in a non-occupationally exposed U.S. population is recommended.

4-Octylphenol diethoxylate.

Exposure. 4-Octylphenol diethoxylate is a detergent commonly found in house dust and is also detected in indoor air (Mitro et al. 2016; Rudel et al. 2003) and water (Kolpin et al. 2002). In a meta-analysis of three studies, the pooled geometric mean concentration in indoor dust was 623 ng/g (95% CI: 330, 1175) (Mitro et al. 2016).

Toxicity. No relevant toxicity data were found. Predictive QSAR modeling suggests that this compound is a developmental and reproductive toxicant (See Excel Table S10).

Biomarker. No biomarker of exposure was found.

Recommendation. Category D. This compound is readily found in house dust and warrants further research. Testing in HTP *in vitro* assays for endocrine, developmental, neurotoxic, and obesity effects should be performed. If concerns for toxicity are discovered, then a biomarker should be developed, and it should be measured in a non-occupationally exposed U.S. population to establish exposure prevalence.

4-Octylphenol monoethoxylate.

Exposure. 4-Octylphenol monoethoxylate is a detergent found in house dust and indoor air (Mitro et al. 2016; Rudel et al. 2003) as well as water (Kolpin et al. 2002). It was detected in the indoor air of 93% of 120 homes (range: <RL to 50 ng/m^3) (Rudel et al. 2003). In a meta-analysis of results from three studies, the pooled geometric mean concentration of 4-Octylphenol monoethoxylate in indoor dust was 552 ng/g (95% CI: 367, 832) (Mitro et al. 2016).

Toxicity. No relevant toxicity data were found. Predictive QSAR modeling suggests that this chemical is a developmental toxicant (See Excel Table S10).

Biomarker. No biomarker was found in the literature.

Recommendation. Category D. The widespread occurrence in house dust warrants conducting HTP *in vitro* assays for endocrine, developmental, neurotoxic, and obesity effects. If concerns for toxicity are discovered, then a biomarker should be developed and its exposure prevalence in a non-occupationally exposed U.S. population should be determined.

Organophosphorus-based Flame Retardants

Category B:

Triethyl phosphate (TEP).

Exposure. TEP is ubiquitous in indoor dust, occurring at a high-detection frequency and hundreds to thousands of ng/g (Ali et al. 2012; Ouyang et al. 2017). In one study, TEP was reported in >90% of 150 water samples (Kim and Kannan 2018). Its occurrence in these media suggest continuous exposure via the inhalation, dermal contact, and ingestion routes. Surprisingly, no reports were found for its measurement in human biofluids. A production volume of 1 to 10 X 10^6 pounds per year was reported (U.S. EPA 2016b). EPA predicted exposure was in the 10^{-7} mg/kg-bw/day range (See Excel Table S2).

Toxicity. Associations between TEP and changes in human sphingolipid homeostasis was reported in humans (Zhao et al. 2016) and reproductive effects in rats (Gumbmann et al. 1968). QSAR modeling results suggest TEP is a developmental toxicant (See Excel Table S10).

Biomarker. Measurement of TEP in urine to assess exposure may be confounded by its formation from organophosphate pesticides.

Recommendation. Category B. A biomarker needs to be developed, and the extent of exposure needs to be determined in a non-occupationally exposed U.S. population. If the biomarker is nonspecific for TEP, then research is needed to determine whether dust measurements correlate with the nonspecific biomarker.

Tris(2,3-dichloropropyl) phosphate (TDCnPP).

Exposure. TDCnPP was measured in house dust ranging from 20-16,560 ng/g (Ali et al. 2012) and in homes, apartments, primary schools, and offices at a 100% detection frequency ranging from 109 to 9220 ng/g (Cristale et al. 2018). TDCnPP was measured in river water at 23-136 ng/L (Ishikawa et al. 1985). It was identified in human seminal fluid (Hudec et al. 1981).

Toxicity. TDCnPP was implicated as a neurotoxicant (AbouDonia 2016) and acts as an immune-suppressant in mice at doses of 25 mg/kg/day (Luster et al. 1981). QSAR predictive modeling results suggest it may be a developmental and reproductive toxicant (See Excel Table S10).

Biomarker. No biomarkers of exposure have been reported for TDCnPP in blood or urine; however, it was measured in seminal fluid (Hudec et al. 1981).

Recommendation. Category B. Testing with HTP *in vitro* assays for endocrine, developmental, neurotoxic, and obesity effects is recommended, and if concerns for toxicity are discovered, then, based on its high levels of occurrence in environmental media, a biomarker should be developed and its exposure prevalence in a non-occupationally exposed U.S. population established.

Category D:

Diquanidine hydrogen phosphate (DHP).

There were no studies reporting exposure, environmental levels, toxicity, or biomarkers for DHP.

Recommendation. Category D. We recommend that DHP be tested in HTP *in vitro* assays for endocrine, developmental, neurotoxic, and obesity effects. Pending results that indicate concerns for its toxicity, a biomarker should be developed and it should be measured in a non-occupationally exposed U.S. population to assess exposure prevalence.

Tris(2-chloro-iso-propyl) phosphate (TCIPP).

Exposure. TCIPP is widely found, but at low levels, in air (Shoeib et al. 2014). It is prevalent in house dust, with reported levels of several thousand ng/g (Araki et al. 2014; Larsson and Berglund 2018; Mitro et al. 2016).

Toxicity. Atopic dermatitis in humans was reported (Araki et al. 2014); however, we have not found endpoints of toxicity/health effects in *in vivo* animal studies relevant to ECHO. One calcium-ion channel assay was activated in HTP *in vitro* testing (See Excel Table S9).

Biomarker. No information on a biomarker for TCIPP was found; however, since it is closely related to tris(1-chloro-iso-propyl)phosphate, for which a biomarker exists, it is expected that one can be developed. Metabolites of tris(1-chloro-2-propyl)phosphate have been isolated, quantitated, and identified in the urine and feces of rats. Identifiable metabolites accounted for 75-78.5% of urinary and fecal radiocarbon at both doses in both sexes, as [bis(l-chloro-2-propyl)]-0-(2-proprionic acid) phosphate was identified as a major metabolite and accounted for >50% of the dose in the urine and feces of both sexes (PubChem 2018i).

Recommendation. (Category D). There is insufficient toxicity data for TCIPP. There is evidence for TCIPP in the environment; however, if concerns for toxicity are discovered, then exposure data are needed once a biomarker is established.

Tris-(tribromoneopentyl) phosphate (TTBNPP).

Exposure. TTBNPP was not detected in fish samples in one study (Santin et al. 2016), but was quantified in river water in Sweden (Gustavsson et al. 2018). There is insufficient TTBNPP data to establish whether exposure occurs.

Toxicity. No reports of health effects or toxicity studies were found during the literature search. QSAR predictive modeling results suggest it may be a reproductive toxicant (See Excel Table S10).

Biomarker. No biomarker of exposure was found.

Recommendation. Category D. Testing with HTP *in vitro* assays for endocrine, developmental, neurotoxic, and obesity effects is recommended, and if concerns for toxicity are discovered, then a biomarker needs to be developed for estimating exposure in a non-occupationally exposed U.S. population.

Perfluoroalkyl Substances

Category A:

Perfluorooctadecanoic acid (PFODA).

Exposure. Biomonitoring data on PFODA are lacking. Although measured in river samples from Spain (Lorenzo et al. 2016), in Canadian tap water (DT, 12%; LOD to 2.9 ng/L)(Kabore et al. 2018), and in foodstuff

packaging materials in the Greek market (Zafeiraki et al. 2015), it was not detected or not reported due to low detection. Low levels in house dust were reported (Larsson and Berglund 2018).

Toxicity. Reproductive and developmental toxicity was reported in one study using rats (Hirata-Koizumi et al. 2012).

Biomarker. It is included in the Biomonitoring California Designated Chemicals list (Biomonitoring California 2015). A method for quantification in human serum was developed recently (Gao et al. 2016), but no application studies have been found.

Recommendation. Category A. Biomonitoring is deferred pending additional data on evidence for exposure. PFODA measurement in a non-occupationally exposed U.S. population is recommended to establish exposure prevalence.

Category D:

Perfluoroheptane sulfonic acid (PFHpS).

Exposure. PFHpS was detected in blood samples from pregnant women in Denmark (detection frequency: 76%; serum median: 0.14 ng/mL) (Bjerregaard-Olesen et al. 2016), Norway (detection frequency: 88%; plasma median: 0.13 ng/mL) (Starling et al. 2014), and Australia (detection frequency: 100%; mean: ranging from 0.15-0.40 ng/mL across all age groups) (Eriksson et al. 2017). It was also detected in the blood of 3-year-old children (detection frequency: 89%; median: 0.12 ng/mL) (Papadopoulou et al. 2016) and adolescent serum (detection frequency: 98%; median: 0.15 ng/mL) in Norway (Averina et al. 2018). It was measured but not detected in hair in a Belgium study (Alves et al. 2015). Using suspect screening, PFHpS was tentatively identified among firefighters and controls (Rotander et al. 2015) and quantified in tap water samples from the Ivory Coast (Kabore et al. 2018). A mega-analysis of house dust reported low levels (Larsson and Berglund 2018).

Toxicity. No *in vivo* or *in vitro* toxicity studies were found. Predictive modeling suggests that PFHpS is an endocrine disruptor (See Excel Table S11).

Biomarker. It is included in the Biomonitoring California Designated Chemicals list (Biomonitoring California 2015). A potential biomarker was available in plasma, serum, and hair (Alves et al. 2015; Averina et al. 2018; Eriksson et al. 2017; Papadopoulou et al. 2016; Starling et al. 2014).

Recommendation. Category D. Even though there is evidence of exposure and considering the availability of a biomarker, it is deferred pending additional data on toxicity. Because PFCs have very closely related chemical properties, PFHpS is also likely an endocrine toxicant, an assumption supported by predictive modeling.

Perfluorohexadecanoic acid (PFHxDA).

Exposure. Although measured in river samples from Spain (Lorenzo et al. 2016), PFHxDA was not detected or not reported due to low detection. It was detected in Greek fast food boxes and in ice cream cup samples (Zafeiraki E 2014). The median of reported medians for PFHxDA in house dust was 1 ng/g (Larsson and Berglund 2018).

Toxicity. No information was found on toxicity.

Biomarker. A method for quantification in human serum was developed (Gao et al. 2016), but no application studies were found.

Recommendation. Category D. *In vivo* animal toxicity studies are recommended. PFHxDA measurement in a non-occupationally exposed U.S. population to establish exposure prevalence is recommended.

Perfluoropentane sulfonic acid (PFPeS).

Exposure. PFPeS was measured but not detected in hair in a Belgium study (Alves et al. 2015). One study compared PFAS exposure among firefighters versus controls using liquid chromatography quadrupole time-of-flight tandem mass spectrometry (LC-QTOF/MS) and tentatively identified PFPeS being present in both firefighters and controls (Rotander et al. 2015). It was also quantified in tap water samples from Canada and the Ivory Coast (ranged from LOD to 3.9 ng/L) (Kabore et al. 2018).

Toxicity. Toxicity information from *in vivo* and *in vitro* studies were lacking, but predictive modeling suggests that PFPeS is an endocrine disruptor (See Excel Table S11) and a reproductive toxicant (See Excel Table S10).

Biomarker. A potential biomarker was available in hair, but PFPeS was not detected in hair samples in the study (Alves et al. 2015).

Recommendation. Category D. Biomonitoring is deferred pending additional toxicity studies on endocrine disruption and reproductive effects. If concerns for toxicity are discovered, then, since a biomarker exists, the extent of exposure in a non-occupationally exposed U.S. population should be determined.

Pesticides

Category A:

Difenoconazole.

Exposure. Difenoconazole, a fungicide, was found in surface water (Wightwick et al. 2012) and food (Gartrell et al. 1986; Lozowicka 2015; USDA 2017). It was detected in 20.9% of 177 samples of tomatoes collected in 2014, but at a low mean of 9 pbb (USDA 2017). EPA predicted exposure was in the 10^{-6} mg/kg-bw/day range (See Excel Table S2).

Toxicity. The EPA reported developmental toxicity in rats, while the WHO concluded that there were no adverse effects because the doses required were high (Vogel 1999), (WHO 2007b). A neurotoxicity reproductive toxicity study in rats showed no adverse effects (EFSA 2011a; WHO 2007b). HTP *in vitro* testing indicated activity in 2 endocrine, 1 neurotoxicity, and 4 obesity assays (See Excel Table S9).

Biomarker. This fungicide was measured in serum of a Chinese population (Chang et al. 2017).

Recommendation: Based on HTP *in vitro* results, environmental levels, and the existence of a biomarker, it is recommended that difenoconazole be measured in the U.S. population.

Metribuzin.

Exposure. Metribuzin is an herbicide and was detected in water. In samples collected in 2011 throughout the United States, metribuzin was detected in 17% of 120 samples, with a range of 37.5 to 29,742 ng/L (USDA 2017). No other information on its occurrence in other environmental and exposure media were found. EPA predicted exposure was in the 10^{-9} mg/kg-bw/day range (See Excel Table S2).

Toxicity. The EPA determined metribuzin was a developmental toxicant in the rat and rabbit (U.S. EPA 2003b). Reproductive studies show decreased body weight and exaggerated liver cell growth in parents and pups. Endocrine effects were observed in rats (increased thyroid weight) and decreased T4 in rabbits (decreased T3) (EFSA 2006a; U.S. EPA 2003b).

Biomarker. The principal urinary metabolites of metribuzin in mice and rats were mercapturic acids, which arise via metribuzin sulfoxide or deaminometribuzin sulfoxide reacting with GSH (Bleeke et al. 1985). Biomonitoring of metribuzin in human serum was conducted (Chang et al. 2017).

Recommendation: There is enough toxicological concern. There is not strong evidence for exposure. Given that there is a biomarker, some U.S. biospecimens should be screened to determine if there is exposure.

Pyraclostrobin.

Exposure. Pyraclostrobin, a fungicide, was detected in <2% of hair samples of 18 farm workers (Schummer et al. 2012). It was detected in 45.7% of 708 samples of grapes collected nationally, with a mean of 47 ng/g (USDA 2017). No other published data for environmental and exposure media were found. EPA predicted exposure was in the 10^{-8} mg/kg-bw/day range (See Excel Table S2).

Toxicity. In rat and rabbit studies, pyraclostrobin was a developmental toxicant (IPCS-INCHEM 2003). MDH concluded that there were no adverse reproductive effects in rats and rabbits (MDH 2016a). No information from *in vivo* studies was found on potential endocrine disruption effects. It has shown positive neurotoxicity effects in an *in vitro* assay (Regueiro et al. 2015). There likely is an overlap between *in vitro* assay activity and predicted exposures (Wetmore et al. 2012), specifically in 1 assay associated with neurotoxicity. HTP *in vitro* testing for neurotoxicity and obesity indicated activity (See Excel Table S9).

Biomarker. A biomarker in hair was reported (Schummer et al. 2012).

Recommendation: There is potential for exposure from food at low levels and enough concern for toxicity. Given there is a biomarker, pyraclostrobin should be measured in a U.S. population to determine exposure.

Tetraconazole.

Exposure. Tetraconazole was measured at a low frequency in food commodities (USDA 2017). For example, it was detected in 10.5% of 706 samples of strawberries collected nationwide, with a mean of 30 ng/g (USDA 2017). EPA predicted exposure was in the 10^{-7} mg/kg-bw/day range (See Excel Table S2).

Toxicity. Canada Health has reviewed the toxicity of tetraconazole and concluded there are no adverse effects on reproduction based on *in vivo* studies (Canada 2012). There likely is an overlap between *in vitro* assay activity and predicted exposures (Wetmore et al. 2012), specifically, in 1 assay, a suspected liver

carcinogen. This pesticide exhibited activity in 1 endocrine, 4 obesity, and 3 neurotoxicity HTP *in vitro* assays (See Excel Table S9). Predictive modeling suggests that it may be a developmental toxicant (See Excel Table S10) and binds to the androgen receptor (See Excel Table S11).

Biomarker. Three metabolites, 5-hydroxy-TBZ, 4-hydroxy-TBZ, and 2-acetylbenzimidazzole, were identified in mice (Fustinoni et al. 2014). Even though a method exists for measuring the parent compound in urine (Fernandez et al. 2001), it has not been validated as a biomarker. The parent compound was sought in human serum in biomonitoring, but it was not detected (Chang et al. 2017).

Recommendation: There is enough toxicity to be concerned. A biomarker exists, but it is unclear if there is likely to be any exposure. Testing in a biofluid should be done to establish exposure. Thus, biomonitoring is deferred pending additional data.

Triclopyr.

Exposure. Measurement of triclopyr was done in water, but data for other media were not found. In 232 samples collected throughout the United States in 2012, it was detected at a 39% frequency with a range of 2.7 to 130 ng/L (USDA 2017). EPA predicted exposure was in the 10^{-8} mg/kg-bw/day range (See Excel Table S2).

Toxicity. The EPA reported triclopyr was a developmental toxicant in rats (U.S. EPA 1998d). Triclopyr was active in 4 obesity HTP *in vitro* assays (See Excel Table S9). Predictive modeling results suggest that triclopyr is a developmental and reproductive toxicant (See Excel Table S10).

Biomarker. Triclopyr was measured in serum of a Chinese population (Chang et al. 2017). Following oral administration, blood levels peaked at 2-3 hours and declined to undetectable levels within 48 hours; more than 80% of the dose was found as unchanged triclopyr in human urine (Carmichael et al. 1989); four minor urinary metabolites have been noted (Timchalk et al. 1990). Kinetics of triclopyr have been reported for occupationally exposed subjects (Gosselin et al. 2005).

Recommendation: There is enough toxicity to be concerned. A biomarker exists, but it is unclear if there is likely to be any exposure. Testing in a biofluid should be done to establish exposure. Thus, biomonitoring is deferred pending additional data.

Category B:

Boscalid.

Exposure. Boscalid, a fungicide, was detected in groundwater, surface water, and food (Reilly et al. 2012; USDA 2017; USGS 2016). For example, in 2014 it was detected in 38% of the 688 samples of blueberries, with a mean of 186 ng/g (USDA 2017). It was detected on several other fruits and vegetables during the reporting period from 2010 to 2016 (USDA 2017). In groundwater it was detected at a 58% frequency with a median of 16 ng/L, and in surface water at a 75% frequency with a median of 22.6 ng/L (Reilly et al. 2012). Exposure was detected using a passive wrist sampler worn by farmers (Aerts et al. 2018). EPA predicted exposure was in the 10^{-8} mg/kg-bw/day range (See Excel Table S2).

Toxicity. Liver and thyroid effects were exhibited in rats and dogs (U.S. EPA 1993, 2003a). It has shown positive neurotoxicity effects in an *in vitro* assay (Regueiro et al. 2015). Boscalid was active in 1 neurotoxicity and 4 obesity HTP *in vitro* assays (See Excel Table S9). Also, predictive modeling suggested that boscalid was a developmental toxicant (See Excel Table S10) and binds to the α-estrogen receptor, although HTP *in vitro* testing did not indicate estrogen activity (See Excel Table S9).

Biomarker. The most important metabolites are hydroxyl or O-glucuronide metabolites and S-glucuronide conjugation products (PubChem 2018e). Boscalid is effectively metabolized and efficiently excreted; the half-life is less than 24 hours (PubChem 2018e). No publications on biomarkers were found.

Recommendation: Even though there are enough toxicity data to be concerned, there is no reported biomarker: therefore, it is deferred pending the development of a biomarker.

Carbendazim (MBC).

Exposure. Carbendazim, a fungicide, appears in surface water, food, and house dust (Eke 1996; Palma et al. 2004; Readman et al. 1997; Salis et al. 2017; USDA 2017; Yess et al. 1991). In house dust samples, it was detected in 26% of 206 samples with a mean of 0.08 ug/g (Salis et al. 2017). It has been detected in 30.7% of 176 strawberry samples collected in 2014 with a mean of 69 ng/g (FDA 2004-2005a, 2011). MBC was detected on many other fruits and vegetables (USDA 2017). EPA predicted exposure was in the 10^{-9} mg/kg-bw/day range (See Excel Table S2).

Toxicity. Developmental and teratogenic effects for carbendazim was found in rats and rabbits (EFSA 2010b; Singh et al. 2016; U.S. EPA 2005). Reproductive studies in rats have shown male infertility. Predictive modeling also suggests that MBC is a reproductive toxicant. Estrogenic and antiestrogenic activity has been also suggested for MBC (Okubo et al. 2004).
Biomarker. No reports of biomarkers for carbendazim were found.
Recommendation: There is enough toxicity to be concerned. Based on the absence of a reported biomarker, biomonitoring is deferred pending its development.

Dimethomorph.
Exposure. A recent study in France found only one detect in ambient air for dimethomorph, a fungicide (Coscolla et al. 2011). However, 27.7% of 708 samples of spinach had detectable levels of dimethomorph and a mean of 351 ng/g (USDA 2017). Also, it was detected with a passive wrist sampler (Aerts et al. 2018) and in indoor dust near schools in Africa (Dalvie et al. 2014). EPA predicted exposure was in the 10^{-8} mg/kg-bw/day range (See Excel Table S2).
Toxicity. Developmental toxicity was reported in a dog study (WHO 2007a). No reproductive or neurotoxicity was noted (WHO 2007a). Dimethomorph exhibited androgen receptor antagonism in an *in vitro* assay (Kleinstreuer et al. 2017; Orton et al. 2011). It showed positive neurotoxicity effects in an *in vitro* assay (Regueiro et al. 2015). HTP *in vitro* results were positive in steroidogenic, 2 endocrine, and 2 neurotoxicity assays (See Excel Table S9). Predictive modeling also suggested that it may be a developmental toxicant (See Excel Table S10) and binds to the β-estrogen receptor (See Excel Table S11).
Biomarker. No papers were found that discussed a biomarker for dimethomorph.
Recommendation: There is enough evidence for likely exposure from food and toxicity; however, based on the absence of a reported biomarker, biomonitoring is deferred pending its development.

Diphenylamine.
Exposure. Diphenylamine, a fungicide, was reported in food commodities (FDA 2004-2005a, 2011; Ripley et al. 2000; Roy et al. 1997; USDA 2017) and found to leach from nursing bottle nipples (Babish JG 1983). In 708 samples of apples collected in 2015, it was detected at a frequency of 82% and a mean of 332 ng/g, as well as in other fruits and vegetables (USDA 2017). It was detected at low levels in ambient air (Coscolla et al. 2011). In a non-targeted analysis, it was identified in dryer lint (Ouyang et al. 2017). EPA predicted exposure was in the 10^{-6} mg/kg-bw/day range (See Excel Table S2).
Toxicity. Developmental toxicity was shown in dogs (U.S. EPA 1987b). Reproductive toxicity in rats was reported (U.S. EPA 1987b). No special sensitivity in infants and children was found (U.S. EPA 1998e). Diphenylamine was active in 4 obesity and 1 neurotoxicity HTP *in vitro* assays (See Excel Table S9). Predictive modeling results suggested diphenylamine is a developmental toxicant (See Excel Table S10).
Biomarker. The major metabolites reported in the rat, rabbit, and man urine were 4-hydroxydiphenylamine and 4,4'-dihydroxydiphenylamine (Alexander et al. 1965), suggesting the availability of biomarkers for exposure.
Recommendation: Even though diphenylamine is prevalent in food, and it exhibits developmental toxicity, it needs a method for measuring its metabolite as a biomarker before biomonitoring is recommended.

Fenbuconazole.
Exposure. Fenbuconazole, a fungicide, was measured in food commodities (USDA 2017). It was detected in 38.9% of 453 samples of cherries collected in 2015 with a mean of 202 ng/g. It was also found in other fruits and vegetables (USDA 2017). EPA predicted exposure was in the 10^{-7} mg/kg-bw/day range (See Excel Table S2).
Toxicity. Fenbuconazole exhibited reproductive and maternal and developmental toxicity in rats and rabbits (Anonymous 1998). There likely was an overlap between *in vitro* assay activity and predicted exposures (Wetmore et al. 2012), specifically with 3 assays associated with thyroid activity. HTP *in vitro* testing found neurotoxic and androgen activity (See Excel Table S9). Predictive modeling suggested that it exhibited reproductive toxicity (See Excel Table S10).
Biomarker. No papers indicating possible biomarkers for fenbuconazole were found.
Recommendation: Even though there is enough concern for toxicity and likely exposure to fenbuconazole, biomonitoring is deferred pending the development of a biomarker.

Fludioxonil.

Exposure. Fludioxonil, a fungicide, was measured in food commodities (FDA 2004-2005a, 2011; USDA 2017). In 578 samples of nectarines collected in 2015, it was detected at a frequency of 71.8% with a mean of 597 ng/g. It was also measured in other fruits and vegetables (USDA 2017). It has been quantified in ambient air, with a mean of 19.29 pg/m^3 (Coscolla et al. 2011). It has been detected with a passive wrist sampler worn by farmers (Aerts et al. 2018). EPA predicted exposure was in the 10^{-9} mg/kg-bw/day range (See Excel Table S2).

Toxicity. Reproductive and developmental toxicity was reported in rats (U.S. EPA 2011; WHO 2007a). *In vitro* endocrine study results suggest that it is an antiestrogenic chemical (Teng et al. 2013), although it was not found to have estrogenic activity in the integrated model (Judson et al. 2015). *In vitro* results suggested it was an ERα and AhR agonists (Medjakovic et al. 2014). There likely was an overlap between *in vitro* assay activity and predicted exposures (Wetmore et al. 2012). Predictive modeling suggested that fludioxonil was a developmental toxicant (See Excel Table S10) and binds to the androgen receptor (See Excel Table S11), although *in vitro* testing did not find androgen activity (Kleinstreuer et al. 2017). HTP *in vitro* testing found neurotoxic and obesity activity (See Excel Table S9).

Biomarker. No papers were found regarding a biomarker for fludioxonil.

Recommendation: The likelihood of exposure exists for fludioxonil from food ingestion, and there is evidence for toxicity; however, biomonitoring is deferred pending the development of a biomarker for exposure.

Thiabendazole (TBZ).

Exposure. Thiabendazole was measured in food commodities (FDA 2004-2005a, 2011; USDA 2017); however, data for other environmental and exposure media were not found. For example, of 708 samples of apples collected in 2015, 67.7% had detectable levels with a mean of 444 ng/g (USDA 2017). Thiabendazole was detected on a passive wrist sampler worn by farm workers (Aerts et al. 2018). EPA predicted exposure was in the 10^{-8} mg/kg-bw/day range (See Excel Table S2).

Toxicity. Reproductive effects were reported in rats and dogs (Cochran 2001; IPCS-INCHEM 1997). In other studies, developmental studies in rats, rabbits, and mice showed a variety of developmental effects (Cochran 2001; IPCS-INCHEM 1997). TBZ was active in 5 HTP *in vitro* obesity assays (See Excel Table S9). Predictive modeling results suggested thiabendazole was a developmental and reproductive toxicant (See Excel Table S10).

Biomarker. Thiabendazole is mainly metabolized to 5-hydroxy-TBZ (5-OH-TBZ) and its conjugates in domestic and laboratory animals. Besides the known metabolites of TBZ, 4-hydroxy-TBZ and 2-acetylbenzimidazole (ABI) were identified as new metabolites of TBZ in the urine of rats (Fujitani et al. 1991). Thiabendazole and its metabolic products were excreted rapidly by the kidney, with 92% of the radioactivity found in the urine (87%) and feces (5%) within 48 hours (Tocco et al. 1966). The bulk of radioactivity appeared in the urine in 24 hours; approximately one-half of the radioactivity in the human urine was associated with compounds which were identified chemically. These compounds were primarily the glucuronide and sulfate esters of 5-hydroxythiabendazole (Tocco et al. 1966). The metabolite, 5-OH-TBZ, was chosen as a biomarker in human urine for quantifying exposure to TBZ (Ekman et al. 2014).

Recommendation: There is likely exposure from food and toxicological concerns for thiabendazole. Thus, a biomarker should be developed.

Triflumizole.

Exposure. Triflumizole, a fungicide, was measured in food commodities, but data for other media were not found (USDA 2017). In 232 samples of cherries collected nationally in 2015, it was detected at a 40% frequency with a mean of 48 ng/g (USDA 2017). EPA predicted exposure was in the 10^{-7} mg/kg-bw/day range (See Excel Table S2).

Toxicity. Reproductive toxicity was reported in rats (WHO 2013). Developmental effects in rats and rabbits, neurotoxicity in rats, and immunotoxicity in mice were reported (WHO 2013). In the HTP *in vitro* tests, triflumizole was active in 10 assays (See Excel Table S9). Predictive modeling suggested that it was a developmental and reproductive toxicant (See Excel Table S10) and binds to the androgen receptor (See Excel Table S11).

Biomarker. No biomarker information was found for triflumizole.

Recommendation: Exposure is likely to triflumizole and there is concern for its toxicity. Biomonitoring is

deferred pending the development of a biomarker.

Category C:

Acetochlor ethane sulfonic acid (ESA).

Exposure. Acetochlor ESA, a microbial fungicide metabolite of Acetochlor, was detected in groundwater, finished, and unfinished drinking water (USDA 2017). For samples collected in 2012, it was measured in 232 finished drinking water samples with a 71% detection frequency and range of 2.7-280 ng/L (USDA 2017). Also, it was detected in samples collected by the USDA in 2011 and 2013 (USDA 2017). In a study conducted in Iowa, it was detected in well samples at a 10% frequency (Kolpin et al. 1998). We were unable to find monitoring data for other environmental media or in human biofluids.

Toxicity. Developmental toxicity was reported based on acetochlor effects on body weight (Gadagbui et al. 2010). Low reproductive toxicity was observed (OEHHA 2012). Acetochlor ESA was a possible endocrine disruptor based on acetochlor (Rollerova et al. 2000; Rollerova and Urbancikova 2000).

Biomarker. We were unable to find published biomarker information for acetochlor ESA.

Recommendation: This pesticide shows enough toxicity to be concerned. Based on the sparse information on exposure to acetochlor ESA and the absence of a reported biomarker, it is deferred pending further data.

Acifluorfen.

Exposure. Acifluorfen, a pre- and post-emergence herbicide, was monitored in ambient air, dry deposition, and water samples in 3 studies in the United States; however, only 2 samples had detectable quantities out of >1000 samples (Hochstedler 2000; Holman et al. 2000; Kresse et al. 1998). EPA predicted exposure is in the 10^{-7} mg/kg-bw/day range (See Excel Table S2).

Toxicity. Reproductive and developmental toxicity was reported in rats (U.S. EPA 2015). Predictive modeling suggested that acifluorfen was a developmental toxicant (See Excel Table S10) and binds to the androgen receptor (See Excel Table S11). A paper by Wetmore indicates that there likely was an overlap between *in vitro* assay activity and predicted exposures (Wetmore et al. 2012), specifically in 1 assay associated with *in vivo* liver and kidney effects. Acifluorfen was active in 5 HTP *in vitro* obesity assays (See Excel Table S9).

Biomarker. No biomarker for acifluorfen was found.

Recommendation: There is enough toxicity information to be concerned from exposure to acifluorfen. However, based on the sparse information on exposure to acifluorfen and the absence of a reported biomarker, acifluorfen is deferred pending developing a biomarker and determining in a U.S. population the prevalence of exposure.

Alachlor ethane sulfonic acid.

Exposure. Alachlor ESA, herbicide metabolite, was detected in groundwater, finished, and unfinished drinking water (USDA 2017). As an example, data from 2010 indicates that alachlor ESA measured in 232 drinking water samples was detected at a frequency of 42% with a range of 2.9-91 ng/L (USDA 2017). Also, Kolpin reported that alachlor ESA was detected in 50% of well samples in Iowa (Kolpin et al. 1998). We did not find papers on its monitoring in other environmental media or human biofluids.

Toxicity. Alachlor ESA hematological systemic and developmental toxicity was reported (Gadagbui et al. 2010; MDH 2016b; Wickerham et al. 2012). Only 2 HTP *in vitro* tests have been conducted on this compound, but one exhibited thyroid activity (See Excel Table S9).

Biomarker. No information on a biomarker was found.

Recommendation: Additional HTP *in vitro* assays should be run. Based on the sparse information on exposure to acetochlor ESA and the absence of a reported biomarker, it is deferred pending further data.

Alachlor oxanilic acid (OA).

Exposure. Alachlor OA, an herbicide metabolite, was detected in groundwater, finished, and unfinished drinking water (USDA 2017). For samples collected in 2012, it was measured in 284 drinking water samples with a 34% detection frequency and a range of 1-28 ng/g (USDA 2017). It was also detected in groundwater and drinking water samples collected by the USDA in 2011 and 2013 (USDA 2017). Ten percent of well samples in Iowa had measurable levels (Kolpin et al. 1998). We found no data for other environmental media or for human biofluids.

Toxicity. Acetochlor effects on the hematological system was reported (Gadagbui et al. 2010; MDH 2016b).

Biomarker. No papers on biomarkers for alachlor OA were found.

Recommendation: There is evidence of developmental toxicity. HTP *in vitro* assays should be conducted to further assess toxicity. Based on the sparse information on exposure to acetochlor ESA and the absence of a reported biomarker, it is deferred pending further data.

Piperonyl butoxide (PBO).

Exposure. Piperonyl butoxide, an insecticide, was measured in personal air from 511 pregnant women samples (Williams et al. 2008). A detection frequency of 75% and a median of 0.42 ng/m^3 were reported (Williams et al. 2008). It was measured in food commodities (USDA 2017). For example, it was detected in 6.9% of 706 strawberry samples collected in 2015, with a mean of 176 ng/g (USDA 2017). PBO was reported in indoor air and dust (Rudel et al. 2003). It was also detected in <10% of passive wrist samplers worn by farm workers (Aerts et al. 2018). EPA predicted exposure was in the 10^{-8} mg/kg-bw/day range (See Excel Table S2).

Toxicity. Epidemiological data from children show respiratory effects and a susceptibility of asthmatics (Liu et al. 2012). An inhalation study in rats showed nasal discharge and histopathology in the larynx. Prenatal exposure to PBO exhibited neurodevelopment effects (Horton et al. 2011). Reproductive and neurotoxicity has been reported in mice (Tanaka and Inomata 2016). A mouse developmental study in rat and rabbits showed fetal effects at the higher doses that were maternally toxic (EU 2017). There likely is an overlap between *in vitro* assay activity and predicted exposures (Wetmore et al. 2012), specifically in 1 assay associated with *in vivo* hematologic and liver effects. PBO was active in 1 thyroid and 4 obesity *HTP in vitro* assays (See Excel Table S9).

Biomarker. The metabolism of PBO is complex, with >25 radioactive components observed after administration of radioactive PBO to rats; 12 urine metabolites were assigned structures, and 4 additional structures were observed in feces (Byard and Needham 2006).

Recommendation: Based on the occurrence of PBO in air, concerns for its respiratory effects and neurotoxicity, and the lack of a biomarker, additional data are needed to establish exposure. If a biomarker can be developed, then PBO should be measured in a non-occupationally exposed U.S. population. If a biomarker cannot be developed, then a proxy exposure approach is recommended.

Quinclorac.

Exposure. Information on occurrence of quinclorac, an herbicide, in environmental and exposure media was sparse. It was measured in food analyzed as part of the U.S. FDA Total Diet Study but at very low levels (FDA 2004-2005a, 2011). EPA predicted exposure was in the 10^{-8} mg/kg-bw/day range (See Excel Table S2).

Toxicity. Developmental toxicity was exhibited in the dog (APVMA (Australian Pesticides and Veterinary Medicines Authority) 2005). A 2-year generation rat study showed parental and neonatal toxicity (APVMA (Australian Pesticides and Veterinary Medicines Authority) 2005). None of the long-term studies reported neurotoxicity effects, and endocrine effects were not addressed in the summaries (APVMA (Australian Pesticides and Veterinary Medicines Authority) 2005). There likely was an overlap between *in vitro* assay activity and predicted exposures (Wetmore et al. 2012), specifically in 1 assay associated with developmental effects. Predictive modeling suggested it was a developmental toxicant (See Excel Table S10).

Biomarker. Papers on biomarkers of quinclorac were not found.

Recommendation: There is enough toxicological concern; however, occurrence of exposure is unclear. Measures in environmental or biospecimens need to be conducted to determine its prevalence. A biomarker needs to be developed. Thus, quinclorac biomonitoring is deferred pending further data.

Spiroxamine.

Exposure. We did not find environmental and exposure data for spiroxamine, a fungicide. In a non-targeted study, it was detected in dryer lint (Ouyang et al. 2017). EPA predicted exposure was in the 10^{-7} mg/kg-bw/day range (See Excel Table S2).

Toxicity. Adrenal toxicity was found in the rat (EFSA 2010b). No reproductive effects were observed from a 2-generation rat study. Developmental study in rabbits showed maternal and fetal effects (EFSA 2010b). Traditional *in vivo* studies did not show evidence of neurotoxicity. There likely was an overlap between *in vitro* assay activity and predicted exposures (Wetmore et al. 2012), specifically in 1 assay associated with *in vivo* neurotoxicity and liver effects. Spiroxamine was active in 5 obesity and 4 neurotoxicity HTP *in vitro* assays (See Excel Table S9).

Biomarker. Papers establishing biomarkers of spiroxamine were not found.
Recommendation: There is enough toxicological concern; however, occurrence of exposure is unclear. Measures in environmental or biofluids need to be conducted to determine its prevalence. A biomarker needs to be developed. Thus, spiroxamine biomonitoring is deferred pending further data.

Category D:

Fenamidone.

Exposure. Fenamidone, a foliar fungicide, was detected in food commodities (USDA 2017), but no reports were found for its monitoring in other environmental or exposure media. In 708 samples of spinach collected in 2015 it was detected at a frequency of 30.8% with a mean of 799 ng/g (USDA 2017). EPA predicted exposure was in the 10^{-8} mg/kg-bw/day range (See Excel Table S2).
Toxicity. A summary of data, largely from proprietary studies, noted no adverse effects in chronic toxicity studies in rat and dogs, reproductive studies in rats, and teratology studies in rats and rabbits.
No neurotoxicity studies were noted, although effects were not observed in traditional studies (Canada 2014). Although HTP in vitro testing for androgen did not show activity (Kleinstreuer et al. 2017), HTP *in vitro* assay results indicate neurotoxic and thyroid activity (See Excel Table S9). Predictive modeling suggested fenamidone was an endocrine disruptor (See Excel Table S10) and a developmental/reproductive toxicant (See Excel Table S10).
Biomarker. No papers were found on a biomarker for fenamidone.
Recommendation: Since the predominate toxicity data were from in vitro tests, more toxicity research is needed, and a biomarker needs to be developed. Biomonitoring of fenamidone is deferred pending additional data.

Fenhexamid.

Exposure. Fenhexamid, a fungicide, was reported in surface water (Robles-Molina et al. 2014) and in food commodities (Esteve-Turrillas et al. 2016; USDA 2017), but no reports were found for other environmental or exposure media. In 708 samples of grapes collected in 2015, it was detected at a frequency of 36% with a mean of 171 ng/g (USDA 2017). It is being used at an increasing rate on vineyards. It has also been measured in strawberries near the European Union maximum residue level of 3.0 mg/kg (Angioni et al. 2004). EPA predicted exposure was in the 10^{-8} mg/kg-bw/day range (See Excel Table S2).
Toxicity. In a summary of available data, critical effects were reported for hepatotoxicity in dogs and decreased body weights, increased food consumption, and liver and kidney effects in rat and mouse studies. Only 1 neurotoxicity study was reviewed; however, the findings were not toxicologically significant. The findings in the developmental studies in rats and rabbits lead to conclusion that fenhexamid is not a developmental toxicant and that it did not impair reproduction (Canada 2003; U.S. EPA 1999). *In vitro* studies suggest that it is an antiestrogenic compound (Judson et al. 2015; Orton et al. 2011; Teng et al. 2013) and estrogen receptor alpha agonist and a week estrogen receptor binder (Medjakovic et al. 2014). There likely was an overlap between *in vitro* assay activity and predicted exposures (Wetmore et al. 2012), specifically for 1 assay associated with *in vivo* hematologic and adrenal effects. It has shown positive neurotoxicity effects in an *in vitro* assay (Regueiro et al. 2015). HTP *in vitro* testing activated 8 assays, including thyroid, estrogen, and androgens (See Excel Table S9). Predictive modeling suggested that fenhexamid may be a developmental toxicant (See Excel Table S10) and binds to the androgen and β-estrogen receptors (See Excel Table S11).
Biomarker. No papers on a biomarker for fenhexamid were found.
Recommendation: Even though there is evidence for endocrine disruption, biomonitoring is deferred until a biomarker is developed.

Fluroxypyr-meptyl.

Exposure. No information regarding environmental media or exposure levels were found for fluroxypyr-meptyl. EPA predicted exposure was in the 10^{-8} mg/kg-bw/day range (See Excel Table S2).
Toxicity. Developmental toxicity was observed but at high doses in the rat (Durkin 2009). Reproductive studies showed no impact on fertility (parental, reproduction, and offspring) nor signs of neurotoxicity (EFSA 2011b; U.S. EPA 1998a). There likely was an overlap between *in vitro* assay activity and predicted exposures (Wetmore et al. 2012), specifically in 6 assays associated with *in vivo* developmental and kidney effects. Fluroxypyr-meptyl was active in 2 obesity and 2 neurotoxicity HTP *in vitro* assays (See Excel Table

S9). Predictive modeling suggested fluroxypyr-meptyl was a developmental toxicant (See Excel Table S10).
Biomarker. Metabolism studies with human and rat skin indicate complete metabolism during its passage through the skin occurred (Hewitt et al. 2000). We did not identify any studies on the identification of biomarkers.
Recommendation: Since the toxicity information is sparse, more research is needed on this compound, and based on the absence of a reported biomarker, biomonitoring is deferred pending further data.

2-Hydroxyatrazine.
Exposure. 2-Hydroxyatrazine, a hydrolysis product of the herbicide atrazine, is found in groundwater, finished, and untreated drinking water (USDA 2017). For example, it was detected in 89% with a range of 2-740 ng/L of 207 samples collected in 2012 (USDA 2017). It has been measured in urine (Barr et al. 2007), although population exposure was underestimated, since it was a minor metabolite.
Toxicity. A review focused on endocrine disruption was published (Jablonowski et al. 2011) that discusses the endocrine disruption in wildlife; however, it notes the lack of studies on human health. Predictive modeling suggested that 2-hydroxyatrazine was a developmental toxicant (See Excel Table S10).
Biomarker. A metabolite biomarker was measured in urine (Barr et al. 2007).
Recommendation: HTP *in vitro* testing is recommended. Based on insufficient data on toxicity and the need to optimize the metabolite choice for a biomarker, biomonitoring is deferred pending further data.

Isoxaben.
Exposure. Biomonitoring and environmental media data were not found for isoxaben, an herbicide. EPA predicted exposure was in the 10^{-8} mg/kg-bw/day range (Table S1).
Toxicity. Developmental toxicity was reported in the rat (U.S. EPA 1988a). Isoxaben was not classified as a "possible adverse effect", due to evidence of maternal toxicity at levels which elicit reproductive effects, and because there was a high reproductive effects NOEL. There likely was an overlap between *in vitro* assay activity and predicted exposures (Wetmore et al. 2012), specifically in 1 assay associated with *in vivo* enzyme and liver effects. In HTP *in vitro* testing, it activated the neurotoxic, obesity, and steroidogenesis assays (See Excel Table S9).
Biomarker. After oral administration to rats, 90% is excreted in the feces within 48 hours and about 10% of isoxaben is converted into about 15-20 metabolites that are excreted in the urine. No accumulation of either parent or metabolites in cell tissue occurs (PubChem 2018c).
Recommendation: Based on some reported *in vitro* studies finding toxicity, a biomarker should be developed and measured in the U.S. population.

Metolachlor.
Exposure. Metolachlor, an herbicide, was reported in water, human hair, serum and cord blood (Schummer et al. 2012; USDA 2017; Whyatt et al. 2003). The frequency of detection in hair of French farmworkers was low at 6% with a median of 14.5 pg/mg (Schummer et al. 2012). Metolachlor was detected in 5% of 199 plasma samples from pregnant women with a range of ND to 5 pg/g CRT, and in 10% of 211 cord blood plasma samples with a range of ND to 11 pg/g CRT (Whyatt et al. 2003). In a longitudinal study, it was detected in 16.9% of urine samples of farmworkers (Arcury et al. 2009). In 232 water samples collected throughout the United States, it was detected at a frequency of 71% with a range of 2.5 to 250 ng/L (USDA 2017). In another study in groundwater it was detected in 33% of the samples with a median of 68.3 ng/L and in surface water at a 62% frequency with a median of 37 ng/L (Reilly et al. 2012). In a study of surface waters in Arkansas, it was detected in 13% of 485 samples with a median of 0.7 ug/L (Senseman et al. 1997). EPA predicted exposure was in the 10^{-7} mg/kg-bw/day range (See Excel Table S2).
Toxicity. No reproductive effects were observed in rat and dog studies as well as neurotoxicity in traditional studies (MDH 2018c; EU 2004; U.S. EPA 1990). *In vivo* studies on developmental and endocrine effects were not identified. HTP *in vitro* testing found neurotoxic and obesity activity (See Excel Table S9). Predictive modeling suggested it was a developmental toxicant (See Excel Table S10).
Biomarker. Metolachlor mercapturate is the major metabolite of metolachlor in human urine (Curwin et al. 2007; Driskell and Hill 1997) and has served as the biomarker of exposure to metolachlor (Arcury et al. 2009; Hines et al. 2003). The parent compound is measured in hair and plasma.
Recommendation: Since information is sparse on developmental and endocrine effects, biomonitoring is deferred pending further toxicity data.

Prometon.

Exposure. Prometon, an herbicide, was measured in water (USDA 2017). It was detected in 76% of 232 finished drinking water samples collected nationally, with a range of 0.28 to 82 ng/g (USDA 2017). Published data for other environmental and exposure media were not found. EPA predicted exposure was in the 10^{-8} mg/kg-bw/day range (See Excel Table S2).

Toxicity. Developmental, reproductive, endocrine disruptor, and neurotoxicity data are sparse. A multi-generational developmental study in rats and rabbits showed no increased susceptibility in fetuses or neonates (U.S. EPA 2008). There likely is an overlap between *in vitro* assay activity and predicted exposures (Wetmore et al. 2012), specifically in 1 assay associated with *in vivo* developmental effects. Prometon was active in 3 obesity HTP *in vitro* assays (See Excel Table S9). Predictive modeling results suggest prometon is a developmental toxicant (See Excel Table S10).

Biomarker. No information was found on biomarkers of exposure for biomonitoring of prometon.

Recommendation: Since the amount of toxicity data is sparse and considering the absence of a developed biomarker, more research is needed in these areas. Thus, biomonitoring is deferred pending further research.

Tetrahydrophthalimide (THPI).

Exposure. THPI is formed as a metabolite of captan through microbial metabolism in the environment; thus, exposure may also occur to it. It has been measured in plasma with a detection frequency of 50% and a range of ND to 43 pg/g, and in cord blood with a detection frequency of 44% and a range of ND to 37 pg/g (Whyatt et al. 2003). THPI was also detected in food commodities (USDA 2017). For example, it was detected 52.7% of 706 strawberry samples collected nationwide, with a mean of 557 ng/g (USDA 2017).

Toxicity. No developmental, reproductive, neurological, and endocrine toxicity was found for THPI.

Biomarker. THPI is a biomarker of exposure that can be measured in serum and cord blood.

Recommendation: Given the absence of toxicity data for THPI, it is deferred from biomonitoring pending additional data. We recommend THPI be tested in HTP *in vitro* assays for toxicity.

Quaternary Amines

Category B:

Benzylhexadecyldimethylammonium chloride (BAC C16).

Exposure. Similar to BAC C12, BAC C16 was detected in commercial grapefruit seed extract in the United States (Takeoka et al. 2005). It is also detected in U.S. surficial sediments (median: 320 ng/g) (Li and Brownawell 2010), in sewage (Clara et al. 2007); and in a waste water treatment plant in Austria (Kreuzinger et al. 2007); and in effluent, sludge, sediment, fish muscle, and fish liver samples in Europe (Kaj et al. 2014). An EPA predicted exposure value in the 10^{-7} mg/kg-bw/day range was reported (See Excel Table S2).

Toxicity. Studies suggested cytotoxicity in human corneal and conjunctival epithelial cell lines (Pellinen et al. 2012) and association with corneal barrier dysfunction (Uematsu et al. 2010). Compared to BAC C12, C16 homolog was less toxic (Pellinen et al. 2012) in both the human corneal and conjunctival epithelial cell lines (Pellinen et al. 2012). BAC C15 activated the calcium ion-channel in the HTP *in vitro* assay test (See Excel Table S9). It has a low affinity for endocrine disruptor receptors based on results from predictive models (See Excel Table S11).

Biomarkers. No information was found.

Recommendation. Category B. A biomarker should be developed, and it should be measured in a non-occupationally exposed U.S. population to determine exposure prevalence.

Didecyldimethylammonium chloride (DDMAC).

Exposure. DDMAC was found in fruits and vegetables in the European Union (Diez et al. 2016). In Austria, it was detected in surface water (Martinez-Carballo et al. 2007), sewage (Clara et al. 2007), sediment and sludge samples (Martinez-Carballo et al. 2007), and a waste water treatment plant (Kreuzinger et al. 2007). It was also detected in U.S. surficial sediments (median: 190 ng/g) (Li and Brownawell 2010) and in effluent, sludge, sediment, fish muscle, and fish liver samples in Europe (Kaj et al. 2014). An EPA predicted exposure value in the 10^{-7}mg/kg-bw/day range was reported (See Excel Table S2).

Toxicity. Several *in vivo* studies suggested DDMAC's potential reproductive and developmental toxicity, including 1) causing neural tube defects in mice and rats (oral dosing and ambient exposure of DDMAC in combination with BAC mixtures) (Hrubec et al. 2017), 2) causing subfertility in mice (oral dosing and

ambient exposure of DDMAC in combination with BAC mixtures) (Melin et al. 2016), and 3) decreasing fertility and fecundity in mice (Melin et al. 2016). Increased body weight and lung weight was reported in rats (Kim et al. 2017; Lim and Chung 2014). In mice, DDMAC was found to induce pulmonary inflammation and fibrosis (intratracheally instilled) (Ohnuma et al. 2010), alter the pulmonary defense system (intratracheally instilled) (Ohnuma et al. 2011), and induce pulmonary cytotoxicity and inflammation (intratracheally instilled, in combination with ethylene glycol) (Do et al. 2016). DDMAC activated 6 neurotoxicity, 6 obesity, and 3 endocrine HTP *in vitro* assays (See Excel Table S9). It has a low affinity for the endocrine disruptor receptors based on results from predictive models (See Excel Table S11).

Biomarker. No information was found.

Recommendation. Category B. Biomonitoring is deferred pending the development of a biomarker and its measurement in a non-occupationally exposed U.S. population to determine exposure prevalence.

N, N-Dimethyl-N-benzyl-N-octadecylammonium chloride (BAC C18).

Exposure. One relatively old study detected BAC C18 from 38 hair care products out of the 53 products tested from the New York area (Morrison et al. 1983). Similar to its C16 homolog, it was detected in U.S. surficial sediments (median: 490 ng/g) (Li and Brownawell 2010), in sewage (Clara et al. 2007), and in a waste water treatment plant in Austria (Kreuzinger et al. 2007); in effluent, sludge, sediment, fish muscle; and fish liver samples in Europe (Kaj et al. 2014). An EPA predicted exposure value in the 10^{-7} mg/kg-bw/day range was reported (See Excel Table S2).

Toxicity. BAC C18 activated two ion-channel assays in HTP *in vitro* testing (See Excel Table S9). Predictive models suggest that BAC C12 has a low affinity for the endocrine disruptor receptors (See Excel Table S11).

Biomarkers. No papers on biomarkers were found.

Recommendation. Category B. There is concern regarding BAC C18 toxicity. A biomarker should be developed, and it should be measured in a non-occupationally exposed U.S. population to determine exposure prevalence.

Category C:

1-(Benzyl)quinolinium chloride.

Exposure. Apart from the predicted exposure range being in the 10^{-8} mg/kg-bw/day range, no information was found regarding exposure (See Excel Table S2).

Toxicity. No *in vivo* toxicity data were found. This compound activated 3 neurotoxicity and 2 obesity HTP *in vitro* assays (See Excel Table S9). Predictive modeling found that 1-(Benzyl)quinolinium chloride can bind to 9 out of the 16 endocrine-related proteins with highest binding propensity (See Excel Table S11), suggesting that it has a very strong affinity for the endocrine disruptors.

Biomarker. No information on biomarkers was found.

Recommendation. Category C. There is enough concern regarding its toxicity that a biomarker should be developed, and measurement of exposure prevalence should be done in a non-occupationally exposed U.S. population.

Category D:

Benzyldimethyldodecylammonium (BAC C12).

Exposure. BAC C12 was detected in commercial grapefruit seed extract in the United States (Takeoka et al. 2005). In Austria, it was detected in surface water (Martinez-Carballo et al. 2007), sewage (Clara et al. 2007), and a waste water treatment plant in (Kreuzinger et al. 2007). In France, roofs can be treated against moss growth with benzalkonium, leading to contamination of the runoff. One study from France showed the presence of BAC C12 in urban storm water samples (Ohnuma et al. 2011). One U.S. study detected BAC C12 in surficial sediments (median: 140 ng/g) (Li and Brownawell 2010). It has also been detected in effluent, sludge, and sediment samples in Europe (Kaj et al. 2014). An EPA predicted exposure value in the 10^{-7} mg/kg-bw/day range was reported (See Excel Table S2).

Toxicity. *In vitro* studies reported cytotoxicity in human corneal and conjunctival epithelial cell lines (Pellinen et al. 2012) and association with corneal barrier dysfunction (Uematsu et al. 2010). Predictive models suggest that BAC C12 has a low affinity for endocrine disruptor receptors (See Excel See Excel Table S10).

Biomarkers. No information was found.

Recommendation. Category D. Testing with HTP *in vitro* assays for endocrine, developmental, neurotoxic,

and obesity effects is recommended, and if concerns for toxicity are discovered, then a biomarker should be developed, and it should be measured in a non-occupationally exposed U.S. population to determine exposure prevalence.

3-Methylbenzethonium chloride.

Exposure. This compound is an over-the-counter (OTC) drug added to ointments for treating diaper rash (FDA 1965). An EPA predicted exposure value in the 10^{-7} mg/kg-bw/day range was reported (See Excel Table S2) (U.S. EPA 2017b).

Toxicity. No information on toxicity was found.

Biomarker. No information on a biomarker for 3-methylbenzethonium chloride was found.

Recommendation. Category D. This compound should be tested in HTP *in vitro* assays for endocrine, developmental, neurotoxic, and obesity effects. If warranted from the toxicity assay results, a biomarker should be developed and measurement of exposure prevalence should be done in a non-occupationally exposed U.S. population.

Dimethyldiallylammonium chloride (DADMAC).

Exposure. DADMAC was detected in Japanese river in samples collected from 1998-2006 (Miura et al. 2008). An EPA predicted exposure value in the 10^{-7} mg/kg-bw/day range was calculated (See Excel Table S2) (U.S. EPA 2017b).

Toxicity. The toxicity of DADMAC is less known. One study found DADMAC was associated with anaphylactoid reactions to general anesthesia (Weston and Assem 1994). Predictive models suggested that DADMAC is carcinogenic and has a low affinity for the endocrine disruptor receptors (See Excel Table S11).

Biomarker. No information was found.

Recommendation. Category D. Biomonitoring is deferred pending testing results from HTP *in vitro* assays for endocrine, developmental, neurotoxic, and obesity effects. Biomarker development should be undertaken, pending problematic toxicity results, and its measurement in a non-occupationally exposed U.S. population is recommended.

N, N, N-Trimethyloctadecan-1-aminium chloride (stearyltrimethylammonium, or ATMAC C18). ***Exposure.*** ATMAC C18 was detected in surficial sediments in the United States. It was measured together with ATMAC C16 (median: 520 ng/g) in effluent (Li and Brownawell 2010), sludge, sediment, fish muscle, and fish liver samples in several countries in Europe (Kaj et al. 2014). An EPA predicted exposure value in the 10^{-6} mg/kg-bw/day range was reported (See Excel Table S2).

Toxicity. ATMAC C18 activated the calcium ion-channel in the HTP *in vitro* assay (See Excel Table S9). Predictive models suggest that ATMAC C18 has a low affinity for endocrine disruptor receptors (See Excel Table S11).

Biomarkers. No information was found.

Recommendation. Category D. Additional data on toxicity are needed. If concerns for toxicity are discovered, then a biomarker should be developed, and it should be measured in a non-occupationally exposed U.S. population to determine exposure prevalence.

Quaternium-15.

Exposure. Quaternium-15 is a formaldehyde releaser (formaldehyde is a known carcinogen) (Becker et al. 2010). We found no exposure information on Quaternium-15.

Toxicity. The Final Report of the Amended Safety Assessment of Quaternium-15 noted that "the weight of evidence suggested that a 0.2% concentration is not a sensitizer and that cosmetic products containing Quaternium-15 up to that level are safe" (Becker et al. 2010). One study of 391 children aged 0-18 years suggested a relationship between this compound and contact dermatitis – 3.6% of the study participants had a positive patch test reaction to Quaternium-15 (Zug et al. 2008). Although no positive results were reported based on predictive models, Quaternium-15 is listed in the Hazardous 100+ List of Chemicals of High Concern as a carcinogen (SaferChemicals 2017).

Biomarker. No information was found.

Recommendation. Category D. Testing in HTP *in vitro* assays for endocrine, developmental, neurotoxic, and obesity effects is recommended. If assays are positive, then development of a biomarker, and its measurement in a non-occupationally exposed U.S. population to determine exposure prevalence should be done.

Quaternium-52. ***Exposure.*** No information was found on exposure to Quaternium-52. ***Toxicity.*** Except for one study that found it was associated with anaphylactoid reactions to general anesthesia (Weston and Assem 1994), little toxicity information was found. ***Biomarker.*** No information was found. ***Recommendation.*** Category D. Testing in HTP *in vitro* assays for endocrine, developmental, neurotoxic, and obesity effects should be done. If warranted by positive assay results, then development of a biomarker and its measurement in a non-occupationally exposed U.S. population to determine its expsoure prevalence should be undertaken.
Additional Quaternary Amines We did not find any relevant literature regarding exposure or toxicity for the following compounds: Category D: QACs, di-C14-18-alkyldimethyl, Me sulfates, Behentrimonium methosulfate, Benzyldimethyl[2-[2-[[4-(1, 1, 3, 3-tetramethylbutyl)-m-tolyl]oxy]ethoxy]ethyl]ammonium chloride, Dodecyldimethyl(4-ethylbenzyl) ammonium chloride, Quaternium-24, Octyl decyl dimethyl ammonium chloride, and Tetradonium bromide ***Recommendation:*** All Category D. Testing in HTP *in vitro* assays for endocrine, developmental, neurotoxic, and obesity effects should be done. If warranted by positive assay results, then development of a biomarker(s) and measurement in a non-occupationally exposed U.S. population should be done to determine expsoure prevalence.

Table S5. Chemicals with a Low Priority for Biomonitoring in ECHO

Since environmental contamination and exposure may change over time, intermittent reassessment of a chemical's priority for biomonitoring is recommended. For chemicals assigned a low priority at this time, we recommend tracking exposure and chemical usage to assess whether exposure levels or uses have increased.

Alternative Flame Retardants

2,3,5,6-Tetrabromo-*p*-xylene (pTBX).

Exposure. Very low levels have been quantified in air and house dust (mean 0.32 ng/g) (Larsson and Berglund 2018; Shoeib et al. 2012; Venier et al. 2012). No information on exposure was found. There is a paucity of information on pTBX exposure in humans.

Toxicity. No relevant information was found.

Biomarker. No papers were found.

Recommendation. Based on the low levels in house dust, pTBX is assigned a low priority for biomonitoring in ECHO at this time.

1,2-Bis(2,4,6-tribromophenoxy) ethane (BTBPE).

Exposure. BTBPE was quantified in ambient air and house dust (Allgood et al. 2017; Cristale et al. 2016; Hoh et al. 2005; Mitro et al. 2016; Stapleton et al. 2014). It was not detected in a Norwegian serum samples (Cequier et al. 2015a). The geometric mean for BTBPE levels in house dust was 27 µg/g (Mitro et al. 2016), and in a mega-analysis of house dust, the median of reported medians was 5.3 ng/g (Larsson and Berglund 2018). BTBPE was not detected in human plasma in one study, while it was quantified in serum and breast milk in other studies; however, the frequency of detection was <5% and the levels were very low (Karlsson et al. 2007; Zhou et al. 2014). The parent compound was the measured biomarker in these studies.

Toxicity. BTBPE was found to affect thyroid hormone deiodinase and sulfotransferase effects in human bioassays (Smythe et al. 2017).

Recommendation. Based on the low detection frequency in human biofluids, BTBPE is assigned a low priority for ECHO at this time.

2,3-Dibromopropyl 2,4,6-tribromophenyl ether (TBP-DBPE).

Exposure. The median levels of TBP-DBPE in house dust were close to the detection limit (Liagkouridis et al. 2017). It was found in some food samples (mean 32 pg/g) (Sahlstrom et al. 2015).

Toxicity. In one *in vitro* study, TBP-DBPE was reported as a possible androgen receptor antagonist and neuronal and endocrine disrupter (Kharlyngdoh et al. 2015). Predictive modeling results suggest a low probability that TBP-DBPE is a developmental and reproductive toxicant (See Excel See Excel Table S10).

Biomarker. No data.

Recommendation. Even though there is an indication of possible endocrine, reproductive, and neurotoxicity effects, based on the low levels in the environment, a low priority is assigned for biomonitoring in ECHO at this time.

Alternate Plasticizers

Di-butyl adipate (DBA).

Exposure. DBA was measured in house dust at very low levels, near detection limits, with a 25% detection frequency, and in indoor air (mean of 45 ng/m^3) (Fromme et al. 2015; Larsson and Berglund 2018), and in another study at 0.002-1.5 µg/m^3 (Takeuchi, S et al. 2014).

Toxicity. No publications on toxicity were found. Predictive QSAR modeling results suggest that DBA is a reproductive toxicant (See Excel Table S10).

Biomarkers. No papers were found on biomarkers.

Recommendation. Based on the very low levels in environmental media, a low priority for biomonitoring in ECHO is recommended.

Di-butyl sebacate (DBS) [dibutyl decanedioate].

Exposure. DBS was measured in processed cheese and cooked meats (76-137 mg/kg and 76-137 mg/kg, respectively) (Castle et al. 1988), but it was not detected in baby foods (Tsumura et al. 2002). It was measured at tens of nanograms in an indoor air study of homes (Takeuchi, S et al. 2014). An EPA predicted median exposure value in the 10^{-7} mg/kg-bw/day range was reported (See Excel Table S2).

Toxicity. ATSDR has evaluated the noncancer oral toxicity data for DBS, but did not derive any oral minimal risk levels (MRLs) due to lack of adequate human and animal data (ITER 2018b).

Biomarker. No information was uncovered on biomarkers.

Recommendation. Based on low exposure and the absence of reported toxicity at this time, a low priority is recommended for biomonitoring in ECHO.

Dioctyl succinate (DOS).

Exposure. Data on exposure, environmental levels, or biomarkers for DOS are scarce. An EPA predicted median exposure value in the 10^{-7} mg/kg-bw/day range was reported (See Excel Table S2).

Toxicity. In a toxicological study with chicks, DOS did not induce encephalomalacia during the experimental period of 22 days, but at the twenty-third day after termination of the experimental period, one chick showed the symptom of encephalomalacia (Ikumo and Yoshida 1974). Relevant toxicity studies were not found. This compound did not activate any *in vitro* assays (See Excel Table S9).

Biomarker. Biomarker studies were not found.

Recommendation. Based on low exposure and the absence of reported toxicity at this time, a low priority for biomonitoring is recommended.

Environmental Phenols

4-Methyl phenol (p-Cresol).

Exposure. 4-Methyl phenol (p-cresol) was measured in air (Borhan et al. 2012; Morville et al. 2006; Sturaro et al. 2010) and water samples (Kolpin et al. 2002). 4-Methyl phenol was detected in urine samples of children (Altieri et al. 2011; Gabriele et al. 2014) and workers (e.g., (Bieniek 1994). An EPA predicted median exposure value in the 10^{-6} mg/kg-bw/day range was reported (See Excel Table S2).

Toxicity. Dietary concentrations of 3,000 ppm appeared to have minimal effect levels for increases in liver and kidney weights and deficits in liver function. Histopathologic changes, including bone marrow hypocellularity, irritation to the gastrointestinal tract and nasal epithelia, and atrophy of female reproductive organs, occasionally occurred at 10,000 ppm, but were more common at the high dose of 30,000 ppm (Dietz 1992). Reproductive toxicity was expressed by a reduced litter size when mice were exposed to ortho-cresol (OCRE) and a mixture of meta and para-cresol in 2 studies (Izard 1992).

Biomarker. Methyl phenol was measured using urinary biomarkers (Bieniek 1994; Brega et al. 1990; Dills et al. 1997).

Recommendation. Because p-cresol is also produced endogenously via metabolism of tyrosine or toluene by gut bacteria (Gabriele et al. 2014), measurement of urinary concentrations is not recommended as a biomarker of exposure to 4-methyl phenol. Instead, a proxy-exposure approach may be required.

Organophosphorus-based Flame Retardants

Tris (2,3-dibromopropyl) phosphate (TBP).

Since 2008, TBP has not been permitted in treatment of children's garments under the Federal Hazardous Substances Act (U.S.CPSC 2008)**.** The last exposure assessment was conducted in 1978 with the measurement of TBP in urine (Blum et al. 1978). A mega-analysis of house dust indicated the median of reported medians was 300 ng/g (Larsson and Berglund 2018). A federal review was conducted by the National Toxicology Program in 2016. There were no new publications on toxicity/health effects found for the past two decades.

Recommendation. Since TBP was banned, a low priority for biomonitoring is recommended.

Pesticides

Imazapyr.

Exposure. Information on the occurrence of imazapyr, an herbicide, in environmental media and human exposure is limited. It was found in treated and untreated drinking water samples (USDA 2017). For example, in 2010, it was detected at a frequency of 50% with a range of 5-375 ng/L (USDA 2017). EPA predicted exposure was in the 10^{-7} mg/kg-bw/day range (See Excel Table S2).

Toxicity. Based on a study in dogs, an RfD of 2.5 mg/kg-d and an FQPA safety factor of 1 was calculated (U.S. EPA 2006b). Multi-generational reproductive and developmental studies report no adverse effects on reproduction or development (AMEC Geomatrix 2009; WHO 2013). Predictive modeling suggested that imazapyr was a developmental toxicant (See Excel Table S10).

Biomarker. No information was found on a biomarker for imazapyr.

Recommendation: Based on the low toxicity reported for imazapyr, it is assigned a low priority for biomonitoring in ECHO.

Metolachlor ethane sulfonic acid (MESA).

Exposure. Metolachlor ethane sulfonic acid is a microbial metabolite of metolachlor. It was detected in groundwater, untreated and finished drinking water, and well water (Sutherland-Ashley K et al. 2017; USDA 2017). It was detected in 88% of 232 water samples collected throughout the United States, with a range of 0.6 to 2,500 ng/L (USDA 2017). In California, it has been detected in 20% of the wells tested (OEHHA 2017). EPA predicted exposure was in the 10^{-7} mg/kg-bw/day range (See Excel Table S2). No other environmental media or exposure data were found.

Toxicity. Minnesota Department of Health has concluded that MESA does not cause developmental effects up to 1000 mg/kg-d (MDH 2018a; Sutherland-Ashley K et al. 2017). No studies were found that address endocrine, reproductive, or neurotoxic effects of MESA (MDH 2018a; Sutherland-Ashley K et al. 2017). U.S. EPA Tier 1 assays for endocrine disruptor screening yielded no convincing evidence, which is consistent with predictive modeling. HTP *in vitro* assay testing was not conducted. Predictive modeling suggested MESA was a developmental toxicant (See Excel Table S10).

Biomarker. No information was found on biomarkers for MESA.

Recommendation: Based on the negative *in vivo* results, MESA is assigned a low priority for biomonitoring in ECHO.

Metolachlor oxanilic acid (MOXA).

Exposure. MOXA was reported to occur in groundwater, untreated and finished drinking water, and well water (Sutherland-Ashley K et al. 2017; USDA 2017). In 232 samples collected throughout the United States, MOXA was detected at an 82% frequency with a range of 3-1,100 ng/L (USDA 2017), and in 20% of well samples in California (Sutherland-Ashley K et al. 2017). EPA predicted exposure was in the 10^{-7} mg/kg-bw/day range (See Excel Table S2). No other environmental media or exposure data were found.

Toxicity. Developmental effects were shown only at high doses, and neurotoxic effects were noted in limited studies (Butenhoff et al. 2012; MDH 2018b; Sutherland-Ashley K et al. 2017). U.S. EPA Tier 1 assays for endocrine disruptor screening yielded no convincing evidence, which is consistent with predictive modeling. HTP *in vitro* assay testing was not conducted.

Biomarker. No information was found on biomarkers for MOXA.

Recommendation: Based on toxic effects shown only at high doses, this pesticide is considered low priority for biomonitoring in ECHO. We note that it may be useful to conduct *in vitro* studies on this chemical.

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