Endocrine Disrupting Chemicals and Obesity

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ALL OF OUR STUDY PARTICIPANTS!

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Objectives

- Define endocrine disrupting chemicals (EDCs)
- Identify potential physiologic pathways for EDCs
- Recognize how EDCs may contribute to development of obesity/diabetes
- Discuss strategies for preventing EDC exposures to benefit health and the economy
“Scientific societies such as the Endocrine Society should partner with other organizations with the scientific and medical expertise to evaluate effects of endocrine disrupting chemicals in humans”

While diet and physical activity remain the leading drivers of obesity, dietary contaminants are increasingly recognized as contributors.

Obesogen hypothesis:

- Xenobiotic chemical exposure in early life can disrupt normal developmental and homeostatic controls over adipogenesis/energy balance and predispose individuals to gain fat mass (Grun/Blumberg 2006)

Safer alternatives and strong policy can produce health and economic benefits
Endocrine Disrupting Chemicals (EDCs)

An agent that interferes with natural hormones in the body responsible for the maintenance of reproduction, development, homeostasis, and behavior.

Transgenerational effects
Types of EDCs

Natural
- Soy
- Fungal estrogens

Synthetic
- Hormones
- DES
- Phthalates
- Bisphenol A
- Some pesticides
- Industrial by-products
- Some persistent organic pollutants (POPs)
Toxicologic Principles

- Dose – low dose vs. high dose
- Timing of Exposure – sensitive windows of vulnerability
- Half-life – short vs. persistent
- Biological Availability - body storage, circulation
- Toxicity – a standard battery of animal studies
- Genetics
- Risk Assessment - “Safe Dose” determined by formula that uses NOAEL (No Observed Adverse Effect Level) and multiplies by a safety factor
Fetal Origins of Adult Disease: The Barker Hypothesis

Env Exposure
Found in all of us

DEHP

MEHHP MEHP MEOHP MBP

DBP
Number of Phthalate Metabolites Found in Infant Urine Samples (N=163)
Male reproductive tract abnormalities like smaller anogenital distance, smaller penile width, reduced testicular descent

Obesity/insulin resistance

Neurodevelopmental changes in young children


Phthalates (DEHP/DBP) – anti-androgens

PPAR-γ

Bisphenol A (BPA) – estrogen

Prenatal exposure associated with:

- Development of tumors in the breast/prostate, obesity/metabolism changes, significant impacts on neurodevelopment (animal studies, low dose)
- Neurodevelopmental changes in children and adverse cardiovascular outcomes in adults
Mechanisms

GR = Glucocorticoid receptor
AhR = Aryl hydrocarbon receptor
PPAR = Peroxisome proliferator activated receptor
TR = thyroid hormone receptor
RXR = retinoid X receptor
PXR = Pregnane receptor

Casals-Casas C, Desvergne B. 2011.
Annu. Rev. Physiol. 73:135–62
Chemicals

Bisphenol A – weak estrogen

Phthalates – anti-androgen, PPAR-γ activator

PCBs/Dioxin – AhR activator, thyroxine
Diethylstilbestrol

Synthetic estrogen used to prevent miscarriage, premature labor

1st Gen: 40x incr risk of breast cancer

2nd Gen: vaginal adenocarcinoma in females, hypospadias in male

3rd Gen: possible increased risk of ovarian cancer, hypospadias

Figure 2
Medical journal advertisement for prenatal tablets with vitamins and diethylstilbestrol
Bisphenol A

Comprehensive, cross-sectional study of dust, indoor and outdoor air, and solid and liquid food in preschool age children suggested that dietary sources constitute 99% of BPA exposure.

Short half-life, excreted rapidly.

Ubiquitous exposure – over 90% of persons exposed in NHANES.

Banned from baby bottles and sippy cups by US Food and Drug Administration.
Bisphenol A and Obesity

Bisphenol A and DES – promotes adipogenesis at LOW DOSES

DES – synthetic potent estrogen
   Exposure in utero → obese offspring that continued to be obese with restricted caloric intake/increased exercise → 3rd generation also obese (increases in leptin, adiponectin, TG)

Bisphenol A – weak estrogen

In NHANES adults, high urinary BPA associated with increased incidence of:

1. Cardiovascular diagnoses
2. Diabetes

Iain A. Lang, PhD; Tamara S. Galloway, PhD; Alan Scarlett, PhD; William E. Henley, PhD; Michael Depledge, PhD, DSc; Robert B. Wallace, MD; David Melzer, MB, PhD JAMA. 2008;300(11):1303-1310. Published online September 16, 2008 (doi:10.1001/jama.300.11.1303).
<table>
<thead>
<tr>
<th>Quartile (Percentile)</th>
<th>Odds of Obesity</th>
<th>Prevalence</th>
<th>BMI Z-score</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (0-24%)</td>
<td>Reference</td>
<td>10.3% (7.5-13.1%)</td>
<td>Reference</td>
</tr>
<tr>
<td>2 (25-49%)</td>
<td>2.24 (1.54-3.24)**</td>
<td>20.1% (14.5-25.6%)</td>
<td>0.12 (-0.02 to 0.27)</td>
</tr>
<tr>
<td>3 (50-74%)</td>
<td>2.08 (1.46-2.96)**</td>
<td>19.0% (13.7-24.2%)</td>
<td>0.16 (0.01 to 0.30)*</td>
</tr>
<tr>
<td>4 (≥75%)</td>
<td>2.57 (1.72-3.83)**</td>
<td>22.1% (16.6-27.9%)</td>
<td>0.22 (0.06 to 0.39)*</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Odds of Obesity</th>
<th>BMI Z-score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Log-transformed BPA concentration</td>
<td>1.24 (1.08-1.44)**</td>
</tr>
</tbody>
</table>

Adjusted for: age group (6-11 and 12-19 years), Gender, Racial/ethnic group, Socioeconomic status, Caregiver education, Serum cotinine level (tobacco smoke exposure), Television watching, Caloric intake, Urinary dilution (creatinine)

Trasande et al. JAMA 2012
Phthalates - plasticizer
<table>
<thead>
<tr>
<th>Parent Compound</th>
<th>Metabolite</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benzyl butyl phthalate (BzBP)</td>
<td>Mono-n-butyl phthalate (MBP)</td>
</tr>
<tr>
<td></td>
<td>Monobenzyl phthalate (MBzP)</td>
</tr>
<tr>
<td>Dibutyl phthalate (DBP)</td>
<td>Mono-n-butyl phthalate (MBP)</td>
</tr>
<tr>
<td>Di-isobutyl phthalate (DiBP)</td>
<td>Mono-isobutyl phthalate (MiBP)</td>
</tr>
<tr>
<td>Diethyl phthalate (DEP)</td>
<td>Monoethyl phthalate (MEP)</td>
</tr>
<tr>
<td>Di-2-ethylhexyl phthalate (DEHP)</td>
<td>Mono-2-ethylhexyl phthalate (MEHP)</td>
</tr>
<tr>
<td></td>
<td>Mono-2-ethyl-5-hydroxyhexyl phthalate (MEHHP)</td>
</tr>
<tr>
<td></td>
<td>Mono-2-ethyl-5-oxohexyl phthalate (MEOHP)</td>
</tr>
<tr>
<td>Di-n-octyl phthalate (DnOP)</td>
<td>Mono-3-carboxypropyl phthalate (MCPP)</td>
</tr>
<tr>
<td>Dimethyl phthalate (DMP)</td>
<td>Mono-methyl phthalate (MMP)</td>
</tr>
</tbody>
</table>
Phthalates - plasticizer

- High production volume chemical – over 1 million pounds produced/used, over 90% of population exposed

- Short half-lives, <24 hours

- Di-2-ethylhexylphthalate (DEHP) is of particular interest because industrial processes to produce food frequently use plastic products containing DEHP.

- Strong evidence of anti-androgenic effects in animal and human studies

- Evidence for phthalates as potential obesogens

Hauser and Calafat 2005; Sathyanarayana 2008; Sathyanarayana et al. 2008; Schettler 2006; Fromme et al. 2007; Wormuth et al. 2006
Phthalate Exposure in Utero

Maternal Phthalate Exposure

Testis Testosterone Production (ng/testis)

Days post conception

• Mono-(2-ethylhexyl) phthalate (MEHP), a DEHP metabolite, increases expression of three peroxisome proliferator-activated receptors (PPARs)

• PPAR-γ in particular plays a critical role in adipogenesis in preadipocyte cell lines

• MEHP and MBzP increase adipogenesis in cell lines via PPAR-γ activation

• MEHP activates PPAR-γ transcription more selectively than rosiglitazone, a drug used to treat type 2 diabetes

Desvergne et al. 2009, Grun and Blumberg 2007
Urinary phthalates was associated with abdominal obesity and insulin resistance in adults in the 1999-2002 US National Health and Nutrition Examination Survey (NHANES).

No associations of quartiled urinary phthalate concentrations in 1209 children and adolescents in 1999-2002 NHANES with unstandardized measurements of BMI (Body Mass Index) or WC (waist circumference), although patterns of association varied by age and gender.

Positive relationships of LMW phthalates measured in 6-8 year olds and associations with BMI and WC one year later, among overweight children.

Race-specific (African American) associations in 2003-8 of LMW phthalates with BMI Z-score, overweight and obesity.

Phthalates – human epidemiology

- Strong cross-sectional association of urinary DEHP metabolites with insulin resistance in adolescents in NHANES 2003-8
- Levels of phthalates (DEHP) have decreased 17-37% in the US between 2001-10
- Increasing substitution with other HMW phthalates, particularly Diisodecyl-phthalate (DIDP) and Diisononyl-phthalate (DINP)
- Newer data (NHANES 2009-10) identify associations of DIDP with insulin resistance (as well as DEHP)

Attina and Trasande JCEM 2015
Polychlorinated Biphenyls (PCBs)/Dioxin

Persistent organic pollutants, very long half lives, high stability, and thermal resistance

- DDT often called most toxic substance to man
- DDT still used in other countries
- PCBs in electronics, waste by products, incineration, light ballasts
- Dioxin and dioxin like PCBs promote fat cell development, increase adipocyte size, and lead to insulin resistance
- PCBs promote inflammation/increased body weight and higher blood glucose levels
- Animals given high fat diet + chemical exposure gained more weight/adverse metabolic effects when compared to animals on high fat diet only

Kim et al. 2016
Arsenescu et al. 2008
Pereria and Rao 2006
Gray et al 2013
Wahlang et al. 2013
<table>
<thead>
<tr>
<th>Reference</th>
<th>Chemical</th>
<th>Study description (n)</th>
<th>Outcome assessment</th>
<th>Adjusted OR (95% CI)*</th>
<th>Exposure contrast†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Codru et al. 2007</td>
<td>PCB153</td>
<td>USA (Akwesasne), Mohawks; CS, ♀ (352)</td>
<td>FBG, medication</td>
<td>2.4 (1.0, 5.6)</td>
<td>T3 vs. T1 ng/g lipid adj</td>
</tr>
<tr>
<td>Codru et al. 2007</td>
<td>PCBs</td>
<td>USA (Akwesasne), Mohawks; CS, ♀ (352)</td>
<td>FBG, medication</td>
<td>3.2 (1.4, 7.5)</td>
<td>T3 vs. T1 ng/g lipid adj</td>
</tr>
<tr>
<td>Jørgensen et al. 2008</td>
<td>PCBs, dioxin-like</td>
<td>Greenland (west coast), Inuit; CS, ♀ (692)</td>
<td>OGTT, FBG</td>
<td>1.2 (0.4, 3.6)</td>
<td>Q4 vs. Q1 ng/g lipid adj (plasma)</td>
</tr>
<tr>
<td>Jørgensen et al. 2008</td>
<td>PCBs, non-dioxin-like</td>
<td>Greenland (west coast), Inuit; CS, ♀ (692)</td>
<td>OGTT, FBG</td>
<td>1.2 (0.4, 3.2)</td>
<td>Q4 vs. Q1 ng/g lipid adj (plasma)</td>
</tr>
<tr>
<td>Lee et al. 2006</td>
<td>PCB153</td>
<td>USA (NHANES 1999–2002); CS, ≥ 20 years; ♀ (2,106)</td>
<td>FBG, self-report</td>
<td>2.5 (1.1, 6)</td>
<td>&lt; 25th %ile vs. ND ng/g lipid adj</td>
</tr>
<tr>
<td>Rignell-Hydbom et al. 2007</td>
<td>PCB153</td>
<td>Spain (Menorca); CS, 6.5 years; ♀ (405)</td>
<td>Self-report</td>
<td>1.4 (0.8, 2.5)</td>
<td>Per 100 ng/g lipid increase, adj (serum, maternal cord)</td>
</tr>
<tr>
<td>Rylander et al. 2005</td>
<td>PCB153</td>
<td>Sweden (national registry), fishermen; CS, ♀ (196)</td>
<td>Self-report</td>
<td>1.20 (1.04, 1.39)</td>
<td>Per 100 ng/g lipid increase, adj</td>
</tr>
<tr>
<td>Rylander et al. 2005</td>
<td>PCB153</td>
<td>Sweden (national registry), fishermen’s wives; CS, ♀ (184)</td>
<td>Self-report</td>
<td>1.06 (0.75, 1.5)</td>
<td>Per 100 ng/g lipid increase, adj</td>
</tr>
<tr>
<td>Turyk et al. 2009b</td>
<td>PCBs</td>
<td>USA (Great Lakes), fish eaters; CS, ♀ (503)</td>
<td>Self-report, HbA1c</td>
<td>1.9 (0.7, 5.2)</td>
<td>Q4 vs. Q1 ng/g lipid adj</td>
</tr>
<tr>
<td>Turyk et al. 2009b</td>
<td>PCBs, dioxin-like</td>
<td>USA (Great Lakes), fish eaters; CS, ♀ (503)</td>
<td>Self-report, HbA1c</td>
<td>2.1 (1.1, 4.2)</td>
<td>T3 vs. T1 ng/g lipid adj</td>
</tr>
<tr>
<td>Uemura et al. 2008</td>
<td>PCBs, dioxin-like</td>
<td>Japan (multisite); CS, ♀ (1,374)</td>
<td>Self-report, HbA1c</td>
<td>3.07 (1.16, 8.81)</td>
<td>≥ 0.76 vs &lt; 13 vs. ≤ 0.76 ng TEO/g lipid adj</td>
</tr>
<tr>
<td>Ukopec et al. 2010</td>
<td>PCBs</td>
<td>Slovakia (eastern, “polluted”); CS, ≥ 21 years, ♀ (2,047)</td>
<td>FBG, 2 hr OGTT</td>
<td>1.77 (1.05, 3.02)</td>
<td>QU4 vs. QU1 ng/g lipid adj</td>
</tr>
<tr>
<td>Lee et al. 2010</td>
<td>PCB153</td>
<td>USA (multisite), CARDIA; NCC, ≥ 18 years; ♀ (180)</td>
<td>FBG, medication</td>
<td>0.8 (0.2, 2.6)</td>
<td>Q4 vs. Q1 ng/g lipid adj</td>
</tr>
<tr>
<td>Rignell-Hydbom et al. 2009</td>
<td>PCB153</td>
<td>Sweden (Lund) WHILA; NCC, ♀ (742)</td>
<td>OGTT</td>
<td>1.6 (0.61, 4)</td>
<td>&gt; 1.79 vs. ≤ 1.79 ng/mL</td>
</tr>
<tr>
<td>Wang et al. 2008</td>
<td>PCBs</td>
<td>Taiwan (Yucheng); NCC, ≥ 30 years; ♀ (244)</td>
<td>Self-report</td>
<td>5.5 (2.3, 13.4)</td>
<td>121.4 vs. 72.6 ng/g</td>
</tr>
<tr>
<td>Wang et al. 2008</td>
<td>PCBs</td>
<td>Taiwan (Yucheng); NCC, ≥ 30 years; ♀ (167)</td>
<td>Self-report</td>
<td>1.7 (0.7, 4.6)</td>
<td>98.4 vs. 53.9 ng/g</td>
</tr>
<tr>
<td>Turyk et al. 2009a</td>
<td>PCBs</td>
<td>USA (Great Lakes); prospective, fish eaters, ♀ (471)</td>
<td>Self-report</td>
<td>1.8 (0.6, 5) IRR</td>
<td>Per 100 ng/g lipid increase, adj</td>
</tr>
<tr>
<td>Vasiliiu et al. 2006</td>
<td>PCBs</td>
<td>USA (Michigan) PBB cohort, prospective, ♀ (696)</td>
<td>Self-report, 1.04 (11.0, 3.78) IDR</td>
<td>5.1–7.0 vs. ≤ 5.0 ng/mL</td>
<td></td>
</tr>
<tr>
<td>Vasiliiu et al. 2006</td>
<td>PCBs</td>
<td>USA (Michigan) PBB cohort, prospective, ♀ (688)</td>
<td>Self-report</td>
<td>1.74 (0.91, 3.34) IDR</td>
<td>&gt; 10 vs. ≤ 5.0 ng/mL</td>
</tr>
</tbody>
</table>

Taylor et al. 2013. EHP
Objective: To determine if following written guidelines to reduce exposures would lead to reductions in urinary BPA and phthalates to a similar extent as a dietary intervention

Randomized Trial
10 families with 2 kids each randomized to:

Arm 1: Provided catered diet made with fresh, organic, local foods and prepared/stored in non-plastics

Arm 2: Provide PEHSU written handouts
Pilot Study Results
### What Led to DEHP Spikes?

<table>
<thead>
<tr>
<th>DEHP (ng/g)</th>
<th>Current Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Butter</td>
<td>595</td>
</tr>
<tr>
<td>Heavy cream</td>
<td>488</td>
</tr>
<tr>
<td>Milk</td>
<td>673</td>
</tr>
<tr>
<td>Cheese</td>
<td>396</td>
</tr>
<tr>
<td>Egg yolk</td>
<td>39</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>DEHP (ng/g)</th>
<th>Current Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spices</td>
<td></td>
</tr>
<tr>
<td>Salt/pepper</td>
<td>&lt;26</td>
</tr>
<tr>
<td>Cayenne</td>
<td>707</td>
</tr>
<tr>
<td>Star anise</td>
<td>&lt;210</td>
</tr>
<tr>
<td>Ground coriander</td>
<td>21,400</td>
</tr>
<tr>
<td>Cumin</td>
<td>&lt;181</td>
</tr>
<tr>
<td>Ground cinnamon</td>
<td>&lt;28</td>
</tr>
</tbody>
</table>

*Sathyanarayana et al. 2012*
Lessons Learned

- Need more intensive intervention than one page handout

- Catered foods prepared with appropriate recommendations may not lead to reductions in exposures

- May take policy change to reduce exposures
Limiting canned food consumption and avoiding processed foods.

Intervention reduced mean concentrations of BPA by 66% and DEHP metabolites by 53–56%.

Rudel et al 2011
UW PATHWAYS (Karr/Sathyanarayana: Multi-PI)
1. Examine multiple exposures (phthalates, air pollution, maternal stress) and child airway and neurodevelopment

2. Examine exposures in relation to placental transcription

NYU Center for Obesity (Trasande: PI)
1. Multiple prenatal chemical exposures in relation to obesity and cardiovascular outcomes at ages 8-9 (PWV/DEXA/BAD)
Foods Might Serve Up High Levels of Chemicals Found in Plastics

Manufacturing process might introduce hormone-disrupting chemicals into dairy products, spices, researcher says

By Carina Storrs
HealthDay Reporter

WEDNESDAY, Feb. 27 (HealthDay News) -- Bisphenol A (BPA) and phthalates, two types of chemicals in plastics that have been linked to a number of health effects, could still find their way into your body even if you avoid foods that are shipped, stored or cooked using plastic materials, new research suggests.

The findings are based on a small study that followed 10 families for five days. Half of the families got catered meals made with fresh, local ingredients that were not stored or prepared with plastics.
Phthalates and BPA Exposure Reduction

We encourage providers to counsel families to prevent endocrine disrupting chemicals exposure to reduce the potential risk of harm.

Overall, women can reduce exposure to phthalates and bisphenol A by (1) reducing the consumption of processed foods, (2) increasing fresh and/or frozen foods, and (3) reducing consumption of canned foods.

Avoid the use of plastics with the recycling codes (often found on the outside bottom of containers) #3 and #7 because they can contain phthalates and/or bisphenol A.

Use a vacuum machine that is fitted with a HEPA filter to get rid of dust that may contain endocrine-disrupting chemicals.

When purchasing new products, ask the manufacturers what type of fire retardants were used.

Take shoes off when entering the house

Have good ventilation within your home
EDC-2: The Endocrine Society’s Second Scientific Statement on Endocrine-Disrupting Chemicals


The Endocrine Society’s first Scientific Statement in 2009 provided a wake-up call to the scientific community about how environmental endocrine-disrupting chemicals (EDCs) affect health and disease. Five years later, a substantially larger body of literature has solidified our understanding of plausible mechanisms underlying EDC actions and how exposures in animals and humans—especially during development—may lay the foundations for disease later in life. At this point in history, we have much stronger knowledge about how EDCs alter gene-environment interactions via physiological, cellular, molecular, and epigenetic changes, thereby producing effects in exposed individuals as well as their descendants. Causal links between exposure and manifestation of disease are substantiated by experimental animal models and are consistent with correlative epidemiological data in humans. There are several caveats because differences in how experimental animal work is conducted can lead to difficulties in drawing broad conclusions, and we must continue to be cautious about inferring causality in humans. In this second Scientific Statement, we reviewed the literature on a subset of topics for which the translational evidence is strongest: 1) obesity and diabetes; 2) female reproduction; 3) male reproduction; 4) hormone-sensitive cancers in females; 5) prostate; 6) thyroid; and 7) neurodevelopment and neuroendocrine systems. Our inclusion criteria for studies were those conducted predominantly in the past 5 years deemed to be
- BPA banned in baby bottles and sippy cups
  - But not in other food uses
- Costs of BPA Exposure in US
  - 12,404 cases of childhood obesity
  - 33,863 cases of newly incident coronary heart disease
  - Estimated social costs of $2.98 billion in 2008

Trasande Health Affairs 2014
HEALTH EFFECTS FROM ENDOCRINE DISRUPTING CHEMICALS COST THE EU 157 BILLION EUROS EACH YEAR. This is the tip of the iceberg: Costs may be as high as €270B.

**€157B Cost by Health Effect**

<table>
<thead>
<tr>
<th>Health Effect</th>
<th>Cost (€B)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male Reproductive Disorders</td>
<td>4</td>
</tr>
<tr>
<td>Premature Death</td>
<td>6</td>
</tr>
<tr>
<td>Obesity &amp; Diabetes</td>
<td>15</td>
</tr>
<tr>
<td>Neurological Impacts (including ADHD)</td>
<td>132</td>
</tr>
</tbody>
</table>

**NOTE:** The economic estimates do not include all costs associated with these conditions.

**€157B Cost by EDC Type**

<table>
<thead>
<tr>
<th>EDC Type</th>
<th>Cost (€B)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pesticides</td>
<td>120</td>
</tr>
<tr>
<td>Plastic: Phthalates &amp; BPA</td>
<td>26</td>
</tr>
<tr>
<td>Flame Retardants</td>
<td>9</td>
</tr>
<tr>
<td>Other</td>
<td>2</td>
</tr>
</tbody>
</table>

**SOME EDC-RELATED HEALTH OUTCOMES NOT INCLUDED:**
- Breast Cancer
- Prostate Cancer
- Immune Disorders
- Female Reproductive Disorders
- Liver Cancer
- Parkinson's Disease
- Osteoporosis
- Endometriosis
- Thyroid Disorders

**SOME EDCs NOT INCLUDED:**
- Atrazine
- 2,4-D
- Styrene
- Triclosan
- Nonylphenol
- Polycyclic Aromatic Hydrocarbons
- Bisphenol S
- Cadmium
- Arsenic
- Ethylene glycol

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Endocrine Disrupting Chemicals (EDCs) interfere with hormone action to cause adverse health effects in people.

**“THE TIP OF THE ICEBERG”**

The data shown to the left are based on fewer than 5% of likely EDCs. Many EDC health conditions were not included in this study because key data are lacking. Other health outcomes will be the focus of future research.

See Trasande et al. The Journal of Clinical Endocrinology & Metabolism
http://press.endocrine.org/edc
Benefits and costs of replacing BPA

- Potential cost of one BPA alternative, oleoresin = $0.022 per can
  - 100 billion aluminum cans are produced annually
  - 100 billion x $0.022 = $2.2 billion

- Potential benefit of replacing BPA with lining free of health effects = $1.74 billion
  - Does not include other effects (cognitive, asthma, breast cancer)

- Sensitivity analyses suggest as high as $13.8 billion

Trasande Health Affairs 2014
Tip of the Iceberg

- 80,000 chemicals in commerce
- Only 1% are tested before entering the consumer market
- Multi-disciplinary research is essential
- Past Co-chair of the Environmental Protection Agency Children’s Health Protection Advisory Committee
- National Academies of Science Committee on Low Dose Toxicity of Endocrine Disrupting Chemicals
Resources

- EPA website
- Washington state department of health website
- PEHSU
- Env Working Group (skin deep, green cleaning, pesticide in foods)
- Env Defense Fund (safe seafood)
- Clean Indoor Air (American Lung Association)
- UCSF Program In Reproductive Health
- Pediatric Environmental Health Specialty Units
While diet and physical activity remain the leading drivers of obesity, **dietary contaminants are increasingly recognized as contributors.**

Obesogen hypothesis asserts that xenobiotic chemical exposure in early life can disrupt normal developmental and homeostatic controls over adipogenesis/energy balance and predispose individuals to gain fat mass. (Grun/Blumberg 2006)

Safer alternatives and strong policy can produce health and economic benefits.
Webinars
Series of scientific webinars that provide a forum for discourse on scientific issues.
Live and On-Demand
Case Conferences
Journal Clubs
Grand Rounds
CE Available

Online Courses
Evidence-based online courses on a variety of children's environmental health topics.
Interactive and Self-Paced
CE Available

Resource Catalog
Fact sheets, journal publications, reports, and other resources for parents, community members, patients and healthcare professionals
Topics included:
Air Quality, Pesticides, Natural Disasters, BPA, Mold, Lead, Mercury