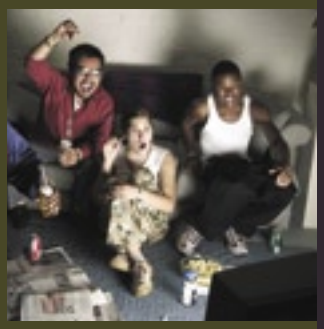

# Sick of Dust

## Chemicals in Common Products— A Needless Health Risk in Our Homes

Safer Products PROJECT

Pat Costner, Beverley Thorpe & Alexandra McPherson

MARCH 2005

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Acknowledgments to all the state groups who took part in the dust sampling and report reviews:

- Ecology Center, Michigan
- Washington Toxics Coalition
- Oregon Environmental Council
- The Alliance for a Healthy Tomorrow, Massachusetts
- Citizens Environmental Coalition, New York State
- Environmental Health Strategy Center, Maine
- Center for Environmental Health, California
- The Silicon Valley Toxics Coalition, California.

We wish to particularly thank the following foundations for their support

- John Merck Fund
- Panta Rhea Foundation
- Homeland Foundation
- Overbrook Foundation
- Mitchell Kapor Foundation
- New York Community Trust

We also thank Lowell Center for Sustainable Production, Pesticide Action Network and Silent Spring Institute.

A project of Clean Production Action


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CONCEPT/DESIGN/PRODUCTION
David Gerratt/NonprofitDesign.com

CHEMICAL HOUSE ILLUSTRATION
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LOGO & PRODUCT ICON ILLUSTRATIONS
Nate Walker

## APPENDIX I: Results of Chemicals Tested For, Occurrence, and Health Concern

| Target Chemicals | Uses | References |
|---|---|---|
| **Phthalates** | 80 – 90 percent of all phthalates are used in flexible PVC products, or vinyl – wall coverings, flooring, furniture, shower curtains, clothing, raincoats, shoes, toys, etc. The rest are used in paint, medical equipment, pesticides, and personal care products (perfume, nail polish, hairspray). DEHP is the predominant phthalate in flexible PVC products, while DEP and DBP are used most often in personal care products. Phthalates are also found in recycled paper products. | 1, 2 |
| **Alkylphenols and Alkylphenol ethoxylates** | Alkylphenols are used primarily as raw materials for the manufacture of alkylphenol ethoxylates. Alkylphenol ethoxylates are used as non-ionic surfactants, emulsifiers, lubricants or anti-oxidants in laundry detergents, textiles, leather, paints, disinfecting cleaners, all-purpose cleaners, spot removers, hair-coloring, cosmetics, adhesives, some plastics and pesticides. Nonylphenol is used as a spermicide. | 3 |
| **Polybrominated diphenyl ethers** | PBDEs are applied to textiles or incorporated into plastics, foams and components of electrical goods to prevent or slow the spread of fire. They are found in polyurethane foam products, foam padding in furniture, textiles, electrical appliances, televisions and computers. Decabrominated diphenyl ether, BDE 209, is used solely as a flame retardant in the hard, dense plastics of consumer electronics products (~ 80% production volume) and in the latex back coating of flame retardant upholstery textiles (~20 % production volume). | 4,5,6 |
| **Pesticides** | Pesticides are applied in and around homes for controlling infestations of various insects; applied to carpets, pre- and post-sale, to prevent or or slow infestations of insects, dust mites, and mold. They are also added to soaps, paints, and household cleaners. Inside uses of chlorpyrifos and diazinon recently restricted by the EPA due to health concerns. In agricultural areas, pesticides from neighboring agricultural use can drift into homes and schools. | |
| **Organotins** | Organotins are used to the greatest extent as heat and light stabilizers in PVC. They are found in PVC water pipes, PVC food packing materials (e.g., dioctyltin), glass coatings (e.g., butyltin trichloride), polyurethane foams and many other consumer products. They also serve as catalysts for the manufacture of polyurethane and silicone elastomers; as antifouling agents for ships and boats; as biocides and fungicides applied to or incorporated in carpets and paints or applied to fruits and vegetables; as surface disinfectants for wood, paper, textiles, paints and some electrical equipment. Monomethyltin, dimethyltin, butyltin and octyltin are the most widely used of the organotins. | 7, 8 |

— *continued* —

| | | |
|---|---|---|
| **Perfluorinated Surfactants** | PFOA and PFOS are used as floor polishes, photographic film, denture cleaners, shampoos, herbicides, insecticides, and adhesives in a wide range of products, as well as surface stain-resistant coatings for fabrics, carpets, and paper and as a coating for cookware. PFOA is the best-known of the PFCs because it is used to make Teflon, Gore-tex, and other oil-, water- and stain-resistant materials used in many common items, including nonstick frying pans, utensils, stove hoods, stainproofed carpets, furniture, and clothes. PFOA is also used in fire-fighting foams, mining and oil well surfactants, and the manufacture of other fluoropolymers. PFOS is thought to be the main, final degradation product of many of the perfluorinated chemicals released into the environment. | 9, 10, 11 |

## Phthalates

Five of the seven phthalates selected for analysis were present at quantifiable concentrations in all of the dust samples, as shown in Table 2.

Di-(2-ethylhexyl) phthalate (DEHP) was present in all of the samples. With a mean concentration of 329 ppm, DEHP was predominant among not only the phthalates but also all 44 contaminants. No federal or state agencies have established regulatory limits on levels of DEHP in house dust. However, even the lowest DEHP concentration measured, 214 ppm, exceeds the acceptable limit of 44 ppm DEHP in residential soils established by the State of Connecticut.[12] On average, DEHP accounted for 78 percent of the total concentration of the target phthalates in the dust samples and 69 percent of the total concentration of the 44 contaminants.

While this study found mean concentrations of 1.4 ppm and 329 ppm for diethyl phthalate (DEP) and di(2-ethylhexyl) phthalate (DEHP) respectively, Rudel et al. (2003) reported higher mean concentrations for both, 8.5 ppm for DEP, and 506 ppm for DEHP, in their study of dust from some 120 homes on Cape Cod, Massachusetts.

As shown in Figure 2, butylbenzyl phthalate (BBP) (in the literature, this chemcial also appears as benxyl-butyl phthalate or BBzP) had the second highest concentration among the phthalates and accounted, on average, for 16 percent of the total mass of phthalates. With a mean concentration of 69 ppm, BBP also ranked second highest among all 44 contaminants.

Di-n-butyl phthalate was detected in all samples with an average concentration of 20.15 ppm.

Dimethyl phthalate (DMP) was found at a very low concentration in only one of the seven samples while di-n-propyl phthalate (DPP) was below detection levels in all samples.

Rudel et al. (2003) did not assay for dimethyl phthalate but did assay for the following phthalates that were not target analytes in the current study: dicyclohexyl phthalate, 2.98 ppm; di-n-hexyl phthalate, 2.6 ppm; and di-n-pentyl phthalate (<rl).

In this study, diisobutyl phthalate was found at an average concentration quite similar to that reported by Rudel et al. (2003). However, much higher concentrations of this phthalate were found in house dust from Belgium, Brazil and the U.K. This may reflect different patterns of phthalate use in products made and used in those countries.

Figure 3 shows the findings of the current study with respect to phthalates in comparison to those of other studies of house dust from Belgium;[13] Brazil;[14] Cape Cod, Massachusetts;[15] and the United Kingdom.[16]

The non-US studies also tested for some phthalates that were not selected for analysis in this study.

- In house dust from Belgium, Al Bitar (2004) found as follows: dicyclohexyl phthalate, 1.7 ppm; di-n-octyl phthalate, 55.7 ppm; di-isononyl phthalate, 162.9 ppm; and di-isodecyl phthalate, 66 ppm.
- In house dust from the U.K., Santillo et al. (2003) also reported di-isononylphthalate (48.5 ppm) and di-isodecylphthalate (20.8 ppm).
- In house dust from Brazil, Costner et al. (2004) reported dicyclohexyl phthalate (0.62 ppm), di-n-octyl phthalate (1.4 ppm), di-isononyl phthalate (71.2 ppm) and di-isodecyl phthalate (93 ppm).

### Phthalates—Production, Use, Occurrence and Effects

**"PVC is neither a biological nor technical nutrient. It is a toxic nightmare."**

*—Michael Braungart, Director, McDonough Braungart Design Chemistry and EPA Green Chemistry Award Winner*

Global production of phthalates is an estimated 3.5 million metric tons per year,[17] of which 80-90 percent is used as additives in flexible polyvinyl chloride (PVC) plastic—commonly known as vinyl.[18] Roughly 50 percent of the market share for phthalates is accounted for by di(2-ethylhexyl) phthalate (DEHP),[19] at least 95 percent of which is added to PVC to give it flexibility.[20] DEHP is present in PVC (vinyl) products such as wall coverings, tablecloths, floor tiles, furniture upholstery, shower curtains, garden hoses, swimming pool liners, rainwear, baby pants, dolls, some toys, shoes, automobile upholstery and tops, packaging film and sheets, sheathing for wire and cable, medical tubing, and blood storage bags.[21]

The remaining small share of phthalates not added to PVC is used in personal care products such as skin creams, hairsprays, lotions, nail polish, and fragrances, and in a variety of other products including adhesives, caulks, detergents, electrical capacitors, inks, solvents, lubricating oils, paints, and pharmaceuticals.[22,23,24]

While environmental releases of industrial chemicals are most commonly associated with their manufacture and disposal, it is estimated that more than 75 percent of phthalate releases to the environment occur during the use of products that contain phthalates.[25] Clausen et al. (2004) documented releases to air of DEHP from PVC flooring.[26] In studying phthalate emissions from PVC skirting, PVC flooring, and other materials, Afshari et al. (2004) concluded:[27]

> *"Plasticizers used in surface materials indoors can be detected in the indoor air and human exposure to plasticizers can be expected. This study shows that the concentration of phthalates in indoor air is independent of ventilation rates and the area of surface materials containing plasticizers, i.e. a small area of plasticizer containing products emits almost as much as a large area.* ***Therefore, if the surface materials contain plasticizers, it is impossible to avoid the phthalates in indoor air.*** *"* [emphasis added]

Phthalates are among the most ubiquitous synthetic chemicals in the environment[28] and are nearly always found at some concentration in virtually all people and wildlife.[29] Phthalates are found in the air and dust in homes and offices.[30]

**80–90 percent of all phthalates are used as additives in flexible polyvinyl chloride (PVC) plastic—or 'vinyl' products such as wall coverings, tablecloths, floor tiles, furniture upholstery, shower curtains, garden hoses, swimming pool liners, rainwear, baby pants, dolls, some toys, shoes, automobile upholstery and tops, packaging film and sheets, sheathing for wire and cable, medical tubing, and blood storage bags. They are now one of the most widespread sysnthetic chemicals**

### Occurrence of Phthalates and Their Metabolites in People

Exposure to phthalates has been associated with

- asthma and other respiratory problems, rhinitis and eczema in children;
- premature breast development in female children; and
- deteriorated semen quality, low sperm counts, and poor sperm morphology in men;

Findings of the studies described below can be summarized as follows:

- Phthalates and their metabolites are common, if not ubiquitous, contaminants in the bodies of U.S. men, women and children;
- A metabolite of diethyl phthalate, which is used in personal care products, is present at higher levels in the urine of U.S. adults who are 20 years old and older;
- Metabolites of DEHP, DBP and benzylbutyl phthalate occurred at higher concentrations in the urine of the youngest people tested, children aged 6 to 11 years;
- Studies in Germany found evidence that DEHP concentrations exceeded U.S. EPA's reference dose in almost one-third of the people tested and were about twice as high among very young children, ages 2 to 6 years, as among their parents and teachers;

- Among 30 pregnant women in New York City and 30 in Krakow, Poland, all had measurable concentrations of four phthalates—diethyl phthalate (DEP), dibutyl phthalate (DBP), diethylhexyl phthalate (DEHP), and butyl benzyl phthalate (BBzP)—in their personal air and metabolites of these same phthalates in their urine;
- A study in Italy found DEHP and/or its metabolite, monoethylhexyl phthalate (MEHP) in more than 80 percent of the cord blood samples from 84 births. Pregnancy durations tended to be shorter for those in which MEHP was present in the cord blood;
- Several phthalate metabolites were detected in pooled breastmilk samples from the U.S.
- Exposure to phthalates has been associated with
  - — asthma and other respiratory problems, rhinitis and eczema in children;
  - — premature breast development in female children; and
  - — deteriorated semen quality, low sperm counts, and poor sperm morphology in men;

In a study of urine phthalate levels in 2,541 U.S. residents, the Centers for Disease Control (CDC) found evidence of widespread exposure, with higher concentrations in women. Concentrations of monoethyl phthalate, a metabolite of diethyl phthalate, were almost two times higher in the urine of people aged 20 years and older than in children aged 6 to 11 years. Diethyl phthalate is used in products such as fragrances, soaps, and hand lotions. The highest levels of metabolites for DEHP, DBP and benzylbutyl phthalate were found in the urine of the youngest people tested, children aged 6 to 11 years. In fact, the concentrations of monobenzyl phthalate, a metabolite of benzylbutyl phthalate, were more than three times higher in the children's urine. Benzylbutyl phthalate is used in products such as adhesives, sealants, and car care products.[31]

In Germany, Koch et al. (2003) concluded that the general population is exposed to DEHP to a much higher extent than previously believed, noting that 31 percent of their subjects had DEHP values that exceeded the U.S. EPA reference dose. They concluded as follows: [32]

> *"This is of greatest importance for public health since DEHP is not only the most important phthalate with respect to its production, use and occurrence and omnipresence but also the phthalate with the greatest endocrine disrupting potency."*

In a later analysis of DEHP metabolites in urine, Koch et al. (2004) estimated the internal exposure of nursery school children, aged 2–6 years, to DEHP to be about twice that of their parents and teachers.[33]

In a study of pregnant women – 30 in New York City and 30 in Krakow, Poland, Adibi et al. (2003) measured concentrations of four phthalates in personal air and the metabolites of these same phthalates in urine. These phthalates—diethyl phthalate (DEP), dibutyl phthalate (DBP), diethylhexyl phthalate (DEHP), and butyl benzyl phthalate (BBzP)—were present in 100 percent of the air samples and their metabolites in 100 percent of urine samples. Their results demonstrate considerable phthalate exposures during pregnancy among these women and indicate that inhalation is an important route of exposure.[34]

**Animal studies have found that phthalates pass from the mother through the placenta to the fetus and through breastmilk to the newborn.**

A study by Latini et al. (2003) found detectable DEHP and/or its metabolite monoethylhexyl phthalate (MEHP) in the cord blood of 88% of 84 newborns in Italy. MEHP-positive newborns showed a significantly lower gestational age compared with MEHP-negative newborns. Their findings confirm that human exposure to DEHP can begin in utero and suggest that phthalate exposure is significantly associated with a shorter pregnancy duration.[35] Calafat et al. (2004) detected several phthalate metabolites in three pooled human breast milk samples in the U.S., suggesting that phthalates can be incorporated into breast milk and transferred to the nursing infant. Three of the phthalate metabolites and three oxidative metabolites were detected in all three pooled samples.[36] Animal studies have also found that phthalates pass from the mother through the placenta to the fetus and through breastmilk to the newborn.[37,38,39]

### Effects of Exposure to Phthalates and Their Metabolites in People

As illustrated by the studies discussed below, exposure to phthalates and their metabolites have been associated with a broad range of health effects:

- asthma and other respiratory problems, rhinitis and eczema in children;
- premature breast development in female children; and

- deteriorated semen quality, low sperm counts, and poor sperm morphology in men.

**Children exposed to household dust with the greatest concentrations of di(2-ethylhexyl) phthalate (DEHP) were 2.9 times as likely to have asthma as were children exposed to the lowest concentrations of that phthalate.**

According to Jaakkola et al. (1999) and Øie et al (1997), the presence of plasticizers in surface materials indoors can increase the risk of bronchial obstructions, asthma, and perhaps the susceptibility to respiratory infections.[40,41] The later noted that indoor inhalation of DEHP-adsorbed particulate matter could be as or more important than inhalation of vapor phase DEHP.[42] Bornehag et al. (2004) found that children exposed to household dust with the greatest concentrations of di(2-ethylhexyl) phthalate (DEHP) were 2.9 times as likely to have asthma as were children exposed to the lowest concentrations of that phthalate. Similarly, children in homes with the greatest concentrations of butyl benzyl phthalate were 3.0 and 2.6 times as likely as the other children to have rhinitis and eczema, respectively.[43]

**The concentration of phthalate esters was significantly higher in infertile men compared with controls and they may be instrumental in the deterioration of semen quality in infertile men.**

A study on premature breast development (thelarche) in female children aged 6 months to 8 years found phthalate esters in 68% of serum samples from the thelarche patients. The phthalate esters with the most common commercial uses, DEHP and DBP, were detected in the highest concentrations. For those samples with high concentrations of DEHP, one of the major DEHP metabolites, mono(2ethylhexyl)phthalate (MEHP), was also detected. DEHP was detected in only 14% of the control samples in lower concentrations. A more sensitive analysis of eight thelarche samples allowed detection of a further two phthalates, DMP and DEP, in two and three samples, respectively.[44]

**A study on premature breast development in female children aged 6 months to 8 years found phthalate esters in 68% of serum samples from the patients.**

Duty et al. (2002) explored whether general levels of phthalates in the U.S. population were associated with altered semen quality and found suggestive evidence of associations between high mono-benzyl phthalate (MBzP) levels and low sperm counts, and between high mono-methy phthalate (MMP) and poor sperm morphology. Mono-n-butyl phthalate (MBP), MBzP and MMP were associated with altered semen quality.[45] In another related study, Duty et al. (2003) found that urinary monoethyl phthalate (MEP), at environmental levels, is associated with increased DNA damage in sperm.[46] Rozati et al. (2002) found that the concentration of phthalate esters was significantly higher in infertile men compared with controls and concluded that they may be instrumental in the deterioration of semen quality in infertile men without an obvious mechanism of action.[47]

### Effects of Exposure to Phthalates and Their Metabolites in Laboratory Animals

Some phthalates and their metabolic products act functionally as anti-androgens during the prenatal period.[48, 49, 50] Developmental effects in males include reduction in androgen-dependent tissues—in the reproductive organs such as seminal vesicles, epididymus, prostate, and anogenital distance.[51,52,53,54] Furthermore, exposure of rats prenatally and during suckling to DEHP and DBP has produced irreversible testicular damage at dose levels that caused only minimal effects in adult animals.[55,56,57,58]

Numerous studies have shown that some phthalates are toxic to embryos and cause developmental malformations in the offspring of exposed rodents. DEHP has these effects in mice. It is also toxic to rat embryos at dose levels that are not toxic to the mother.[59,60] DBP generally is toxic to the fetus of rats and mice in the absence of maternal toxicity, and it has harmful developmental effects only at doses high enough to be toxic to the mother.[61] In very recent studies, rats exposed to DBP during pregnancy and shortly after birth, a number of effects were seen in the male offspring including: decreased anogenital distance, absent or underdeveloped epididymis and seminal vesicles, cleft penis (hypospadias), decreased reproductive organ weights, and widespread germ cell loss in the testis. In contrast, vaginal opening, age at first estrus, and estrous cyclicity were not affected in the female offspring indicating that DBP does not mimic estrogenic but rather acts as an anti-androgen.[62]

Exposure during gestation and through breastmilk to large doses of dibutyl phthalate (DBP) and its

metabolite, monobutyl phthalate (MBP), causes male reproductive tract malformations in rats.[63,64] DBP reduces the production of testosterone by the fetal testis through an anti-androgenic mechanism.[65] Exposure to high doses of DBP results in spontaneous abortion of rat pups, demasculinization of baby male rats, and decreased testicle size in rats, mice, ferrets, and guinea pigs.[66,67]

In male rats, high doses of DEHP have resulted in decreased testicle weights and smaller tubules.[68] The DEHP metabolite, mono-2-ethylhexyl phthalate (MEHP) may be the active agent.[69,70]

Although fewer studies have been carried out on female animals, existing studies suggest that long-term exposures of adult female rats result in adverse effects, including effects on the ovulation cycles and cysts of the ovaries.[71] A recent study suggests that DEHP, through MEHP, suppresses the production of the hormone estradiol in the ovary, interfering with egg production.[72]

Several phthalates can also be carcinogenic in rodents.[73,74,75] DEHP, DBP, and their monoester metabolites appear to have the greatest potential toxicity. DEHP is a peroxisome proliferator hepatocarcinogen in rodents,[76] but the relevance of carcinogenicity by this mechanism in humans is being debated.[77,78,79]

## Alkylphenols

Six of the seven alkylphenols and alkylphenol ethoxylates selected for analysis were detected in all samples. These chemicals were the second most abundant group of contaminants.

Nonylphenol diethoxylate was present at the highest concentration in six of seven samples. The combined concentrations of 4-nonylphenol, nonylphenol monoethoxylate and nonylphenol diethoxylate accounted for 75–95 percent of the total concentrations of this group of contaminants. 4-Nonylphenol is widely known as an endocrine disruptor and is one of the major metabolites of the nonylphenol ethoxylate surfactants.

Octylphenyl monoethoxylate and octylphenol diethoxylate were detected in all samples, but 4-octylphenol was not quantifiable in any. As shown in Figure 4, Rudel et al. (2003) reported a low concentration of 4-octylphenol in their Cape Cod study. Similarly, Santillo et al. (2003) found 4-octylphenol at a low concentration, 0.3 ppm, in only one sample in their study of house dust in the U.K.

While bisphenol-A was not selected as an analyte in this study, Al Bitar (2004) detected this compound at 2.2 ppm in Belgian house dust.

### Alkylphenols and Alkylphenol Ethoxylates—Production, Use, Occurrence and Effects

Alkylphenols (APs) are used primarily as raw materials for the manufacture of alkylphenol ethoxylates (APEs). They are also used in the preparation of phenolic resins, polymers, heat stabilizers, antioxidants, and curing agents. Almost half of global APE production takes place in the U.S. [80] With a total U.S. production of more than 500 million pounds per year, nonylphenol ethoxylates account for about 80 percent of total APE use. Most of the remaining production consists of octylphenol ethoxylates.[81]

APs are also formed as degradation products of APEs. During wastewater treatment, APEs are degraded to form APs. In the liver, enzymes break down the toxic APEs to form APs.[82]

The most widely recognized hazard associated with alkylphenols is their ability to mimic natural estrogen hormones. This can lead to altered sexual development in some organisms.

The major uses of APEs are: industrial applications, 55 percent; industrial and institutional cleaning products, 30 percent; household cleaning products, 15 percent; and other uses, less than 1 percent. [83] APEs are used as nonionic surfactants in detergents; applied as dispersing agents in paper and pulp production and de-inking agents in paper recycling; emulsifying agents in latex paints, pesticide and herbicide formulations, and fiberglass and polystyrene products; as wetters in peats; as additives in cosmetics and in polyvinyl chloride used for food packaging; flotation agents, industrial cleaners, cold cleaners for cars, and in the textile industry; in the form of tris(nonylphenol)phosphates as antioxidants in plastics. Nonylphenol is the active ingredient in spermicides. Nonylphenol or a derivative is also apparently used in food wrapping films, food-contacting plastics, and some toys because the chemical has been found to leach from these materials and products.[84,85,86,87,88,89,90]

Nonylphenol is regarded as a ubiquitous environmental contaminant.[91,92]

### Occurrence of Alkylphenols and Alkylphenol Ethoxylates in People

There is very little information on the occurrence of alkylphenols and alkylphenol ethoxylates in people. Thus far, the Centers for Disease Control and Prevention (CDC) has chosen not to included alkylphenols and alkylphenol ethoxylates in their biomonitoring program, as reflected in the agency's National Report

on Human Exposure to Environmental Chemicals of 2001 and the succeeding 2003 report.[93,94] This will also be the case in the CDC's upcoming third report. [95]

In a very recent study, Calafat et al. (2005) analyzed archived urine samples from a reference population of 394 people in the U.S. and found 4-nonylphenol in 51 percent of the samples and bisphenol A in 95 percent of the samples at concentrations of 0.1 parts per billion (ppb) or more.[96] Scientists in Japan found quantifiable concentrations of 4-nonylphenol and 4-*tert*-octylphenol in human plasma[97] but not in urine.[98] Nonylphenol has also been detected in umbilical cords in Japan,[99] confirming that this chemical is passed from the mother to the developing fetus through the placenta. A very recent study in Germany has found nonylphenol in breastmilk[100] confirming that this chemical can also pass from the mother to her nursing infant.

### Health Effects of Exposure to Alkylphenols and Alkylphenol Ethoxylates in People

No studies were found of the health impacts in people. However, the most widely recognized hazard associated with APs, both nonylphenol and octylphenol, is their ability to mimic natural estrogen hormones. This can lead to altered sexual development and impact reproduction in some organisms.

Hazards to human health are not yet well defined, although a number of studies with animals, such as those described below, serve to highlight concerns that are very relevant to public health.

### Effects of Exposure to Alkylphenols and Alkylphenol Ethoxylates in Laboratory Animals

Most laboratory studies of the effects of alkylphenols and alkylphenol ethoxylates have explored their potential roles as endocrine disruptors and associated reproductive and developmental effects. For example, the ability of alkylphenols to mimic estrogen has been known for years.[101] As such, alkylphenols have been shown to reduce testicular function in rats.[102] Of several alkylphenols and alkylphenol ethoxylates tested, tertiary octylphenol showed the highest estrogenic activity.[103]

**The estrogenicity of alkylphenols has been known for years and, as estrogenic compounds, alkylphenols have been shown to reduce testicular function in rats.**

Both nonylphenol and octylphenol show estrogenic and anti-androgenic activities.[104,105] Although nonylphenol has the lower binding affinity to estrogen receptors, it exerts more estrogenic potency because serum has a more protective effect against octylphenol. This is an important finding to consider when comparing levels in the diet and actual effects of these and other estrogen mimics.[106,107]

The reproductive and developmental effects of alkylphenols are illustrated in two low-dose developmental studies in rodents:

- Sharpe et al. (1995) showed that exposure before and after birth to octylphenols caused a reproducible and consistent decrease in testicular size and daily sperm production in rats during a relatively short period;[108] and
- A multigenerational mouse study demonstrated that nonylphenol affected both the parents and offspring, most notably by diminishing the size of male reproductive organs, reducing sperm quality and decreasing fertility.[109]

Preliminary studies suggest that nonylphenol may also disrupt the human immune system. For example, in laboratory studies, nonylphenol inhibits the production of a chemical that attracts and activates an important group of white blood cells.[110] In mice, nonylphenol also increases the production of a specific chemical messenger in T lymphocytes and increases levels of certain antibodies. This suggests that nonylphenol may enhance allergic responses because both the chemical messenger and the antibody are key factors in allergies.[111]

Dietary exposure of female rats to octylphenol throughout pregnancy and lactation can interfere with sexual development of male offspring by changing sexual behavior and decreasing adult body weight, testis weight, and the average diameter of the seminiferous tubules.[112] Similar evidence has come from studies with newborn rats,[113,114,115,116,117,118,119] and from studies where the exposure has been carried out for longer periods into adulthood.[120], [121]

The estrogenic potency of octylphenol is evidenced by persistent estrus in females exposed in adulthood to octylphenol[122] and caused the production of an hormone related to milk production (prolactin) both in the adult males[123] and after newborn exposure[124] as well as in several other study models.[125,126,127,128,129,130]

In a three-generation study with pigs, octylphenol exposure extended pregnancy length, induced basel cell proliferation in the cervical epithelium of the

parental generation, accelerated onset of puberty, and reduced litter size in the first generation females. In the first generation offspring of female pigs treated with the low dosage of octylphenol, onset of puberty was accelerated. When first generation young female and male pigs originating from sows treated with high dosages of octylphenol were bred, the litter size was reduced. When several alkylphenols and alkylphenol ethoxylates were tested, tertiary octylphenol tended to exhibit the highest estrogenic activity.[131]

## Pesticides

**"We have now acquired a fateful power to alter and destroy nature. But man is a part of nature, and his war against nature is inevitably a war against himself."**

*—Rachel Carson, author of* Silent Spring, *who first raised awareness of the toxicity and persistence of DDT pesticides, quoted on CBS News, 1964*

This group of chemicals included eleven pesticides and one synergist (piperonyl butoxide). Together, these chemicals were the third most abundant of the six groups of contaminants. Each of the dust samples contained quantifiable concentrations of five compounds: 4,4'-DDT, pentachlorophenol, cis-permethrin, trans-permethrin, and piperonyl butoxide. Only one of the samples contained quantifiable concentrations of the two chlordane isomers and dieldrin. Chlorpyrifos also occurred in only one sample. Three of the target pesticides—diazinon, dicofol and pentachloronitrobenzene—were not present at quantifiable concentrations in any of the samples.

Combining the concentrations of both permethrin isomers, this pesticide had the highest mean concentration (9.7 ppm) of all targeted pesticides in six samples. Pentachlorophenol had the highest concentration, 7.3 ppm, in the seventh sample, and the second highest mean concentration,1.246 ppm, in the six sample dominated by permethrin.

Piperonyl butoxide had the third highest mean concentration in this contaminant group, 0.69 ppm. This chemical is used as a synergist in formulations of permethrin, other pyrethrins and pyrethroids to increase the effectiveness of the insecticides.[132] As such it is sometimes relied upon as an indicator of the presence of permethrin and other pyrethroids.[133]

As shown in Figure 5, Rudel et al. (2003) also reported major contributions by permethrin and piperonyl butoxide.[134] However, the latter chemical was present at a far higher concentration, 15.8 ppm, in the dust from Cape Cod than the mean concentration found in this study. It is possible that because our samples were composited (all 10 of the samples put together), the variations was lessened.

### Pesticides: Permethrins, Piperonyl butoxide, Pentachlorophenol and DDT

Permethrin, a synthetic pyrethroid, is used to kill pest insects in agriculture, home pest control, forestry, and in public health programs, including head lice control. It was first marketed in 1973. Worldwide, the dominant use of permethrin is on cotton, accounting for about 60 percent (by weight) of the permethrin used.[135] In the U.S., almost 70 percent of the permethrin used in agriculture is used on corn, wheat, and alfalfa.[136] It is widely used in U.S. homes, and yards and gardens. Permethrin, like all synthetic pyrethroids, kills insects by strongly exciting their nervous systems.

Because of its ubiquitous use, the Food and Drug Administration's monitoring program routinely finds permethrin on food. In 2001, it was the 8th most commonly detected pesticide[137] with DDT being number 1.

### Human and animal health effects

Experiments with laboratory animals indicate that the immune system "appears to be a sensitive target for permethrin activity." Ingestion of permethrin reduces the ability of immune system cells called T-lymphocytes to recognize and respond to foreign proteins. Doses equivalent to 1/100 of the LD50 , inhibited T-lymphocytes over 40 percent. Permethrin ingestion also reduced the activity of a second type of immune system cell, natural killer cells, by about 40 percent.[138] In tests using mouse cell cultures, permethrin had similar effects on the immune system via the inhibition of two kinds of lymphocytes.[139] Researchers concluded that "the immune system is exquisitely sensitive…at exposure levels that cause no overt toxicity."

Based on tests with laboratory animals, it appears children may be more sensitive to permethrin than adults. Permethrin is almost 5 times more acutely toxic to 8-day-old rats than it is to adult rats.[140]

**Experiments with laboratory animals indicate that the immune system appears to be a sensitive target for permethrin activity. Permethrin also affects both male and female reproductive systems.**

Permethrin affects both male and female reproductive systems. It binds to receptors for androgen, a male sex hormone, in skin cells from human males, causing

researchers to "advise protection from any form of contact or ingestion of the pyrethroids."[141] Permethrin was mutagenic in three tests with human cell cultures, one with hamster cells, and one with fruit fly larvae. In cultures of human lymphocytes (white blood cells), permethrin exposure caused an increase in chromosome aberrations, chromosome fragments,[142] and DNA lesions[143] often linked to cancer development.

According to the EPA, permethrin is a possible human carcinogen.[144] The EPA found that permethrin increased the frequency of lung tumors in female mice, and increased the frequency of liver tumors in male and female mice.[145] The World Health Organization reports that permethrin increased the frequency of lung tumors in females in two out of the three mouse studies it reviewed. Lung tumors increased with increasing permethrin exposure in the third study, but the increase was not statistically significant.[146]

**Piperonyl butoxide** is used in formulations of permethrin, other pyrethrins and pyrethroids as a synergist to increase the effectiveness of the insecticides As such it is sometimes relied upon as an indicator of the presence of permethrin and other pyrethroids. It does not by itself have pesticidal properties. However, when added to insecticide mixtures, their potency is increased considerably. Pyrethrin with piperonyl butoxide kills parasites and their eggs. Together they are used to treat scabies and lice infestations of the head, body, and pubic area. Pyrethrin with piperonyl butoxide does not prevent these infestations.

Piperonyl butoxide is a potent inhibitor of a family of enzymes (Cytochrome P450) that helps to break down many pesticides and is considered the principal detoxification pathway for these chemicals. Hindering this detoxification process allows higher concentrations of the active insecticide to remain within the target animal for a longer period.

It is still being debated whether the substance is oncogenic, mutagenic, or teratogenic in humans. The EPA has classified piperonyl butoxide as a possible human carcinogen.[147]

### Pentachlorophenol

In the U.S., most exposure to pentachlorophenol (PCP) comes from PCP its past use on treated wood and soil. From 1987 to 1993, the EPA recorded releases of PCP to land and water, mostly from treated wood and military munitions factories, totaling nearly 100,000 pounds.[148]

PCP uses have been limited since 1984 to use by certified applicators for certain purposes such as a preservative on wooden utility poles, railroad ties and wharf pilings.[149] It is also still used in California, mostly on almonds and structural pest control.[150]

### Health Effects

Technical grade PCP is frequently contaminated with dioxins and hexachlorobenzene[151] making it difficult to differentiate between the health effects that are due to pentachlorophenol itself and those caused by its common contaminants.

It is unclear whether exposure of the developing fetus to pentachlorophenol will result in birth defects or other developmental effects in people, but laboratory animals exposed to high levels during development experience health effects including low body weight, decreased growth and skeletal problems.[152] PCP is a suspected endocrine disruptor, interfering with the natural function of estrogen, androgen and thyroid hormones.

The EPA has determined that pentachlorophenol is a probable human carcinogen and the International Agency for Cancer Research classifies it as possibly carcinogenic to humans.[153,154]

### DDT

DDT is no longer registered for use in the United States, although it is still used in other (primarily tropical) countries for malaria control. It is in the EPA Toxicity Class II, moderately toxic. DDT was banned from use in the United States in 1972, and remains banned barring public health emergency (e.g., outbreak of malaria).[155] The early popularity of DDT, a member of the chlorinated hydrocarbon group, was due to its reasonable cost, effectiveness, persistence, and versatility. During the 30 years prior to its cancellation, a total of approximately 1,350,000,000 pounds of DDT was used domestically.

After 1959, DDT usage in the U.S. declined greatly, dropping from a peak of approximately 80 million pounds in that year to just under 12 million pounds in the early 1970s. Of the quantity of the pesticide used in 1970-72, over 80 percent was applied to cotton crops, with the remainder being used predominantly on peanut and soybean crops. The decline in DDT usage was the result of increased insect resistance; the development of more effective alternative pesticides; growing public concern over adverse environmental side effects; and increasing government restrictions on DDT use.[156]

**Occurrence and Effects**

Even though current dietary levels are quite low, past and current exposures may result in measurable body burdens due to its persistence in the body. There is evidence that DDT causes reproductive effects in test animals. DDT is not metabolized very rapidly by animals; instead, it is deposited and stored in the fatty tissues. The biological half-life of DDT is about eight years; that is, it takes about eight years for an animal to metabolize half of the amount it assimilates. If ingestion continues at a steady rate, DDT builds up within the animal over time. In a recent body burden study by the Centers for Disease Control and Prevention (CDC), scientists found DDT in blood of 99% of those sampled—the highest incidence of any pesticide sampled.[157]

Research by Cohn *et al.* reveals an unexpected association between DDT and delays in pregnancy in the daughters of exposed women, 30 years after birth. This is the first scientific report ever of a link between DDT and reproductive outcome in women exposed to the contaminant in the womb. Their statistical assessment indicates that the association is unlikely to be a result of chance.[158]

We are still learning the consequences of past DDT use. Strong hints that there might be more yet to learn surfaced recently, when Centers for Disease Control scientists reported a striking relation between DDT and the likelihood of preterm birth.[159] Longnecker *et al.* demonstrate a powerful association between levels of the breakdown product of DDT, DDE, in mothers' serum and the likelihood of premature birth. The higher the contamination level, the more likely was preterm birth. They also show that contamination is linked to the baby's size, with babies more likely to be small for their gestational age when born to mothers with higher DDE levels.

More information on DDT & malaria is available at: *http://www.panna.org/campaigns/docsPops/docsPops_030317.dv.html*

## Polybrominated diphenyl ethers

**"This stuff is everywhere"**

*— Dr. Jake Ryan, Health Canada*

PBDEs were fourth in abundance among the contaminant groups. Three of the seven PBDEs that were selected for analysis—BDE 47, BDE 99, and BDE 209—were present at quantifiable concentrations in all dust samples. BDE 100 (penta) reached quantifiable concentrations in four samples, and BDE 153 and BDE 154 (hexa), in two samples. No quantifiable levels of BDE 183 were found in any of the samples.

As shown in Figure 7, decabrominated diphenyl ether, BDE 209, predominated in 4 of the 7 samples and, as shown in Figure 6, had the highest mean concentration, followed by BDE 47 and BDE 99. On average, these three PBDEs accounted for 95 percent of the total concentration of this contaminant group.

As shown in Figure 7, a higher mean concentration of total PBDEs, 9.524 ppm, was found in this study than by Stapleton et al. (2005)[160] in their study of house dust from the Washington, DC area, 5.65 ppm. However, BDE 209 was the largest contributor in both cases. The apparent lack of similarity to the findings by Rudel et al. (2003) are due primarily to differences in the PBDEs selected for analysis, e.g., the Rudel study did not test for BDE 154 , BDE 183, and BDE 209. Also, while total PBDEs in UK and Belgian dust fell within the range of those measured in this study, the PBDE "profile" was very different in that decabromodiphenyl ether (BDE 209) accounted for a far larger share of total PBDE concentrations in the European samples. This may be attributed to mandated and voluntary actions in the European Union to phase out the use of those PBDEs that were thought to pose greater health threats.

**Polybrominated Diphenyl Ethers—Production, Use, Occurrence and Effects**

**"The accumulation and ongoing increase in the levels of PBDEs calls for immediate measures to stop the environmental pollution and human exposure to PBDEs."**

*—Noren and Meironyte, 2000.*[161]

More than 70 brominated chemicals or groups of chemicals are used as flame retardants in plastics, textiles and other materials. Polybrominated diphenyl ethers (PBDEs) are one of the three groups that dominate the market for flame retardants.[162] In 1999, total global production of the three major commercial PBDE products was 67,125 metric tons: deca-BDE, 82 percent; penta-BDE, 13 percent; and octa-BDE, 6 percent; and penta-BDE, 13 percent. Ninety-eight percent of penta-BDE is used in North America.[163]

PBDEs are applied to or incorporated into many common household products, such as furniture, carpeting, mattresses, televisions, coffee makers, and hair dryers.[164] Decabromodiphenyl ether (Deca-BDE or BDE 209) is most commonly used in plastics and textiles, in electrical components, and in styrene rubbers

used in carpet backing and furniture.[165] Sunlight and UV light can degrade BDE 209 to form less brominated BDEs, such as the pentabromodiphenyl ethers (penta-BDEs).[166]

PBDEs have been found in air, water, fish, birds, marine mammals, and humans, and in many cases, concentrations are increasing over time.[167] Diet is regarded as the most likely route of PBDE exposure for the general population.[168] However, air inside homes and offices can carry PBDE concentrations that are estimated to be almost ten times higher than levels in the air outside the buildings.[169] Moreover, house dust has been identified as an important pathway of PBDE exposure for young children.[170] Despite the ubiquity of PBDEs, information on their toxicology is limited.[171]

**Occurrence of Polybrominated Diphenyl Ethers in People**

The Centers for Disease Control and Prevention (CDC) does not monitor PBDEs in the U.S. population. No data on PBDE levels are given in CDC's National Report on Human Exposure to Environmental Chemicals of 2001 or the succeeding 2003 report,[172,173] and PBDE data will not be included the CDC's upcoming third report.[174]

Many other studies have found PDBEs in human breast milk,[175,176, 177,178,179,180] blood,[181,182] umbilical cord blood, [183], [184] and adipose tissue.[185],[186],[187],[188] Interestingly, the German national biomonitoring program found that men had PBDE levels from 20 to 75 percent higher than women, depending on the sampling year.[189]

PBDE concentrations in human serum and breast milk have been increasing at exponential rates for more than two decades.[190,191,192,193] During the period of 1972 to 1997, levels of organochlorine contaminants in the breast milk of Swedish mothers were falling while PBDE concentrations were rising.[194] However, a recent study indicates that PBDEs in Swedish breast milk began to decrease in 1997, possibly due to a voluntary phase-out of penta-BDE.[195]

Studies of breast milk in the U.S. have found PBDE concentrations 10 to more than 100 times higher than those in Europe.[196,197,198] Moreover, contrary to claims by PBDE producers that BDE 209 (deca) is neither mobile nor bioavailable, three recent studies have identified BDE 209 in 20 to 80 percent of breast milk samples.[199,200,201] BDE 209 has also been identified as the dominant PBDE in several U.S. food groups.[202]

PBDE levels in mother's milk and blood were shown to have a strong correlation with PBDE levels in fetal blood in a study carried out in Indianapolis, Indiana, where maternal and fetal blood levels were 20–106 times higher [203] than the levels reported previously in Swedish mothers and infants [204] and 20 times higher than Norwegian blood samples.[205] Twenty-three women from the San Francisco Bay Area were found to have PBDE concentrations in adipose tissue from their breasts higher than have been reported to date in human tissues. [206]

The tetrabrominated PBDE congener, BDE-47, is the most abundant PBDE congener in all human samples tested, making up 53–65% of total PBDEs detected. The other major congeners include, BDE-99, 100, 153, and 154.[207,208,209]

**Effects of Polybrominated Diphenyl Ethers in Human**

Several relatively dated studies cited by Darnerud (2003) found no effects: no skin sensitization was observed among human volunteers exposed to decaBDE products and no effects were noted in four epidemiologic studies of workers from workplaces where flame retardants were used.[210]

However, in a U.S. study, workers exposed to PBBs and PBDEs during manufacture had higher-than-normal rates of primary hypothyroidism and significant reductions in conducting velocities in sensory and motor neurons were reported. However no conclusions were drawn about the causative role of PBBs and/or PBDEs.[211]

A study of fish consumers in the Baltic region found that higher levels of BDE 47 were weakly associated with lower plasma levels of the thyroid hormone, thyrotropin.[212] The researchers noted that the weak positive correlation could have been due to chance. However the findings of the U.S. study suggest that the correlation may not have been a chance occurrence.

**Effects of Polybrominated Diphenyl Ethers in Other Species**

Laboratory studies indicate that some PBDEs may trigger dioxin-like responses, although at concentrations that are far higher than those required for the most potent dioxin congener.[213] Similarities have also been drawn to the PCBs, with PBDEs associated with birth defects, liver and kidney damage, thyroid imbalances and neurological damage to animals and humans.

In studies with laboratory animals, mice and rats exposed to one or more PBDEs have shown a wide variety of effects including evidence of endocrine disruption,[214,215,216,217,218] reproductive/developmental

toxicity including neurotoxicity,[219,220,221,222] and cancer.[223] Mice exposed to some PBDEs three or ten days after birth exhibited changes in spontaneous behavior (locomotion, rearing, and total activity) when two-months old, while post-natal exposure to other PBDEs resulted in impaired learning and memory.[224,225,226] Such effects are similar to those seen after exposure to DDT or PCBs.[227]

Generally, the pentaBDEs seem to cause adverse effects at lower doses while much higher doses of decaBDE are required to produce effects. The more critical effects of pentaBDEs are on neurobehavioural development and thyroid hormone levels. Both rats and rabbits exhibited fetal toxicity/teratogenicity following exposure to octaBDEs and changes in thyroid, liver and kidney morphology after exposure to decaBDE. At very high levels, decaBDE was carcinogenic in animals. However, IARC (1990) considers decaBDE not to be classifiable with regard to carcinogenicity in humans.[228] Other PBDEs have shown genotoxic effects in mammalian cell lines, which suggests that they may be cancer promoters. [229]

After neonatal exposure to PBDE 99 and PBDE 153, adult mice also exhibited learning and memory effects. The induction of permanent aberration in spontaneous behaviour was induced during limited period of the neonatal brain development.[230,231] The altered spontaneous behaviour also worsened with age.[232,233] Developmental neurotoxic effects after neonatal exposure to PBDE 209 are suggested to be caused by a metabolite.

**Common metabolites of the PBDEs are reported to compete strongly with the thyroid hormone, thyroxin raising the potential for a broad range of effects on growth and development, including permanent neurobehavioral impacts, that are comparable to the thyroid disrupting effects of PCBs.**

PBDEs are thought to have low acute toxicity. However chronic exposure to low levels during gestation and via lactation can cause irreversible changes in development. Studies with laboratory animals indicate that PBDEs are transferred from the mother to the fetus via the placenta and from the mother to the nursing offspring through breast milk.[234,235,236,237,238,239,240] Following gestational and/or lactional exposure, offspring show signs of thyroid disruption and developmental neurotoxicity,[241,242,243,244,245,246] as well as other endocrine and genetic effects.[247,248,249,250,251,252,253]

**Laboratory animals exposed to PBDEs during the perinatal period exhibited exhibited behavioral changes when they reached adulthood. These changes included marked hyperactivity and learning and memory deficits.**

Chronic exposure, particularly during gestation, can interfere with brain and skeletal development in rats [254] and lead to permanent neurological effects.[255] Common metabolites of PBDEs are reported to compete strongly with the thyroid hormone, thyroxin, raising the potential for a broad range of effects on growth and development, including permanent neurobehavioral impacts, comparable to the thyroid disrupting effects of PCBs. (Meerts et al. 1998, 2000, 2001). Other researchers have also raised the possibility that, considering the structural similarity of PBDEs with PCBs and the known health effects of PCBs, the two groups of chemicals could work through the same mechanism to cause developmental neurotoxicity.[256]

When mice are exposed shortly after birth, PBDEs, including BDE 209, have been shown to distribute throughout the body and concentrate in the brain. They induce developmental neurotoxic effects in adult mice that worsen with age and lead to abnormal behaviour. [257] For example, laboratory animals exposed to PBDEs during the perinatal period exhibited exhibited behavioral changes when they reached adulthood. These changes included marked hyperactivity[258,259] and learning and memory deficits.[260]

A range of PBDEs show estrogenicity in human cells lines and bind to the estrogen receptors.[261] PBDEs are also metabolized to form hydroxylated-PBDEs that are even more potent estrogen mimics.[262]

## Organotins

As far as we could establish, this is the first time that organotins were analyzed in American household dust. Of the seven organotins that were tested, four were quantified in all samples: monobutyltin, dibutyltin, tributyltin, and di-n-octyltin. The other three—tetrabutyltin, tricyclohexyltin and triphenyltin—were below reportable limits in all samples. The total concentration of all selected organotins ranged from 388 to 911 parts per billion (ppb). Monobutyltin was the predominant organotin in three samples and dibutyltin dominated in three samples. In the remaining sample, the concentrations of these two compounds were almost the same.

As shown in Figure 8, total organotin concentrations

in this study are in the same general range as the mean value reported by Costner et al. (2004) in dust from Brazil and by Fromme et al. (2005)[263] in dust from German homes. However, they are markedly lower than the total mean concentrations reported by Al Bitar (2004) for Belgian dust samples.

Monooctyltin was not selected as an analyte in this study. However, Fromme et al. (2005) found this compound at a mean concentration of 10 ppb in house dust in Germany and Costner et al. (2004) reported a mean of 62.5 ppb in house dust in Brazil.

### Organotins—Production, Use, Occurrence and Effects

> **"At pharmacologic levels butyltins might contribute to the onset of developmental disorders of the male reproductive system."**
>
> *— Doering et al. (2002)* [264]

No information on current production rates for organotins was found. However, major use of organotins began some 40 years ago in parallel with mass production of PVC plastic (vinyl).[265] Between 1955 and 1992, organotin production increased by a factor of ten[266] and, reached about 40,000 metric tons per year in 1996.[267] Mono- and dialkyltins account for 81 percent of total organotin use: 76 percent used as heat and light stabilizers for PVC and 5 percent as catalysts for polyurethane and silicone elastomers. The remaining total organotin use consists mainly of tributyl, triphenyl- and tricyclohexyltin, about 10 percent of which is used as antifouling biocides and 8 percent as pesticides.[268,269]

Organotins are found in PVC water pipes, PVC food packing materials (e.g., dioctyltin), glass coatings (e.g., butyltin trichloride), polyurethane foams.[270] Other uses, mainly of butyltin, include rigid PVC profiles and sidings. Venetian blinds, rain gutters, window profiles and, in particular in the U.S., building sidings.[271] Organotins also occur in textile products that contain polymer parts, such as t-shirts with prints, sanitary napkins, bandaids and diapers and they are used as fungicides on textiles that are exposed to extreme conditions, such as canvas. [272] Organotins were found in 50 percent of ordinary plastic products purchased in a Japanese supermarket—diaper covers, sanitary napkins, polyurethane gloves, cellophane wrap, dishwashing sponges and baking parchments. Organotins were also found in the cookies baked on the parchment.[273] Another study in Japan found organotins in children's PVC toys—face masks, balls, soft toys and food toys.[274]

Organotins have also been detected in drinking water transported through PVC pipe.[275,276,277,278] Elevated levels of organotins, particularly tributyltin, have also found in PVC flooring and somewhat lower concentrations in carpets.[279]

Organotins are found in ambient air and precipitation,[280,281] freshwater resources, ocean water, soils and sediments.[282,283,284] Organotins, particularly tributyltin (TBT), have been identified in many species including mollusks, fish, marine birds, marine mammals, and freshwater birds,[285] as well as various terrestrial mammals.[286] In short, these chemicals are ubiquitous in the environment.[287]

### Occurrence of Organotins in Humans

The Centers for Disease Control and Prevention (CDC) does not monitor organotins in the U.S. population. No data on organotin levels are given in CDC's National Report on Human Exposure to Environmental Chemicals of 2001 or the succeeding 2003 report,[288,289] and organotins will not be included the CDC's upcoming third report.[290]

Few studies of the occurrence of organotins in human tissues are available. However, in a 1999 study, organotins were tested in the blood of people living in Michigan: monobutyltin (MBT) was present in 53 percent of the samples; dibutyltin (DBT), 81 percent; and tributyltin (TBT), 70 percent. Concentrations were in the order of MBT > DBT $\geq$ TBT. The findings were taken to suggest exposure to MBT and DBT, which are used in a variety of consumer products.[291]

Organotins occurred with less frequency in blood samples from the Environmental Specimen Bank/ Human Specimen Bank in Germany: MBT was measurable in 17 percent of the samples; DBT in 3 percent; and no other organotins were detected.[292] Organotins have also been detected in the liver tissues of people in Japan,[293] Poland,[294] and Denmark.[295] Methyltins have also been detected in human urine[296]

### Effects of Organotins in Humans

Human fatalities from widespread poisoning with organotin occurred in France and Algeria in 1954 when Stalinon capsules, containing 15 mg of diethyltin, were used to treat staphylococcal skin infections.[297]

Accidental poisoning with trimethyltin has resulted in memory deficits, seizures, altered emotional affect, hearing loss, disorientation, and death. Limited evidence suggests that mono- and dimethyl tin are neurotoxic in humans.[298]

**Effects of Organotins in Other Species**

Organotins are toxic at relatively low levels of exposure. Tributyltins (TBT) and triphenyltins (TPT) are all listed as poisons and described as respiratory toxins, fetotoxins, reproductive toxins, immunotoxins, possible carcinogens, skin and respiratory irritants, and allergens.[299,300] TPT interferes with the cell components responsible for moving chromosomes into place before a cell divides and acts synergistically with pentachlorobiphenyl, a PCB, to induce abnormal chromosome arrangements in mitosis at very low concentrations.[301]

The relative toxicities of the butyltins are often considered to be TBT > DBT > MBT.[302] However, DBT is more toxic than TBT to certain enzyme systems,[303,304] and DBT is more toxic than TBT to the immune systems in fish.[305]

Studies have reported that MBT, DBT, and TBT act synergistically when exposed in combination (ie. their combined effects is more pronounced than the effects of each one added together). Butyltin compounds in blood have also been reported to be able to interact with other classes of contaminants such as organochlorines to lead to adverse effects.[306]

Organotins are also toxic to the immune system in rats and other mammals.[307] The immune system toxicity of DBT, TBT and triphenyltin stems from their capacity for destroying or limiting the functionality of various white blood cells.[308,309,310,311,312,313,314.315,316] DBT is frequently shown to be more toxic to the immune system than TBT.[317,318]

DBT is neurotoxic to mammalian brain cells.[319] DBT has been shown to exert toxic effects on the immune system at concentrations comparable to those reported in human blood.[320] DBT also had toxic effects on the nervous system at levels that were lower than those reported in human blood and some forty times lower than the lowest toxic concentration of trimethyltin, a known neurotoxicant.[321] These findings suggest that chronic, low-level exposure to DBT in human populations may have impacts on both the immune and nervous systems.

Organotins are transported through the placenta, as demonstrated by their adverse developmental effects.[322] A recent study suggests some *in utero* developmental effects may result from the impacts of relatively low doses of TBT on maternal thyroid function. Effects included: reduced maternal weight gain; increased post-implantation loss; decreased litter sizes; decreased fetal weights; delayed fetal skeletal development; and abnormalities in genital development in male fetuses.[323]

Some of the reproductive and developmental effects of organotins on mice and rats summarized in a recent review are: [324]

- TPT caused decreased fertility due to degenerative changes in testicular tissue and ovarian impairment.
- Triphenyltin chloride (TPTCl) and diphenyltin chloride (DPTCl) exposure during early pregnancy caused implantation failure.
- TPT exposure during pregnancy caused embryonic/fetal death, suppressed fetal growth at doses toxic to the mother, and, at lower doses, resulted in behavioral changes in the offspring.
- Maternal exposure to tributyltin chloride (TBTCl) decreased weights of male reproductive organs, decreased sperm counts, decreased serum estradiol levels, delayed vaginal opening, impaired estrous cyclicity, and increased female anogenital distance in the offspring. Given during early pregnancy, TBTCl or dibutyltin chloride (DBTCl) resulted in implantation failure.
- Maternal exposure to TBT resulted in embryonic/fetal deaths, suppressed fetal growth and cleft palate at doses toxic to the mother and, at lower doses, behavioral changes in offspring.
- DBT produced fetal malformations.

## Perfluorinated Chemicals

All dust samples contained quantifiable concentrations of the two target perfluorinated chemicals—perfluorooctanoic acid (PFOA) and perfluorooctanyl sulfonate (PFOS). PFOS concentrations were highest in all samples, with a mean of 424 ppm and a range of 76.4 to 1,170 ppm, while the mean concentration of PFOA was 78.7 ppm with a range of 18.5 to 205 ppm.

As illustrated in Figure 9, the mean total PFOS/PFOA concentration, 503 ppb, found in this study is quite close to that reported by Moriwaki et al. (2003) for house dust in Japan. However, PFOS was, by far, the largest contributor to the total concentration of these two perfluorinated chemicals in the dust samples of the present study, while PFOA was largest in dust samples from Japan. PFOS is generally regarded as the final product of degradation for other perfluorooctanyl compounds. The difference in the PFOS/PFOA ratios of these two groups of house dust samples may conceivably reflect earlier use of perfluorinated surfactants in consumer products in the U.S. and, consequently, greater opportunity for these substances to degrade to PFOS.

### Perfluorinated Chemicals—Production, Use, Occurrence and Effects

**PFOA is detectable in the blood of most humans and animals worldwide, which is problematic because it is only slowly eliminated in mammals, is potentially toxic, has no known metabolic or environmental degradation pathway, and is potentially carcinogenic.**

*— Ellis et al., 2005*[325]

The two perfluorinated chemicals (PFCs) that were selected as analytes for this study—perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS)—are only two of the already quite large and still growing number of PFCs that are manufactured and/or found in the environment. PFOA is the best-known of the PFCs because it is used to make Teflon, Goretex, and other oil-, water- and stain-resistant materials used in many common items, including nonstick frying pans, utensils, stove hoods, stainproofed carpets, furniture, and clothes.[326]

PFOA is used to make fluoropolymers, which had a global consumption rate of 112 thousand metric tons valued at $2.1 billion in 2001,[327] and fluoroelastomers, with a global consumption rate of 15 thousand metric tons valued at $700 million in 2002.[328] Polytetrafluoroethylene (PTFE or vinyl fluoride), commonly marketed as Teflon, accounted for 60-65 percent of all fluoropolymer consumption in the US., Western Europe and Japan in 2001.[329] DuPont has almost 50 percent of the global market share for fluoropolymers,[330] while the U.S. accounts for 45 percent of the world's fluoroelastomer consumption.[331] PFOA and PFOS may also be formed as products of the degradation of other PFCs.

Both fluoropolymers and fluoroelastomers are used in soil, stain, grease, and water-resistant coatings for textiles, carpet, cookware and automobiles. PFOA is also used widely in fire-fighting foams. PFOS has been used in refrigerants, surfactants, polymers, pharmaceuticals, flame retardants, lubricants, adhesives, cosmetics, paper coatings, and insecticides. However, the U.S. manufacturer, 3M, discontinued PFOS production in 2000.[332]

Relatively high concentrations of some PFCs have been found in the indoor air of homes. In Canada, three PFCs were detected in indoor air of two homes and a laboratory at concentrations about 100 times higher than in outdoor air. A PFC that is widely used as a stain repellent on carpets was the most abundant of the three PFCs in both indoor and outdoor air.[333] A later, more comprehensive study involving more than 50 homes confirmed that some PFCs are present in indoor air at very high concentrations.[334]

A PFC that is found as a by-product in oil- and water-repellent coatings for paper and paperboard used for food packaging was detected in more than 55 percent of composite fast food samples in Canada. This PFC is also used as a pesticide in the U.S.[335]

PFCs are pervasive contaminants in the global environment. PFOS and other PFCs are found in freshwater and marine mammals, fish, birds, shellfish, and domestic cattle.[336,337,338,339,340,341,342,343] Although distribution is global, including remote locations in the Arctic and North Pacific Oceans, concentrations of PFCs are relatively greater in or near the more populated and industrial regions.

**It was known as early as 1975 that fumes from hot pans coated with polytetrafluoroethylene (PTFE, Teflon) can kill pet birds. Broiler chicks have died after exposure to polytetrafluoroethylene-coated light bulbs.**

### Occurrence of Perfluorinated Chemicals in Humans

The Centers for Disease Control and Prevention (CDC) does not monitor PFCs in the U.S. population. No data on PFC levels are given in CDC's National Report on Human Exposure to Environmental Chemicals of 2001 or the succeeding 2003 report,[344,345] and no PFCs will be included the CDC's upcoming third report.[346] However, U.S. EPA has proposed that CDC include PFOS, PFOA and other perfluorinated chemicals in the next national study.[347]

A number of studies have found PFCs to be pervasive contaminants in the blood of the general population of the U.S. A broad survey of individual blood samples from adult Red Cross blood donors,[348] children from a clinical trial, [349]and a group of elderly people from Seattle, Washington.[350] A number of studies of fluorochemical production workers found PFC levels in blood that were, on average,20-30 times higher than the concentrations found in the general population.[351,352,353,354]

For very thorough and detailed descriptions of the findings of these studies as well as potential health impacts and other aspects of the perfluorinated chemicals, see the Environmental Working Group report, "PFCs: A Family of Chemicals That Contaminate the Planet," and related materials at *http://www.ewg.org/reports/pfcworld/*

PFCs are also found in the blood of the general populations of Italy, Colombia, Brazil, Belgium, Poland, India, Malaysia, and Korea[355] as well as Sweden,[356,357] Japan,[358] and in indigenous peoples in Northern

Canada.[359] PFCs are also found in the liver tissue of the general U.S. population.[360]

The presence of PFCs in cord blood has been demonstrated,[361,362] indicating that these chemicals pass from the mother through the placenta to the developing fetus.[363] PFCs have been detected in human breast milk in one very limited study that found fewer specific PFCs at concentrations below those in blood serum.[364] This study, a study with rats,[365] and a study of wood mice[366] all indicate that PFCs are passed from mother to breastfeeding infant.

**Effects of Perfluorinated Chemicals in Humans**

The following text is excerpted from the summary and conclusions of the OECD hazard assessment of PFOS and its salts: [367]

> *"Several occupational studies have been conducted on volunteers at the 3M plants in Decatur, Alabama and Antwerp, Belgium.… In a mortality study, which followed workers for 37 years, mortality risks for most of the cancer types and non-malignant causes were not elevated. However, a statistically significant risk of death from bladder cancer was reported. Three male employees in the cohort died of bladder cancer (0.12 expected), and all of them had been employed at the plant for more than 20 years. All of them had also worked in high exposure jobs for at least 5 years. In order to screen for morbidity outcomes, an "episode of care" analysis was undertaken for employees who had worked at the plant between 1993 and 1998. Many different types of cancer and other non-malignant conditions were examined. Increased risks were not reported for most of the conditions or did not reach statistical significance. However, an increased risk of episodes was reported for neoplasms of the male reproductive system, the overall category of cancers and benign growths, and neoplasms of the gastrointestinal tract. These risk ratios were highest in employees with the highest and longest exposures to fluorochemicals."*

**Effects of Perfluorinated Chemicals in Other Species**

It was known as early as 1975 that fumes from hot pans coated with polytetrafluoroethylene (PTFE, Teflon) can kill pet birds, and broiler chicks have died after exposure to polytetrafluoroethylene-coated light bulbs.[368]

In 1979, 3M administered four doses of PFOS to monkeys and all the monkeys in all treatment groups died within weeks.[369] Note that as mentioned above, 3M did not discontinue production of this chemical until 2000.

A study of female laboratory rats indicates that PFOS can affect the neuroendocrine system. Exposed to PFOS, female rats evidenced loss of appetite, interrupted estrus cycles, and elevated stress hormone levels. PFOS was found to accumulate in brain tissue, particularly the hypothalamus, suggesting that PFOS crosses the blood-brain barrier and may interfere with reproductive hormones through the pituitary-hypothalamus process that stimulates their production.[370]

One recent review noted that studies in monkeys, rats, fish and humans have found that subchronic exposure to PFOS led to significant weight loss, liver toxicity, reduced serum cholesterol, and reduced thyroid hormones. In rats, rabbits and mice, developmental effects included reduced fetal weight, cleft palate, edema, delayed ossification of bones and cardiac abnormalities.[371]

Exposure of rats and mice to PFOS during pregnancy resulted in both toxic effects to the mother and birth defects in the offspring. Exposed mothers exhibited a broad range of effects: dose-dependent suppression of maternal weight gain, reduced maternal thyroid hormone levels (thyroxine and triiodothyronine); reduced maternal serum triglycerides; and marked enlargement of the liver at higher doses. PFOS was detected in the livers of rat fetuses at levels about half of those of their mothers. Among the resulting birth defects were cleft palate, generalized edema, ventricular septal defect, and enlargement of the right atrium.[372] The follow-up study found that PFOS exposure during pregnancy severely compromised survival of the offspring, caused delays in growth and development, and was accompanied by reduced thyroid hormone levels. At the highest PFOS dosages, 95 percent of the newborn rats and mice died within 24 hours of birth. Survival improved at lower dosages.[373]

Wood mice from a nature reserve near two fluorochemical facilities in Belgium PFOS-contaminated areas in Belgium showed an age-dependent increase in PFOS concentrations in liver tissue, increased liver weights at high PFOs concentrations in the liver, decreased serum triglyceride levels with increased PFOS exposure, evidence of possible liver damage, and evidence of maternal PFOS transfer to the young during pregnancy and/or lactation.[374]

Recent laboratory studies with PFOA involving rats show low birth weight, small pituitary gland, altered maternal care behavior, high pup mortality, and significant changes in the brain, liver, spleen, thymus, adrenal gland, kidney, prostate, testes and epididymides.[375]

# APPENDIX II: Company Rankings on Chemical Policies

## Explanation of Color Coding in Company Ranking

| Color Code | Rating Description |
|---|---|
| **Gold Star** | Company adheres to the Safer Chemicals Pledge |
| **Green** | Company avoids the use of the Priority OSPAR[1] list of chemicals in their product line; this is stated to us via the questionnaire or through detailed information about their chemical policy on their website. |
| **Yellow** | Company is transitioning out of one or more high priority chemicals - this is stated to us via the questionnaire or through detailed information about their chemical policy on their website or through publications. |
| **Orange** | Company has not replied to the questionnaire, has no detailed information about chemical policy on their website but has recently made a public announcement to phase out one or more of the OSPAR group of chemicals. |
| **Red** | Company has not replied nor gives any detailed information online to their consumers about their chemical policy. |

* OSPAR (1992) List of Chemicals for Priority Action are internationally recognized chemicals of high concern which North Atlantic European countries have committed to eliminate over the long term (www.ospar.org)

## Cosmetic Manufacturers

| Color Code | Company | Incorporation of a Sustainable Chemicals Policy | Contact Information |
|---|---|---|---|
| **Yellow** | Aveda | In accordance with the company's Material Use Guidelines, Aveda is working to eliminate the use of any materials considered persistent organic toxins. | http://www.aveda.com/contactus/contactus.tmpl |
| **Yellow** | Unilever | Unilever is committed to phasing out phthalates in products sold in the USA. The company has adopted improved screening methodologies to avoid the use of chemicals that disrupt the endocrine system. The company does not use alkylphenols in European products, it is unclear whether or not US products contain these chemicals. | http://www.unilever.com/home/contactus/ |
| **Orange** | L'Oreal | L'Oreal is committed to phasing out phthalates. The company does not have a public chemical policy to ensure that their suppliers replace alkylphenols and other chemicals of concern with safer alternatives. | http://www.loreal.com/_en/_ww/tools/index.aspx?contact/contact_1.aspx |
| **Orange** | Revlon | Revlon is committed to phasing out phthalates. The company does not have a public chemical policy to ensure that their suppliers replace alkylphenols and other chemicals of concern with safer alternatives. | http://www.revlon.com/information/contactform.asp |
| **Orange** | Estee Lauder Inc. | Estee Lauder Inc. is committed to phasing out phthalates. The company does not have a public chemical policy to ensure that their suppliers replace alkylphenols and other chemicals of concern with safer alternatives. | http://www.elcompanies.com/htm/frameset/frm_m5.htm |
| **Orange** | Procter & Gamble | Procter and Gamble is committed to phasing out phthalates. The company does not have a public chemical policy to ensure that their suppliers replace alkylphenols and other chemicals of concern with safer alternatives. | http://www.pg.com/getintouch/index.jhtml |

## Retailers

| Color Code | Company | Incorporation of a Sustainable Chemicals Policy | Contact Information |
|---|---|---|---|
| **Yellow** | IKEA | Most of the OSPAR high priority chemicals are not used in IKEA products. IKEA has phased out the use of brominated flame retardants (BFRs) in mattresses, carpets, and furniture. IKEA is still working to phase out BFRs in their lighting fixtures. With the exception of cables, IKEA phased out all uses of PVC by 1996, thereby significantly reducing and often eliminating the use of phthalates and organotins. They have a phthalate ban on all children's products. IKEA bans pesticides from their products. IKEA also bans the use of carcinogens in their products. | http://info.ikea-usa.com/IKEAContactUs/Contact.aspx |
| **Red** | Target | Target does not have a public chemical policy to ensure that their suppliers avoid OSPAR and other chemicals recognized as posing a risk to human health. | 1-800-440-0680 |
| **Red** | Sears | Sears does not have a public chemical policy to ensure that their suppliers avoid OSPAR and other chemicals recognized as posing a risk to human health. | 1-800-349-4358 |
| **Red** | Walmart | Walmart does not have a public chemical policy to ensure that their suppliers avoid OSPAR and other chemicals recognized as posing a risk to human health. | 1-800-WAL-MART |
| **Red** | Home Depot | Despite partnerships with Natural Step and a commitment to green alternative products, Home Depot does not have a public chemical policy to ensure that their suppliers avoid OSPAR and other chemicals recognized as posing a risk to human health. | 1-800-553-3199 |

## Televisions

| Color Code | Company | Chemical Policy in the US | Contact Info |
|---|---|---|---|
| **Yellow** | Samsung Electronics | Samsung Electronics is committed to phasing out brominated flame retardants by the end of 2005. The company is also committed to finding safer alternatives for other OSPAR chemicals including phthalates, and organotins as well as replacing materials such as PVC that release chemicals of concern. | http://www.samsung.com/ContactUs/ContactUs.htm |
| **Yellow** | Sony | Sony has a public policy to transition out of all applicable OSPAR chemicals. For brominated flame retardants, Sony is committed to replacing them with halogen free alternatives, including TBBPA. Sony restricts the use of PVC in certain applications and has started to use biobased plastics in some of their products. | http://esupport.sony.com/feedback/feedback.html |
| **Orange** | Panasonic | Despite a strong commitment to increase the use of halogen free (PVC free) plastics and lead-free solders, Panasonic does not have a clear chemicals policy for other hazardous materials. In Europe, they will introduce chlorine free wiring within 2005 and eliminate the use of PVC by the end of March 2006. It is unclear whether or not this is a global commitment. | http://www.panasonic.com/environmental/contact_us.asp |
| **Orange** | Philips | Despite a strong commitment to sustainability and the removal of lead from their products, Philips does not have a clear public policy on thier use of other hazardous materials in US products. In Europe, Philips is committed to removing brominated flame retardants by 2006, but it is unclear whether or not this is a global commitment. | 1-888-744-5477 |
| **Red** | JVC | JVC does not have a public chemical policy to ensure that their suppliers avoid OSPAR and other chemicals recognized as posing a risk to human health. | 1-800-252-5722 |

## Computers

| Color Code | Company | Chemical Policy | Contact Info |
|---|---|---|---|
| **Yellow** | Dell | Dell has a public policy to transition out of all applicable OSPAR chemicals. Brominated flame retardants (BFRs) in Dell's desktops, notebooks, and server chassis plastic parts were to be replaced by year-end 2004. Dell is committed to replacing BFRs, including TBBPA, with halogen free alternatives. Dell is working to avoid PVC and all halogenated plastics, which will reduce phthalate and organotin exposure. | Tel: 888-560-8324 |
| **Yellow** | Hewlett Packard | HP has a public policy to transition out of some OSPAR chemicals. HP has replaced deca-BDE with a halogen free alternative, but is not committed to non-halogenated alternatives for other BFRs, such as TBBPA. HP restricts the use of PVC in certain applications. | http://welcome.hp.com/country/us/en/contact/email_2.html |
| **Yellow** | Apple | Apple has a public policy to transition out of most OSPAR chemicals. Apple is committed to phasing out all PBDEs, but does not commit to a halogen free alternative for Deca. Apple does not have a phase-out target for TBBPA. Apple does not have a policy to restrict the use of plastics, such as PVC that contribute to phthalate and organotin exposure. | https://www.apple.com/contact/ |
| **Yellow** | IBM | IBM has a public policy to transition out of some OSPAR chemicals. In 1991, IBM phased out all PBDEs, but has no defined commitment to replace TBBPA with safer alternatives. IBM does not have a policy to restrict the use of plastics, such as PVC that contribute to phthalate and organotin exposure. | https://www.ibm.com/contact/us/en/query |
| **Orange** | Toshiba | Toshiba is in compliance with the Restriction of Hazardous Substances Directive(RoHS), which means it is phasing out PBDEs—brominated flame retardants. Depsite a comprehensive website dedicated to Toshiba's environmental initiatives, it is unclear whether or not they are globally phasing out other hazardous chemicals besides those targeted in the RoHS Directive. | http://www.toshiba.com/taisnpd/contactus/email.html |

## Mattresses

| Color Code | Company | Chemical Policy | Contact Information |
|---|---|---|---|
| Green | IKEA | IKEA has phased out the use of brominated flame retardants (BFRs) and phthalates in mattresses. IKEA also bans the use of carcinogens in their products. | http://info.ikea-usa.com/IKEAContactUs/Contact.aspx |
| Orange | Serta | As of January 2005, Serta plans to have their mattresses free of brominated flame retardants (BFRs). Serta does not have a public chemical policy to ensure that their suppliers avoid OSPAR and other chemicals recognized as posing a risk to human health. | customer.service@serta.com |
| Orange | Sealy | In the US, Sealy does not have a public chemical policy to ensure that their suppliers avoid OSPAR and other chemicals recognized as posing a risk to human health. In England, Sealy is committed to moving away from OSPAR chemicals. | 1-800-MY-SEALY |
| Red | Simmons | Simmons does not have a public chemical policy to ensure that their suppliers avoid OSPAR and other chemicals recognized as posing a risk to human health. | 1-877-399-9397 |
| Red | Sears-0-Pedic | Sears does not have a public chemical policy to ensure that their suppliers avoid OSPAR and other chemicals recognized as posing a risk to human health. | 1-800-349-4358 |

## Carpets and Flooring

| Color Code | Company | Chemical Policy | Contact Information |
|---|---|---|---|
| **Green** | IKEA | IKEA has phased out the use of brominated flame retardants (BFRs) in carpets. IKEA bans the use of PVC in most of its products, which reduces and in some cases eliminates the use of organotins and phthalates. IKEA also bans the use of carcinogens in their products. | http://info.ikea-usa.com/IKEAContactUs/Contact.aspx |
| **Yellow** | Shaw | Shaw Inc. is committed to using MBDC's Cradle to Cradle protocol* to replace persistent bioaccumulative toxins with safer alternatives. EcoWorx® Backing, their new carpet back does not contain PVC. | http://www.shawfloors.com/about/Shaw/Contact_Shaw.asp |
| **Yellow** | Interface | Interface is committed to moving away from petro-based materials to avoid the release and use of harmful chemicals. Their Ingeo™ carpet line uses bio-based, plant-derived fibers and their i2™ flooring products was designed using biomimicry to ensure safe material use that can be reused and recycled infinitely. | http://66.110.208/contactus.aspx?source=interfaceinc |
| **Red** | Mohawk | Mohawk does not have a public chemical policy to ensure that their suppliers avoid OSPAR and other chemicals of concern. | mohawkind@mohawkind.com |

* McDonough Braungart Design Chemistry (www.mbdc.com)

## Furniture

| Color Code | Company | Chemical Policy | Contact Information |
|---|---|---|---|
| **Yellow** | IKEA | IKEA has eliminated brominated flame retardants and is working to replace persistent bioaccumlative chemicals with safer alternatives in all their product lines | http://info.ikea-usa.com/IKEAContactUs/Contact.aspx |
| **Yellow** | Herman Miller | Herman Miller is committed to using MBDC's Cradle to Cradle protocol* to replace persistent bioaccumulative chemicals with safer alternatives in all their product lines. As a result, their new chair, MIRA, does not contain BFRs or PVC. | www.Hermanmiller.com 1-888 443 4357 |
| **Yellow** | Steel Case | Steel Case is committed to using MBDC's Cradle to Cradle protocol to replace persistent bioaccumulative chemicals with safer alternatives for all their product lines. | http://www.steelcase.com/na/askus.aspx?f=13918&p=18518 |
| **Red** | Century Furniture | Century Furniture does not have a public chemical policy to ensure that their suppliers avoid OSPAR and other chemicals recognized as posing a risk to human health. | webcs04@centuryfurniture.com |
| **Red** | La-Z-Boy | La-Z-Boy does not have a public chemical policy to ensure that their suppliers avoid OSPAR and other chemicals recognized as posing a risk to human health. | http://www.la-z boy.com/contactus/ |

* McDonough Braugart Design Chemistry (www.mbdc.com)

TABLE 7

## The Twelve Principles of Green Chemistry

**1. Prevention**
It is better to prevent waste than to treat or clean up waste after it has been created.

**2. Atom Economy**
Synthetic methods should be designed to maximize the incorporation of all materials used in the process into the final product.

**3. Less Hazardous Chemical Syntheses**
Wherever practicable, synthetic methods should be designed to use and generate substances that possess little or no toxicity to human health and the environment.

**4. Designing Safer Chemicals**
Chemical products should be designed to effect their desired function while minimizing their toxicity.

**5. Safer Solvents and Auxiliaries**
The use of auxiliary substances (e.g., solvents, separation agents, etc.) should be made unnecessary wherever possible and innocuous when used.

**6. Design for Energy Efficiency**
Energy requirements of chemical processes should be recognized for their environmental and economic impacts and should be minimized. If possible, synthetic methods should be conducted at ambient temperature and pressure.

**7. Use of Renewable Feedstocks**
A raw material or feedstock should be renewable rather than depleting whenever technically and economically practicable.

**8. Reduce Derivatives**
Unnecessary derivatization (use of blocking groups, protection/deprotection, temporary modification of physical/chemical processes) should be minimized or avoided if possible, because such steps require additional reagents and can generate waste.

**9. Catalysis**
Catalytic reagents (as selective as possible) are superior to stoichiometric reagents.

**10. Design for Degradation**
Chemical products should be designed so that at the end of their function they break down into innocuous degradation products and do not persist in the environment.

**11. Real-time analysis for Pollution Prevention**
Analytical methodologies need to be further developed to allow for real-time, in-process monitoring and control prior to the formation of hazardous substances.

**12. Inherently Safer Chemistry for Accident Prevention**
Substances and the form of a substance used in a chemical process should be chosen to minimize the potential for chemical accidents, including releases, explosions, and fires.

# APPENDIX III: Analytical Methods and Data Quality of Chemical Concerntrations Measured in Household Dust Composites from Seven U.S. States

## Introduction

Numerous organic chemicals have been detected in house dust, both in the United States (Camann et al., 2002; Rudel et al., 2003) and in the United Kingdom (Santillo et al., 2003). Clean Production Action contracted with an EPA-accredited laboratory (name provided on request) to measure the concentrations of selected chemicals in composited dust samples from ten households from each of seven states of the United States. The laboratory is currently certified by the National Environmental Laboratory Accreditation Program and possesses the ISO 9001:2000 Certificate of Registration. The chemicals which were targeted are 7 phthalate diesters, 7 brominated diphenyl ethers, 13 pesticides, 7 alkylphenol compounds and pentachlorophenol, 7 organotins, and 2 perfluorinated organics; the specific chemicals are identified in Table 1. The laboratory determined the concentrations of each of these chemicals in the composite dust sample from each state.

## Methods and Materials Sieving and Compositing of Bag Dust Samples

Bag dust samples from ten households in each of seven states (CA=California, MA=Massachusetts, ME=Maine, MI=Michigan, NY=New York, OR=Oregon, and WA=Washington) were received at the laboratory between September 28 and October 18, 2004, and stored frozen until sieving. A composite sieved dust sample was prepared from the ten designated bag dust samples from each state. Each designated bag from a state was opened consecutively and about 30 g of dust from 9 specific representative locations was passed through a cleaned 150-um stainless steel sieve, to obtain a 3.0 g weighed aliquot of each fine dust. The ten 3.0 g aliquots were then combined and passed twice through the sieve to create a homogenized state composite, which was then split into separate 2.0 g aliquots (including those labeled N=Neutrals, P=Phenols, F=PFOA/PFOS, and O=Organotins) for the necessary extractions and analyses. At least one 2 g dust aliquot was fortified with all the target analytes of a specific method before extraction and analysis, in order to

TABLE 1

### Target Chemicals in Dust Survey for Clean Production Action

**26 Neutral Chemicals*** (by GC/MS on 60 m column):

- 7 Phthalate Diesters: dimethyl (DMP), diethyl (DEP), di-n-propyl (DPP), diisobutyl (DiBP), di-n-butyl (DnBP), butylbenzyl (BBzP), di(2-ethylhexyl) (DEHP)
- 6 Brominated Diphenyl Ethers: BDE 47, BDE 99, BDE 100, BDE 153, BDE 154, BDE 183
- 13 Pesticides: chlorpyrifos, alpha-chlordane, gamma-chlordane, 4,4'-DDT, diazinon, dicofol + 4,4'-dichlorobenzophenone (breakdown product), dieldrin, methoxychlor, pentachloronitrobenzene, cis-permethrin, trans-permethrin, piperonyl butoxide, propoxur

**Decabrominated Diphenyl Ether (BDE 209)** (by GC/MS on 15 m column):

**8 Phenolic Chemicals*** (by GC/MS):

- 7 Alkylphenol Compounds: 4-nonylphenol (4NP), nonylphenol monoethoxylate, nonylphenol diethoxylate, 4-octylphenol (4OP), octylphenol monoethoxylate, octylphenol diethoxylate, 4-(1,1,3,3-tert-methylbutyl) phenol (4TMBP)
- Pentachlorophenol

**7 Organotins** (by GC/MS):

- 7 Organotins: monobutyltin (MBT), dibutyltin (DBT), tributyltin (TBT), tetrabutyltin (TeBT), dioctyltin (DOT), tricyclohexyltin (TCHT), triphenyltin (TPT)

**2 Perfluorinated Organics** (by LC/MS negative electrospray):

- Perfluorooctanoic acid (PFOA), Perfluorooctanyl sulfonate (PFOS)

* The laboratory extracted and analyzed split composited fine (<150 um) dust samples for the targeted compounds, generally as described in Rudel et al (2003).

assess the extraction efficiency of each target analyte from dust.

**Extraction and Analytical Methods**

**Neutral-extracted Chemicals.** Approximately 2 g of sieved fine dust was spiked with five extraction surrogates, equilibrated for 30 minutes at room temperature, then Soxhlet-extracted first using 6% ether in hexanes and then hexane:acetone (1:1), for 16 hours each. The extracts were combined and concentrated to 10 mL, from which 1 mL was removed for cleanup. The cleaned eluent was concentrated to a final volume of 2 mL with 10% ether in hexanes.

Analysis for the 26 neutral target chemicals was performed using an Agilent 6890/5973 GC/MS in selected ion monitoring (SIM) mode, as described by Rudel et al (2003). A 60 m x 0.25 mm i.d. ZB-5MS column was used as the GC analytical column. The GC/MS instrument was scanned to monitor 2 or 4 selected ions per analyte. Quantification was performed using labeled compounds similar to the analytes as internal standards. Analysis for BDE-209 was performed by GC/MS/SIM using a DB-5MS 15 m x 0.25 mm GC column.

**Phenolic Chemicals.** Approximately 2 g of sieved fine dust was acidified, spiked with 2,4,6-tribromophenol as the extraction surrogate, equilibrated for 30 minutes at room temperature, and extracted by sonication with dichloromethane. The extract was solvent-exchanged to hexane at a final volume of 20 mL.

Target phenols in extracts and calibration standards were converted to their silyl derivatives prior to GC/MS analysis. Analysis for the 8 phenolic chemicals was performed using an Agilent 6890/5973 GC/MS in selected ion monitoring (SIM) mode, as described by Rudel et al (2003). A 30 m x 0.25 mm i.d. DB-5MS column was used as the GC analytical column. Quantification was performed using 3,4,5-trichlorophenol as the internal standard.

**Perfluorooctanoic Acid (PFOA) and Perfluorooctanyl Sulfonate (PFOS).** Approximately 2 g of sieved fine dust was sonicated in a polyethylene container for 30 minutes in 10 mL of methanol for the extraction of PFOA and PFOS. The samples were then centrifuged and ~ 1 mL of the extract was removed for LC/MS analysis. Perfluorononanoic acid (PFNA) was added prior to extraction as a surrogate to monitor the extraction efficiency of the compounds. Analysis was performed using liquid chromatography/mass spectrometry (LC/MS). Analytical conditions were based on reverse phase HPLC separation with negative mode electrospray ionization mass spectrometry. Quantitation was performed using the external standard method.

**Organotins.** Approximately 1 g of sieved fine dust was extracted by shaking in 4 mL diethyl ether:hexanes (4:1) with 0.1% tropolone for the organotin compounds. Tri-n-propyltin chloride was added prior to extraction as a surrogate to monitor the extraction efficiency of the compounds. The samples were then centrifuged and 2 mL of the extract was removed for derivatization. The 2 mL aliquot was derivatized using n-pentylmagnesium bromide (Grignard reagent) to form volatile pentyl derivatives suitable for GC/MS analysis. The derivatized extract was diluted two-fold prior to GC/MS analysis. Calibration standards were derivatized in the same manner.

Analysis was performed using GC/MS. A six point calibration standard curve ranging from 0.4 – 0.01 µg/mL was used to quantitate the compounds in the extract. Due to the different salt forms of organotins available, the concentration values were based solely on the cation portion of the compound. For example, tri-n-butyltin chloride was used in the calibration standard but the concentrations were calculated based on tri-n-butyltin only. The WA and CA dust samples were selected as matrix spike samples. Tetrabutyltin was recovered in both samples. Most of the recoveries of the organotin compounds ranged from 10–40% with the exception of monobutyltin with recoveries less than 10%.

**Data Reporting Qualifiers.** Data reporting qualifiers were used to report special circumstances that may affect interpretation of the analyte=s value for a sample:

U (< detection limit) Analyte not detected. Nominal DL = lowest standard/3.0

B Analyte present in solvent blank. Blank value not subtracted.

BS Blank subtracted. Analyte level in solvent blank subtracted from measured level.

E Analyte amount is elevated due to interference peak

J Imprecise quantification: amount below lowest standard

## Evaluation of Analytical Data Quality

### Solvent Blanks

A solvent blank was processed through all extraction steps for each extraction method and analyzed along with the dust samples. The solvent blank results were evaluated. The only target chemicals present in the solvent blanks were diethyl phthalate (DEP) (at 2.31 ug/extract) and di(2-ethylhexyl) phthalate (DEHP) (at 2.26 ug/extract) in the neutrals method solvent blank, and 4-nonylphenol (4NP) (at 4.69 ug/extract) in the phenols method solvent blank. There is no indication of laboratory-introduced contamination for any of the other targeted chemicals.

### Blank Subtraction

If 2 grams of dust had been extracted, the detected solvent blank contaminant levels are equivalent to 1.15 ug/g of DEP, 1.13 ug/g of DEHP, and 2.35 ug/g of 4NP. Lab-introduced DEHP (of about 1.13 ug/g) contributed less than 1% to the DEHP composite dust measurements, which ranged from 216 ug/g to 425 ug/g. However, lab-introduced DEP (of about 1.15 ug/g) contributed substantially to the measured DEP composite dust measurements, which ranged from 1.89 ug/g to 4.73 ug/g. Similarly, lab-introduced 4NP (of about 2.35 ug/g) contributed considerably to the measured 4NP composite dust measurements, which ranged from 6.08 ug/g to 12.86 ug/g. The final corrected concentrations of diethyl phthalate, di(2-ethylhexyl) phthalate, and 4-nonylphenol were obtained by subtracting the solvent blank concentrations of DEP, DEHP, and 4NP from the measured dust concentrations of these chemicals, in order to adjust for the laboratory-introduced levels, as indicated by the solvent blanks. After blank subtraction, the corrected dust concentrations of the state composites ranged from 0.74 ug/g to 3.58 ug/g for DEP, and from 3.74 ug/g to 10.5 ug/g for 4NP.

### Surrogate Recovery

Extraction surrogate compounds were spiked into each dust sample and QC sample before extraction by each method to indicate the adequacy of the extraction procedure. The five neutrals extraction surrogates were decachlorobiphenyl for the organochlorines, chlorfenvinphos for the organophosphate pesticides, and dibenzyl phthalate, diphenyl isophthalate, and diphenyl phthalate for the phthalate diesters and brominated diphenyl ethers. Recovery of the extraction surrogates in each extracted sample was evaluated. Recoveries of each of the five neutrals surrogates ranged between 50% and 109% in the seven composite dust samples, indicating adequate neutral extraction. Recovery of 2,4,6-tribromophenol, the phenols surrogate, was good (87% to 98%) from most dust composites, but very low from the MA (22%) and MI (45%) composites. Low recovery of 2,4,6-tribromophenol may indicate inefficient extraction of pentachlorophenol from the MA and MI composites. Recovery of perfluorononanoic acid (PFNA), the PFOA/PFOS surrogate, was generally elevated (102% to 192%) from the dust composites, but indicates that PFOA and PFOS were completely extracted from the dust. Recovery of tri-n-propyltin, the organotin surrogate, was low but consistent (55% to 65%) from the dust composites, indicating equivalent organotin extraction of each state dust composite, but recovery of tri-n-propyltin was lower (32.5%) in both organotin matrix spikes.

### Dust Matrix Spike Recoveries

At least one matrix spike of all targeted analytes was prepared into a composite dust aliquot and extracted and analyzed by each method, and the spike recoveries of each analyte were calculated. One matrix spike (of the NY composite dust) with all neutrals- and phenols-targeted analytes was performed and analyzed. Two matrix spikes (of the NY and OR composite dusts) with PFOA and PFOS were performed and analyzed. Two matrix spikes (of the CA and WA composite dusts) with the seven organotins were performed and analyzed. The dust matrix spike recoveries obtained for every targeted analyte by its extraction method were evaluated. Most neutral targeted compounds were extracted quite efficiently (70% to 130%) from the NY composite neutrals matrix spike, although recoveries were low for DEP (54%) and BDE 153 (62%) and were high for piperonyl butoxide (305%), DEHP (237%), butylbenzyl phthalate (215%), trans-permethrin (185%), and cis-permethrin (176%). All targeted phenolic compounds were extracted quite efficiently (80% to 136%) from the NY composite phenols matrix spike, with the exception of the low recovery of pentachlorophenol (52%). The matrix spike recovery of 4-n-nonylphenol (80%) was used to estimate the recovery of the multi-component 4-nonylphenol (4NP). Recoveries of PFOA were good (69% and 98%) from the NY and OR composite perfluorinated organic matrix spikes, but PFOS recoveries were elevated (176% and 197%). Recoveries of tetrabutyl-

tin were good (95% and 100%) from the CA and WA composite organotin matrix spikes, but recovery of the other six organotins was very poor but consistent from the CA and WA organotin matrix spikes. The mean recoveries were 37% for tributyltin, 26% for di-n-octyltin, 26% for dibutyllin, 23% for tricyclohexyltin, 14% for triphenyltin, and only 1% for monobutyltin. Another organotin extraction method gave similar matrix spike recoveries and similar measured dust concentrations as the reported organotin results.

**Accuracy of Measured Concentrations in Dust Composites Deduced from Matrix Spike Recoveries**

Per the laboratory's recommendation, the reported concentrations of the targeted chemicals in dust were not adjusted for the matrix spike recoveries. Instead, the matrix spike recoveries were used to assess the accuracy of the measured concentrations of the targeted neutrals chemicals, phenolic chemicals, PFOA and PFOS, and organotin chemicals in the seven state composite fine dust samples. The blank-subtraction corrected concentrations (denoted with the flag BS) were reported and used for diethyl phthalate, di(2-ethylhexyl) phthalate, and 4-nonylphenol, as recommended by the laboratory.

The dust matrix spike recoveries obtained provide an indication of the accuracy of the reported chemical concentrations in the state dust composite samples. The reported dust concentrations are probably quite accurate for those chemicals which were extracted efficiently. However, the reported dust concentrations may be overestimates for chemicals with high matrix spike recoveries (piperonyl butoxide, DEHP, butylbenzyl phthalate, trans-permethrin, cis-permethrin, and PFOS) and underestimates for chemicals with low matrix spike recoveries (DEP, BDE 153, pentachlorophenol, tributyltin, di-n-octyltin, dibutyllin, tricyclohexyltin, triphenyltin, and monobutyltin). Since the recoveries of some chemicals vary considerably from different dust matrices (Rudel et al., 2003) due to the variable composition of house dust, the degree of over-estimation or under-estimation in the reported dust concentrations of a chemical may also vary across the dust samples.

## References

Camann DE, Colt JS, and Zuniga MM. Distributions and Quality of Pesticide, PAH, and PCB Measurements in Bag Dust from Four Areas of USA. In: *Proceedings of Indoor Air 2002*, H. Levin, ed., Santa Cruz, CA, 2002, Vol. 4, pp 860-864.

Rudel RA, Camann DE, Spengler JD, Korn LR, Brody JG, 2003. Phthalates, Alkylphenols, Pesticides, Polybrominated Diphenyl Ethers, and Other Endocrine-Disrupting Compounds in Indoor Air and Dust. *Environ. Sci. Technol.*, 37 (20), 4543-4553.

Santillo D, Labunska I, Davidson H, Johnston P, Strutt M, Knowles O, 2003. Consuming Chemicals: Hazardous chemicals in house dust as an indicator of chemical exposure in the home. Greenpeace Research Laboratories, Department of Biological Sciences, University of Exeter, Exeter, UK

## Phthalates

| Phthalates | µg/g, parts per million (ppm) | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | CA | ME | NY | WA | OR | MI | MA | Mean, 7 samples | Belgium | Brazil | Cape Cod, MA | UK |
| dimethyl phthalate | <rl | 0.272 | <rl | <rl | <rl | <rl | <rl | 0.039 | 1.5 | 1.4 | na | 0.12 |
| diethyl phthalate | 0.74 | 0.74 | 3.58 | 1.07 | 1.41 | 0.86 | 1.47 | 1.41 | 10 | 2.5 | 8.5 | 12.2 |
| di-n-propyl phthalate | <rl | <rl | <rl | <rl | <rl | <rl | <rl | <rl | na | na | <rl. | <rl |
| diisobutyl phthalate | 8.35 | 3.01 | 3.80 | 3.05 | 4.49 | 1.61 | 2.20 | 3.79 | 74.6 | 29 | 2.92 | 52 |
| di-n-butyl phthalate | 13.0 | 10.5 | 28.0 | 20.8 | 49.5 | 7.8 | 11.4 | 20.15 | 32.4 | 52.2 | 27.3 | 50.2 |
| butylbenzyl phthalate | 137 | 71.4 | 64.2 | 56.9 | 42.1 | 49.9 | 63.9 | 69.4 | 195.8 | 3.2 | 124 | 56.5 |
| di(2-ethylhexyl) phthalate | 393 | 425 | 342 | 338 | 301 | 292 | 215 | 329 | 338.7 | 241.5 | 506 | 191.5 |

Belgium—Al Bitar (2004) also assayed dicyclohexy phthalate (1.7 ppm), di-n-octyl phthalate (55.7 ppm), di-isononyl phthalate (162.9 ppm), and di-isodecyl phthalate (66 ppm), but did not assay di-n-propyl phthalate

Cape Cod, MA—Rudel et al. (2003) also assayed dicyclohexyl phthalate (2.98 ppm), di-n-hexyl phthalate (2.6 ppm), di-n-pentyl phthalate (<rl), but did not assay dimethyl phthalate.

U.K.—Santillo et al. (2003) assayed di-isononylphthalate (48.5 ppm) and di-isodecylphthalate (20.8 ppm),

Brazil—Costner et al. (2004 also assayed dicyclohexyl phthalate (0.62 ppm), di-n-octyl phthalate (1.4 ppm), di-isononyl phthalate (71.2 ppm), and di-isodecyl phthalate (93 ppm), but did not assay di-n-propyl phthalate.

## Alkylphenols

| Alkylphenols | µg/g, parts per million (ppb) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | CA | WA | MI | OR | MA | NY | ME | Mean, 7 samples | Cape Cod, MA |
| 4-Nonylphenol | 5.830 | 8.550 | 5.430 | 10.500 | 3.950 | 3.740 | 3.820 | 5.974 | 2.73 |
| Nonylphenol monoethoxylate | 14.800 | 9.290 | 8.470 | 4.670 | 6.820 | 5.520 | 3.720 | 7.613 | 2.58 |
| Nonylphenol diethoxylate | 17.900 | 12.300 | 8.730 | 8.180 | 9.340 | 6.930 | 5.850 | 9.890 | 7.87 |
| 4-Octylphenol | <rl | <rl | <rl | <rl | <rl | <rl | <rl | – | 1.00 |
| Octylphenol monoethoxylate | 3.410 | 0.571 | 1.010 | 0.486 | 0.483 | 0.667 | 0.394 | 1.003 | 0.33 |
| Octylphenol diethoxylate | 8.550 | 0.570 | 1.610 | 0.532 | 0.395 | 0.786 | 0.649 | 1.870 | 0.44 |
| 4-t-methylbutylphenol | 0.962 | 0.391 | 0.291 | 0.222 | 0.400 | 0.190 | 0.154 | 0.373 | na |

Cape Cod, MA - Rudel et al (2003) also assayed nonylphenol ethyoxycarboxylate (2940 ppb) but did not assay 4-t-methylbutylphenol

## Pesticides

| Pesticides | µg/g, parts per million (ppm) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | OR | ME | MA | WA | MI | NY | CA | Mean, 7 samples | Cape Cod, MA |
| 4,4'-DDT | 0.0913 | 0.327 | 1.89 | 0.308 | 0.364 | 0.188 | 0.363 | 0.504 | 0.971 |
| alpha-chlordane | <rl | <rl | <rl | <rl | <rl | <rl | <rl | 0.020 | 0.328 |
| gamma-chlordane | <rl | <rl | <rl | <rl | <rl | <rl | 0.138 | 0.020 | 0.383 |
| chlorpyrifos | 0.21 | <rl | <rl | <rl | <rl | <rl | <rl | 0.029 | 2.54 |
| diazinon | <rl | <rl | <rl | <rl | <rl | <rl | 0.140 | | 0.505 |
| dicofol | <rl | <rl | <rl | <rl | <rl | <rl | <rl | | 0.058 |
| dieldrin | <rl | <rl | <rl | <rl | <rl | <rl | 0.720 | 0.103 | 0.132 |
| methoxychlor | <rl | 0.326 | 0.164 | <rl | 0.532 | 0.313 | <rl | 0.334 | 1.08 |
| pentachloronitrobenzene | <rl | <rl | <rl | <rl | <rl | <rl | <rl | | na |
| pentachlorophenol | 0.444 | 7.310 | 0.0481 | 0.0968 | 0.126 | 0.553 | 0.148 | 1.246 | 1.13 |
| cis-permethrin | 11.6 | 2.09 | 3.42 | 2.28 | 2.04 | 1.34 | 0.607 | 3.34 | 2.68 |
| trans-permethrin | 21.0 | 3.52 | 7.67 | 4.52 | 4.17 | 2.67 | 1.31 | 6.41 | 5.03 |
| piperonyl butoxide | 0.572 | 0.325 | 0.345 | 0.553 | 0.147 | 0.705 | 2.18 | 0.69 | 15.8 |
| propoxur | <rl | <rl | <rl | <rl | 0.129 | <rl | 0.130 | 0.037 | 0.691 |

Cape Cod, MA – Rudel et al. (2003) also assayed 4,4'-DDD (0.031 ppm), 4,4'-DDE (0.036 ppm), alachlor (0.002 ppm), aldrin (<rl), atrazine (<rl), bendocarb (0.781 ppm), carbaryl (1.43 ppm), carbofuran (<rl), chlorothalonil (0.153 ppm), 3,5,6-trichloro-2-pridinol (0.987 ppm), cyanazine (<rl), cypermethrin (1.61 ppm), endrin (<rl), heptachlor (0.010 ppm), lindane (0.017), malathion (0.033 ppm), HPTE (<rl), methyl parathion (0.16 ppm), metalachlor (<rl), nitrofen (<rl), parathion (<rl), o-phenylphenol (0.345 ppm), prometon (0.001 ppm), simazine (<rl), and trifluralin (<rl)

## Polybrominated Diphenyl Ethers

| Polybrominated diphenyl ethers | µg/g, parts per million (ppm) | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | OR | WA | CA | MI | MA | NY | ME | Mean, 7 samples | Belgium | Cape Cod, MA | UK | Washington, D.C. |
| BDE 47, a tetraBDE | 1.37 | 5.24 | 3.22 | 1.84 | 1.82 | 0.674 | 0.550 | 2.10 | 0.026 | 0.719 | 0.223 | 1.220 |
| BDE 99, a pentaBDE | 1.06 | 4.13 | 2.67 | 1.27 | 1.68 | 0.595 | 0.474 | 1.70 | 0.054 | 1.290 | 0.044 | 1.700 |
| BDE 100, a pentaBDE | <rl | 0.762 | 0.509 | 0.229 | 0.315 | <rl | <rl | 0.454 | 0.006 | 0.166 | na | 0.274 |
| BDE 153, a hexaBDE | <rl | 0.376 | 0.251 | <rl | <rl | <rl | <rl | 0.314 | 0.002 | 0.538 | 0.034 | 0.181 |
| BDE 154, a hexaBDE | <rl | 0.325 | 0.231 | <rl | <rl | <rl | <rl | 0.296 | <rl | na | na | 0.156 |
| BDE 183, a heptaBDE | <rl | <rl | <rl | <rl | <rl | <rl | <rl | — | <rl | na | 0.192 | 0.031 |
| BDE 209, decaBDE | 10.04 | 0.90 | 3.51 | 6.35 | 5.72 | 3.59 | 2.53 | 4.66 | 4.401 | na | 9.820 | 2.090 |

Belgium—Al Bitar (2004) also assayed octabromodiphenyl ether (no detects), hexabromo cyclododecane (4.8 ppm), and tetrabromobisphenol A (0.068 ppm)

Cape Cod, MA—Rudel et al. (2003) did not assay BDE 154, BDE 183, or BDE 209, but assayed for PCB-52 (0.165 ppm), PCB-105 (0.248 ppm), and PCB-153 (0.538 ppm).

UK—Santillo et al. (2003) assayed BDE-28 (4.14 ppb), hexabromocyclododecane (3158 ppm), and tetrabromobisphenol-A (116 ppm), but did not assay BDE 100 and BDE154.

Washington, D.C.—Stapleton et al. (2005) also assayed BDE-28 (21 ppm).

## Organotins

| Organotins | ng/g, parts per billion (ppb) | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | ME | OR | WA | MI | MA | NY | CA | Mean, 7 samples | Belgium | Brazil | Germany |
| Monobutyltin | 205.1 | 361.4 | 227.8 | 219.2 | 106.0 | 184.8 | 140.1 | 206.3 | 567.0 | 90.0 | 160.0 |
| Dibutyltin | 314.7 | 321.5 | 204.2 | 236.6 | 265.4 | 186.7 | 115.8 | 235.0 | 1417.0 | 252.5 | 510.0 |
| Tributyltin | 193.1 | 44.8 | 99.4 | 49.2 | 55.6 | 71.7 | 44.7 | 79.8 | 280.0 | 56.7 | 20.0 |
| Tetrabutyltin | <rl | <rl | <rl | <rl | <rl | <rl | <rl | – | | | |
| Di-n-octyltin | 198.5 | 76.4 | 108.8 | 71.7 | 128.8 | 95.8 | 87.6 | 109.7 | 113.0 | 187.5 | 20.0 |
| Tricyclohexyltin | <rl | <rl | <rl | <rl | <rl | <rl | <rl | – | na | na | na |
| Triphenyltin | <rl | <rl | <rl | <rl | <rl | <rl | <rl | – | na | 17.5 | na |

Germany—Fromme et al (2005) also assayed monooctyltin (10 ppb) but did not assay tricyclohexyltin or triphenyltin

Belgium—Al Bitar et al. (2004) also assayed monooctyltin and tetrabutyltin but did not assay tricylclohexyltin or triphenyltin

Brazil—Costner et al (2004) also assayed monooctyltin (62.5 ppb) and tetrabutytin (zero detects) but did not assay tricyclohexyltin

U.K.—Santillo et al.(2003)also assayed monooctyltin (450.6 ppb) and tetrabutyltin (zero detects).

## Perfluorinated Chemicals

| Perfluorinated Chemicals | ng/g, parts per billion (ppb) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | CA | ME | MA | MI | NY | OR | WA | Mean, 7 samples | Japan |
| PFOA | 127.8 | 66.5 | 18.5 | 68.3 | 31 | 33.5 | 205.1 | 78.7 | 380 |
| PFOS | 413.5 | 76.4 | 104.8 | 243.8 | 431.1 | 1170.9 | 530 | 424.4 | 200 |

## ENDNOTES


1 Betts, K., 2003. Are US Homes a Haven for Toxics? Environ. Sci. Technol. 407A-411A. Nov. 1, 2003.

2 Nerin, C., Asensio, E., Jimenez, C., 2002. Supercritical fluid extraction of potential migrants from paper and board intended for use as food packaging materials. Anal. Chem. 74: 5831-5836.

3 Betts, K., 2003. Are US Homes a Haven for Toxics? Environ. Sci. Technol. 407A-411A. Nov. 1, 2003.

4 Rudel in Betts, K., 2003. Are US Homes a Haven for Toxics? Environ. Sci. Technol. 407A-411A. Nov. 1, 2003.

5 Butt, C., Diamong, M., Truong, J., 2004. Spatial distribution of polybrominated diphenyl ethers in southern Ontario as measured in indoor and outdoor window organic films. Environ. Sci. Technol.2004, 38,724-731.

6 American Chemistry Council's Brominated Flame Retardant Industry Panel for the Voluntary Children's Chemical Evaluation Program, 2003. Submission to Report of the Peer Consultation Meeting on Decabromodiphenyl Ether. 2-3 April, 2003, Cincinnati, Ohio.

7 Fromme, H., Mattulat, A., Lahrz, T., Ruden, H., 2004. Occurrence of organotin compounds in house dust in Berlin (Germany). Chemosphere. In Press

8 Fromme, H., Mattulat, A., Lahrz, T., Ruden, H., 2004. Occurrence of organotin compounds in house dust in Berlin (Germany). Chemosphere. In Press

9 Key, B., Howell, R., Criddle, C., 1997. Fluorinated organics in the biosphere. Environ. Sci. Technol. 31: 2445-2454.

10 Kissa, E., "Fluorinated Surfactants." Dekker, New York, 1994.

11 Moriwaki, H., Takata, Y., Arakawa, R., 2003. Concentrations of perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOS) in vacuum clean dust collected in Japanese homes. J. Environ. Monit. 5: 753-757.

12 BNA. 2001. Environment and Safety Library on the Web. States and Territories. Washington, D.C: http//www.esweb.bna.com/. Bureau of National Affairs, Inc.

13 Al Bitar, F., 2004. Hazardous chemicals in Belgian house dust. Brussels: Greenpeace Belgium.

14 Costner, P., Butcher, J., Peters, R., 2004. Hazardous Chemicals in Brazilian House and Office Dust. Sao Paulo: Greenpeace Brazil.

15 Rudel, R., Camann, D., Spengler, J., Korn, L., Brody, J., 2003. Phthalates, alkylphenols, pesticides, polybrominated diphenyl ethers, and other endocrine-disrupting compounds in indoor air and dust. Environ. Sci. Technol. 37: 4543-4553.

16 Santillo, D., Labunska, I., Davidson, H., Johnston, P., Strutt, M., Knowles, O., 2003. Consuming chemicals: Hazardous chemicals in house dust as an indicator of chemical exposure in the home. GRL-TN-01-2003. Exeter, UK: Greenpeace Research Laboratories.

17 Bornehag, C.-G., Sundell, J., Weschler, C., Sigsgaard, T., Lundgren,B., Hasselgren, M., Hagerhed-Engman, L., 2004. The association between asthma and allergic symptoms in children and phthalates in house dust: A nested case–control study. Environ Health Perspect 112: 1393–1397

18 Bizzari, S., Gubler, R., Kishi, A., 2003. CEH Report: Plasticizers. http://ceh.sric.sri.com/Public/Reports/576.0000/. Accessed 4 February 2005.

19 Bornehag, C.-G., Sundell, J., Weschler, C., Sigsgaard, T., Lundgren,B., Hasselgren, M., Hagerhed-Engman, L., 2004. The association between asthma and allergic symptoms in children and phthalates in house dust: A nested case–control study. Environ Health Perspect 112: 1393–1397

20 Langenkamp, H., Marmo, L. (eds), 2000. Proceedings: Workshop on Problems Around Sludge, 18-29 November 1999, Stresa, Italy. EUR 19657 EN, European Commission Joint Research Center.

21 U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry (ATSDR), 2002. Toxicological Profile for Di(2-ethylhexyl)phthalate. Atlanta, GA: Agency for Toxic Substances and Disease Registry.

22 Centers for Disease Control and Prevention, 2001. National Report on Human Exposure to Environmental Chemicals.

23 Houlihan, J,, Wiles, R., 2000. Beauty Secrets: Does a common chemical in nail polish pose risks to human health? Environmental Working Group

24 Phthalate Esters Panel. How are phthalates used? American Chemistry Council. http://www.phthalates.org. 2002.

25 Kenny, A.., Thomas, K., Peeler, E., Osborn, D., Waldock, M., Matthiessen, P., Shore, R., Wright, J. and Feist, S. 2002). Assessing the potential of environmental monitoring for detecting and responding to damage caused by the use of chemicals. Report by the Centre for Environment, Fisheries and Aquaculture Science (CEFAS) for the Royal Commission on Environmental Pollution.

26 Clausen, P., Hansen, V., Gunnarsen, L., Afshari, A., Wolkoff, P., 2004. Emission of Di-2-ethyhexyl phthalate from PVC flooring into air and uptake into dust: emission and sorption experiments in FLEC and CLIMPAQ. Environ. Sci. Technol. 38: 2531-2537

27 Afshari, A., Gunnarsen, L., Clausen, P., Hansen, V., 2004. Emission of phthalates from PVC and other materials. Indoor Air 14: 120-128.

28 Dorey, C., 2003. Chemical Legacy Contamination of the Child. London: Greenpeace UK

29 Health Care Without Harm, 2002. Aggregate Exposures to Phthalates in Humans. Arlington, VA: Health Care Without Harm. www.noharm.org/details.cfm?type=document&id=796

30 Rudel, R., Camann, D., Spengler, J., Korn, L., Brody, J., Phthalates, alkylphenols, pesticides, polybrominated diphenyll ethers, and other endocrine-disrupting compounds in indoor air and dust. Environ. Sci. Technol. 2003. 37: 4543-4554.

31 Centers for Disease Control and Prevention. 2003. Second National Report on Human Exposure to Environmental Chemicals. NCEH Pub. No. 02-0716.

32 Koch, H., Drexler, H., Angerer, J., 2003. An estimation of the daily intake of di(2-ethylhexyl)phthalate (DEHP) and other phthalates in the general population. Int. J. Hyg. Environ. Health 206: 77-83.

33 Koch, H., Drexler, H., Angerer, J., 2004. Internal exposure of nursery-school children and their parents and teachers to di(2-ethylhexyl)phthalate (DEHP). Int. J. Hyg. Environ. Health 207(1):15-22.

34 Adibi, J., Perera, F., Jedrychowski, W., Camann, D., Barr, D., Jacek, R., Whyatt, R., 2003. Prenatal exposures to phthalates among women in New York City and Krakow, Poland. Environ. Health Perspect. 111:1719–1722.

35 Latini, G., De Felice, C., Presta, G., Del Vecchio, A., Paris, I., Ruggieri, F., Mazzeo, P., 2003. In utero exposure to di-(2-ethylhexyl)phthalate and duration of human pregnancy. Environ. Health Perspect. 111. 1783-1785.

36 Calafat, A., Slakman, A., Silva, M., Herbert, A., Needham, L., 2004. Automated solid phase extraction and quantitative analysis of human milk for 13 phthalate metabolites. J. Chromatog. B, 805: 49-56.

37 Dostal et al., 1987

38 Parmar et al., 1985

39 Srivasta et al., 1989

40 Jaakkola, J.J.K., Øie, L., Nafstad, P., Botten, G., Lødrup-Carlson, K.C., Samuelsen, S.O. and Magnus, P. (1999) Interior surface materials in the home and the development of bronchial obstruction in young children in Oslo, Norway, Am. J. Public Health, 89, 188–192.

41 Øie, L., Hersoug, L.G. and Madsen, J.Ø. (1997)Residential exposure to plasticizers and its possible role in the pathogenesis of asthma, Environ. Health. Perspect., 105, 972–978.

42 Øie, L., Hersoug, L.G. and Madsen, J.Ø. (1997)Residential exposure to plasticizers and its possible role in the pathogenesis of asthma, Environ. Health. Perspect., 105, 972–978.

43 Bornehag, C.-G., Sundell, J., Weschler, C., Sigsgaard, T., Lundgren, B., Hasselgren, M., Hagerhed-Engman, L., 2004. The association between asthma and allergic symptoms in children and phthalates in house dust: A nested case-control study. Environ Health Perspect 112:1393–1397.

44 Colon, I., Caro, D., Bourdony, C. J. & Rosario, O. ( 2000) Identification of phthalate esters in the serum of young Puerto Rican girls with premature breast development. Environmental Health Perspectives 108( 9): 895- 900

45 Duty, S., Silva, M., Barr, D., Brock, J., Ryan, L., Zuying, C., Herrick, R., Christiani, D., Hauser, R., 2002. Urinary phthalate monoesters at general populations exposure levels are associated with altered semen quality. Epidemiology July 2002, Volume 13 Number 4 Supplement

46 Duty, S., Singh, N., Silva, M., Barr, D., Brock, J., Ryan, L., Herrick, R., Christiani, D., Hauser, R., 2003. The relationship between environmental exposures to phthalates and DNA damage in human sperm using the Neutral Comet Assay. Environ. Health Perspect. 111: 1164-1169.

47 Rozati, R., Reddy, R., Reddanna, P., Mujtaba, R., 2002. Role of environmental estrogens in the deterioration of male factor fertility. Fertility & Sterility 78: 1187-1194

48 Moore RW, Rudy TA, Lin TM, Ko K, Peterson RE. 2001. Abnormalities of sexual development in male rats with in utero and lactational exposure to the antiandrogenic plasticizer di(2-ethylhexyl) phthalate. Environ Health Perspect 109:229-237.

49 Mylchreest E, Cattley RC, Foster PMD. 1998. Male reproductive tract malformations in rats following gestational and lactational exposure to di(n-butyl) phthalate: Anantiandrogenic mechanism? Toxicol Sci 43:47-60.

50 Parks LG, Ostby JS, Lambright CR, Abbott BD, Klinefelter GR, Barlow NJ, et al. 2000.The plasticizer diethylhexyl phthalate induces malformations by decreasing fetal testosterone synthesis during sexual differentiation in the male rat. Toxicol Sci 58:339- 349.

51 Agarwal DK, Eustis S, Lamb JC, Reel JR, Kluwe WM. 1986. Effects of di(2-ethylhexyl) phthalate on the gonadal pathophysiology, sperm morpho ogy, and reproductive-performance of male-rats. Environ Health Perspect 65: 343–350.

52 Ema M, Miyawaki E. 2001. Adverse effects on development of the reproductive system in male offspring of rats given monobutyl phthalate, a metabolite of dibutyl phthalate, during late pregnancy. Reprod Toxicol 15: 189-194.

53 Foster PMD, Mylchreest E, Gaido KW, Sar M. 2001. Effects of phthalate esters on the developing reproductive tract of male rats. Human Reprod Update 7:231-235.

54 Mylchreest E, Wallace DG, Cattley RC, Foster PMD. 2000. Dose-dependent alterations in androgen-regulated male reproductive development in rats exposed to di(n-butyl) phthalate during late gestation. Toxicol Sci 55:143-151.

55 CSTEE (2000). CSTEE opinion on lead - Danish notification 98/595/DK. The Scientific Committee on Toxicity, Ecotoxicity and the Environment (CSTEE) of DG XXIV, Consumer Policy and Consumer Health Protection, Brussels, 5th May 2000.

56 WHO (1997a). Di-n-butyl phthalate. Environmental Health Criteria 189. World Health Organisation, International Programme on Chemical Safety, Geneva.

57 Nielsen E and Larsen PB (1996). Toxicological evaluation and limit values for DEHP and phthalates other than DEHP. Environmental Review No. 6 1996, Danish Environmental Protection Agency.

58 Mylchreest E and Foster PMD (1998). Antiandrogenic effects of di(nbutyl) phthalate on male reproductive development: a nonreceptormediated mechanism. CIIT 18(9).

59 CSTEE (1998). CSTEE opinion on phthalate migration from soft PVC toys and childcare articles. The Scientific Committee on Toxicity, Ecotoxicity and the Environment (CSTEE) of DG XXIV, Consumer Policy and Consumer Health Protection. November 1998.

60 Nielsen E and Larsen PB (1996). Toxicological evaluation and limit values for DEHP and phthalates other than DEHP. Environmental Review No. 6 1996, Danish Environmental Protection Agency.

61 WHO (1997a). Di-n-butyl phthalate. Environmental Health Criteria 189. World Health Organisation, International Programme on Chemical Safety, Geneva.

62 Mylchreest E and Foster PMD (1998). Antiandrogenic effects of di(nbutyl) phthalate on male reproductive development: a nonreceptormediated mechanism. CIIT 18(9).

63 Ema M, Miyawaki E. 2001. Adverse effects on development of the reproductive system in male offspring of rats given monobutyl phthalate, a metabolite of dibutyl phthalate, during late pregnancy. Reprod Toxicol 15: 189-194.

64 Mylchreest E, Cattley RC, Foster PMD. 1998. Male reproductive tract malformations in rats following gestational and lactational exposure to di(n-butyl) phthalate: An antiandrogenic mechanism? Toxicol Sci 43:47-60.

65 Mylchreest E, Sar M, Wallace DG, Foster PMD. 2002. Fetal testosterone insufficiency and abnormal proliferation of Leydig cells and gonocytes in rats exposed to di(n-butyl)phthalate. Reprod Toxicol 16:19-28.

66 ATSDR. 1999. Toxicological Profile for Di-n-butyl phthalate. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry.

67 Mylchreest E, Cattley RC, Foster PMD. 1998. Male reproductive tract malformations in rats following gestational and lactational exposure to di(n-butyl) phthalate: An antiandrogenic mechanism? Toxicol Sci 43:47-60.

68 ATSDR. 2000. Toxicological Profile for Di(2-ethylhexyl)phthalate. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry.

69 Gray TJB, Beamand JA. 1984. Effect of some phthalate esters and other testicular toxins on primary cultures of testicular cells. Food Chem Toxicol 22:123-131.

70 Gray TJB, Gangolli SD. 1986. Aspects of the testicular toxicity of phthalate-esters. Environ Health Perspect 65:229-235.

71 Davis BJ, Maronpot RR, Heindel JJ. 1994. Di-(2-ethylhexyl) phthalate suppresses estradiol and ovulation in cycling rats. Toxicol Appl Pharmacol 128:216-223.

72 Lovekamp- Swan T, Davis BJ ( 2003). Mechanisms of phthalate ester toxicity in the female reproductive system. Environ Health Perspect; 111( 2): 139–45

73 ATSDR. 2000. Toxicological Profile for Di(2-ethylhexyl) phthalate. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry.

74 Huber WW, GraslKraupp B, SchulteHermann R. 1996. Hepatocarcinogenic potential of di(2 ethylhexyl) phthalate in rodents and its implications on human risk. Crit Revs Toxicol 26:365-481.

75 Kluwe WM, Mcconnell EE, Huff JE, Haseman JK, Douglas JF, Hartwell WV. 1982. Carcinogenicity testing of phthalate-esters and related-compounds by the National Toxicology Program and the National Cancer Institute. Environ Health Perspect 45:129–133.

76 ATSDR. 2000. Toxicological Profile for Di(2-ethylhexyl) phthalate. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry.

77 Doull J, Cattley R, Elcombe C, Lake BG, Swenberg J, Wilkinson C, et al. 1999. A cancer risk assessment of di (2-ethylhexyl)phthalate: Application of the new US EPA risk assessment guidelines. Regul Toxicol Pharmacol 29: 327-357.

78 Melnick, RL. 2001. Is peroxisome proliferation an obligatory precursor step in the carcinogenicity of di-(2-ethylhexyl)phthalate (DEHP)?. Environ Health Perspect 109:437-442.

79 Barr et al., 2003; Dana B. Barr, Manori J. Silva, Kayoko Kato, John A. Reidy, Nicole A. Malek, Donald Hurtz, Melissa Sadowski, Larry L. Needham, and Antonia M. Calafat Assessing Human Exposure to Phthalates Using Monoesters and Their Oxidized Metabolites as Biomarkers Environ Health Perspect 111:1148-1151 (2003).

80 U. S. International Trade Commission, 1995. Synthetic organic chemicals U. S. production and sales. U. S. International Trade Commission Publication 2933. Washington, DC.

81 APE Research Council, 1999. Alkylphenols and Alkylphenol Ethoxylates: An Overview of Safety Issues. http://www.aperc.org/. Accessed 8 Jan 2005.

82 Loo TW, Clarke DM (1998). Nonylphenol ethoxylates, but not nonylphenol, are substrates of the human multidrug resistance P-glycoprotein. Biochem Biophys Res Commun;247(2):478–80.

83 APE Research Council, 1999. Alkylphenols and Alkylphenol Ethoxylates: An Overview of Safety Issues. http://www.aperc.org/. Accessed 8 Jan 2005.

84 Guenther, K., Heinke, V., Thiele, B., Kleist, E., Prast, H., Raecker, T., 2002. Endocrine disrupting nonylphenols are ubiquitous in food. Environ. Sci. Technol. 36:1676–80.

85 Bogh, I., Christensen, P., Dantzer, V., Groot, M., Thofner, I., Rasmussen, R., Schmidt, M., Greve, T., 2001. Endocrine disrupting compounds: Effect of octylphenol on reproduction over three generations. Theriogenology 55: 131-150

86 K. Inoue, S. Kondo, Y. Yoshie, K. Kato, Y. Yoshimura, M. Horie, H. Nakazawa, Food Addit. Contam. 18 (2001) 157.

87 K. Inoue, N. Kobayashi, Y. Yoshimura, M. Horie, H. Nakazawa, Jpn. J. Food Chem. 9 (2002) 46.

88 T. Isobe, N. Nakada, Y. Mato, H. Nishiyama, H. Kumata, H. Takada, Jpn. J. Environ. Chem. 12 (2002) 621.

89 Y. Kawamura, T. Maehara, H. Iijima, T. Yamada, Jpn. J. Food Hyg. Soc. 41 (2000) 212.

90 Guenther, K., Heinke, V., Thiele, B., Kleist, E., Prast, H., Raecker, T., 2002. Endocrine disrupting nonylphenols are ubiquitous in food. Environ. Sci. Technol. 36:1676–80.

91 Guenther, K., Heinke, V., Thiele, B., Kleist, E., Prast, H., Raecker, T., 2002. Endocrine disrupting nonylphenols are ubiquitous in food. Environ. Sci. Technol. 36 ( 8), 1676–1680.

92 Toppari J, Larsen JC, Christiansen P, Giwercman A, Grandjean P, Guillette LJ Jr, Jegou B, Jensen TK, Jouannet P, Keiding N, et al. Male reproductive health and environmental xenoestrogens. Environ Health Perspect 104( suppl 4): 741 – 803 ( 1996)

93 Centers for Disease Control and Prevention, 2001. National Report on Human Exposure to Environmental Chemicals.

94 Centers for Disease Control and Prevention. 2003. Second National Report on Human Exposure to Environmental Chemicals. NCEH Pub. No. 02-0716.

95 Centers for Disease Control and Prevention, 2005. Third National Report on Human Exposure to Environmental Chemicals. In Press.

96 Calafat, A., Kuklenyik, Z., Reidy, J., Caudill, S., Ekong, J., Needham, L., 2005. Urinary concentrations of bisphenol A and 4- nonylphenol in a human reference population. Environ. Health Perspect. In Press.

97 Kawaguchi, M., Inoue, K., Sakui, N., Ito, R., Izume, S.-i., Makino, T., Okanouchi, N., Nakazawa, H., 2004. Stir bar sorptive extraction and thermal desorption-gas chromatography-mass spectrometry for the measurement of 4-nonylphenol and 4-tert-octylphenol in human biological samples. J. Chromatogr. B 799 : 119-125.

98 K. Inoue, M. Kawaguchi, F. Okada, N. Takai, Y. Yoshimura, M. Horie, S. Izumi, T. Makino, H. Nakazawa, Anal. Chim. Acta 486 (2003) 41. [In Kawaguchi]

99 Takada H, Isobe T, Nakada N, Nishiyama H, Iguchi T, Irie H, Mori C (1999). Bisphenol A and nonylphenols in human umbilical c o rds. Proceedings of the International Scientific Conference on Environmental Endocrine Disrupting Chemicals, Monte Verita, Ascona, Switzerland, March 7–12, 1999.

100 Guenther, K., Heinke, V., Thiele, B., Kleist, E., Prast, H., Raecker, T., 2002. Endocrine disrupting nonylphenols are ubiquitous in food. Environ. Sci. Technol. 36:1676–80.

101 Loo TW, Clarke DM (1998). Nonylphenol ethoxylates, but not nonylphenol, are substrates of the human multidrug resistance P-glycoprotein. Biochem Biophys Res Commun;247(2):478–80.

102 Blake CA, Boockfor FR. Chronic administration of the environmental pollutant 4-tert-octylphenol to adult male rats interferes with the secretion of luteinizing hormone, follicle-stimulating hormone, prolactin, and testosterone. Biol Reprod 1997;57:255–66.

103 Bogh, I., Christensen, P., Dantzer, V., Groot, M., Thofner, I., Rasmussen, R., Schmidt, M., Greve, T., 2001. Endocrine disrupting compounds: Effect of octylphenol on reproduction over three generations. Theriogenology 55: 131-150

104 Lee HJ, Chattopadhyay S, Gong EY, Ahn RS, Lee K (2003a). Antiandrogenic effects of bisphenol A and nonylphenol on the function of androgen receptor. Toxicol Sci;75(1):40–6.

105 Paris F, Balaguer P, Terouanne B, Servant N, Lacoste C, Cravedi JP, Nicolas JC, Sultan C (2002). Phenylphenols, biphenols, bisphenol-A and 4-tertoctylphenol exhibit alpha and beta estrogen activities and antiandrogen activity in reporter cell lines. Mol Cell Endocrinol; 193(1–2):43–9.

106 Nagel SC, vom Saal FS, Thayer KA, Dhar MG, Boechler M, Welshons WV (1997). Relative binding affinity-serum modified access (RBA-SMA) assay predicts the relative in vivo bioactivity of the xenoestrogens bisphenol A and octylphenol. Environ Health Perspect;105(1):70–6

107 Nagel SC, vom Saal FS, Welshons WV (1999). Developmental effects of estrogenic chemicals are predicted by an in vitro assay incorporating modification of cell uptake by serum. J Steroid Biochem Mol Biol;69(1–6): 343–57.

108 Sharpe RM, Fisher JS, Millar MM, Jobling S, Sumpter JP (1995). Gestational and lactational exposure of rats to xenoestrogens results in reduced testicular size and sperm production. Environ Health Perspect;103 (12): 1136–43.109 Kyselova V, Peknicova J, Buckiova D, Boubelik M (2003). Effects of pnonylphenol and resveratrol on body and organ weight and in vivo fertility of outbred CD-1 mice.Reprod Biol Endocrinol; 1(1):30.

110 Inadera H, Sekiya T, Yoshimura T, Matsushima K (2000). Molecular analysis of the inhibition of monocyte chemoattractant protein-1 gene expression by estrogens and xenoestrogens in MCF-7 cells. Endocrinology;141(1):50–9.

111 Lee MH, Chung SW, Kang BY, Park J, Lee CH, Hwang SY, Kim TS (2003b). Enhanced interleukin-4 production in CD4+ T cells and elevated immunoglobulin E levels in antigen-primed mice by bisphenol A and nonylphenol, endocrine disruptors: Involvement of nuclear factor-AT and Ca2+. Immunology;109(1): 76–86.

112 Pocock VJ, Sales GD, Wilson CA, Milligan SR. Effects of perinatal octylphenol on ultrasound vocalization, behavior and reproductive physiology in rats. Physiol Behav 2002;76:645–53. As cited in Haavisto, T., Adamsson, N., Myllymaki, S., Toppari, J., Paranko, J., 2003. Effects of 4-tert-octyphenol, 4-tert-butylphenol, and diethylstilbestrol on prenatal testosterone surge in the rat. Reprod. Toxicol. 17: 593-605.

113 Herath CB, Watanabe G, Katsuda Si, Yoshida M, Suzuki AK, Taya K. Exposure of neonatal female rats to p-tert-octylphenol disrupts afternoon surges of luteinizing hormone, follicle-stimulating hormone and prolactin secretion, and interferes with sexual receptive behavior in adulthood. Biol Reprod 2001;64:1216–24. As cited in Haavisto, T., Adamsson, N., Myllymaki, S., Toppari, J., Paranko, J., 2003. Effects of 4-tert-octyphenol, 4-tert-butylphenol, and diethylstilbestrol on prenatal testosterone surge in the rat. Reprod. Toxicol. 17: 593-605.

114 Katsuda S, Yoshida M,Watanabe G, Taya K, Maekawa A. Irreversible effects of neonatal exposure to p-tert-octylphenol on the reproductive tract in female rats. Toxicol Appl Pharmacol 2000;165:217–26. As cited in Haavisto, T., Adamsson, N., Myllymaki, S., Toppari, J., Paranko, J., 2003. Effects of 4-tert-octyphenol, 4-tert-butylphenol, and diethylstilbestrol on prenatal testosterone surge in the rat. Reprod. Toxicol. 17: 593-605.

115 Khurana S, Ranmal S, Ben Jonathan N. Exposure of newborn male and female rats to environmental estrogens: delayed and sustained hyperprolactinemia and alterations in estrogen receptor expression. Endocrinology 2000;141:4512–7. As cited in Haavisto, T., Adamsson, N., Myllymaki, S., Toppari, J., Paranko, J., 2003. Effects of 4-tert-octyphenol, 4-tert-butylphenol, and

diethylstilbestrol on prenatal testosterone surge in the rat. Reprod. Toxicol. 17: 593-605. As cited in Haavisto, T., Adamsson, N., Myllymaki, S., Toppari, J., Paranko, J., 2003. Effects of 4-tert-octyphenol, 4-tert-butylphenol, and diethylstilbestrol on prenatal testosterone surge in the rat. Reprod. Toxicol. 17: 593-605.

116 Saunders PT, Majdic G, Parte P, Millar MR, Fisher JS, Turner KJ, et al. Fetal and perinatal influence of xenoestrogens on testis gene expression. Adv Exp Med Biol 1997;424:99–110.

117 Yoshida M, Katsuda S, Takenaka A, Watanabe G, Taya K, Maekawa A. Effects of neonatal exposure to a high-dose p-tert-octylphenol on the male reproductive tract in rats. Toxicol Lett 2001;121:21–33. As cited in Haavisto, T., Adamsson, N., Myllymaki, S., Toppari, J., Paranko, J., 2003. Effects of 4-tert-octyphenol, 4-tert-butylphenol, and diethylstilbestrol on prenatal testosterone surge in the rat. Reprod. Toxicol. 17: 593-605.

118 Fisher JS, Turner KJ, Brown D, Sharpe RM. Effect of neonatal exposure to estrogenic compounds on development of the excurrent ducts of the rat testis through puberty to adulthood. Environ Health Perspect 1999; 107:397–405. As cited in Haavisto, T., Adamsson, N., Myllymaki, S., Toppari, J., Paranko, J., 2003. Effects of 4-tert-octyphenol, 4-tert-butylphenol, and diethylstilbestrol on prenatal testosterone surge in the rat. Reprod. Toxicol. 17: 593-605.

119 Nagao T, Yoshimura S, Saito Y, Nakagomi M, Usumi K, Ono H. Reproductive effects in male and female rats from neonatal exposure to p-octylphenol. Reprod Toxicol 2001;15:683–92.

120 Blake CA, Boockfor FR. Chronic administration of the environmental pollutant 4-tert-octylphenol to adult male rats interferes with the secretion of luteinizing hormone, follicle-stimulating hormone, prolactin, and testosterone. Biol Reprod 1997;57:255–66. As cited in Haavisto, T., Adamsson, N., Myllymaki, S., Toppari, J., Paranko, J., 2003. Effects of 4-tert-octyphenol, 4-tert-butylphenol, and diethylstilbestrol on prenatal testosterone surge in the rat. Reprod. Toxicol. 17: 593-605.

121 Boockfor FR, Blake CA. Chronic administration of 4-tert-octylphenol to adult male rats causes shrinkage of the testes and male accessory sex organs, disrupts spermatogenesis, and increases the incidence of sperm deformities. Biol Reprod 1997;57:267–77. As cited in Haavisto, T., Adamsson, N., Myllymaki, S., Toppari, J., Paranko, J., 2003. Effects of 4-tert-octyphenol, 4-tert-butylphenol, and diethylstilbestrol on prenatal testosterone surge in the rat. Reprod. Toxicol. 17: 593-605.

122 Yoshida M, Katsuda S, Ando J, Kuroda H, Takahashi M, Maekawa A. Subcutaneous treatment of p-tert-octylphenol exerts estrogenic activity on the female reproductive tract in normal cycling rats of two different strains. Toxicol Lett 2000;116:89–101. As cited in Haavisto, T., Adamsson, N., Myllymaki, S., Toppari, J., Paranko, J., 2003. Effects of 4-tert-octyphenol, 4-tert-butylphenol, and diethylstilbestrol on prenatal testosterone surge in the rat. Reprod. Toxicol. 17: 593-605.

123 Blake CA, Boockfor FR. Chronic administration of the environmental pollutant 4-tert-octylphenol to adult male rats interferes with the secretion of luteinizing hormone, follicle-stimulating hormone, prolactin, and testosterone. Biol Reprod 1997;57:255–66. As cited in Haavisto, T., Adamsson, N., Myllymaki, S., Toppari, J., Paranko, J., 2003. Effects of 4-tert-octyphenol, 4-tert-butylphenol, and diethylstilbestrol on prenatal testosterone surge in the rat. Reprod. Toxicol. 17: 593-605.

124 Khurana S, Ranmal S, Ben Jonathan N. Exposure of newborn male and female rats to environmental estrogens: delayed and sustained hyperprolactinemia and alterations in estrogen receptor expression. Endocrinology 2000;141:4512–7. As cited in Haavisto, T., Adamsson, N., Myllymaki, S., Toppari, J., Paranko, J., 2003. Effects of 4-tert-octyphenol, 4-tert-butylphenol, and diethylstilbestrol on prenatal testosterone surge in the rat. Reprod. Toxicol. 17: 593-605.

125 Sweeney T, Nicol L, Roche JF, Brooks AN. Maternal exposure to octylphenol suppresses ovine fetal follicle-stimulating hormone secretion, testis size, and Sertoli cell number. Endocrinology 2000;141:2667–73. As cited in Haavisto, T., Adamsson, N., Myllymaki, S., Toppari, J., Paranko, J., 2003. Effects of 4-tert-octyphenol, 4-tert-butylphenol, and diethylstilbestrol on prenatal testosterone surge in the rat. Reprod. Toxicol. 17: 593-605.

126 Toft G, Baatrup E. Sexual characteristics are altered by 4-tert-octylphenol and 17beta-estradiol in the adult male guppy (Poecilia reticulata). Ecotoxicol Environ Saf 2001;48:76–84. As cited in Haavisto, T., Adamsson, N., Myllymaki, S., Toppari, J., Paranko, J., 2003. Effects of 4-tert-octyphenol, 4-tert-butylphenol, and diethylstilbestrol on prenatal testosterone surge in the rat. Reprod. Toxicol. 17: 593-605.

127 Paris F, Balaguer P, Terouanne B, Servant N, Lacoste C, Cravedi JP, et al. Phenylphenols, biphenols, bisphenol-A and 4-tert-octylphenol exhibit [alpha] and [beta] estrogen activities and antiandrogen activity in reporter cell lines. Mol Cell Endocrinol 2002;193:43–9. As cited in Haavisto, T., Adamsson, N., Myllymaki, S., Toppari, J., Paranko, J., 2003. Effects of 4-tert-octyphenol, 4-tert-butylphenol, and diethylstilbestrol on prenatal testosterone surge in the rat. Reprod. Toxicol. 17: 593-605.

128 Knudsen FR, Arukwe A, Pottinger TG. The in vivo effect of combinations of octylphenol, butylbenzylphthalate and estradiol on liver estradiol receptor modulation and induction of zona radiate proteins in rainbow trout: no evidence of synergy. Environ Pollut 1998;103: 75–80. As cited in Haavisto, T., Adamsson, N., Myllymaki, S., Toppari, J., Paranko, J., 2003. Effects of 4-tert-octyphenol, 4-tert-butylphenol, and diethylstilbestrol on prenatal testosterone surge in the rat. Reprod. Toxicol. 17: 593-605.

129 Bendsen E, Laursen S, Olesen C,Westergaard L, Andersen C, Byskov A. Effect of 4-octylphenol on germ cell number in cultured human fetal gonads. Hum Reprod 2001;16:236–43. As cited in Haavisto, T., Adamsson, N., Myllymaki, S., Toppari, J., Paranko, J., 2003. Effects of 4-tert-octyphenol, 4-tert-butylphenol, and diethylstilbestrol on prenatal testosterone surge in the rat. Reprod. Toxicol. 17: 593-605.

130 Abraham EJ, Frawley LS. Octylphenol (OP), an environmental estrogen, stimulates prolactin (PRL) gene expression. Life Sci 1997; 60:1457–65. As cited in Haavisto, T., Adamsson, N., Myllymaki, S., Toppari, J., Paranko, J., 2003. Effects of 4-tert-octyphenol, 4-tert-butylphenol, and diethylstilbestrol on prenatal testosterone surge in the rat. Reprod. Toxicol. 17: 593-605.

131 Bogh, I., Christensen, P., Dantzer, V., Groot, M., Thofner, I., Rasmussen, R., Schmidt, M., Greve, T., 2001. Endocrine disrupting compounds: Effect of octylphenol on reproduction over three generations. Theriogenology 55: 131-150

132 U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry (ATSDR), 2003. Toxicological Profile for Pyrethrins and Pyrethroids. Atlanta, GA: Agency for Toxic Substances and Disease Registry.

133 Whyatt, R., Camann, D., Kinney, P, Reyes, A., Ramirez, J., Dietrich, J., Diaz, D., Holmes, D., Perera, F., 2002. Residential pesticide use during pregnancy among a cohort of urban minority women. Environ. Health Persp. 110: 507-514.

134 Rudel, R., Camann, D., Spengler, J., Korn, L., Brody, J., 2003. Phthalates, alkylphenols, pesticides, polybrominated diphenyl ethers, and other endocrine-disrupting compounds in indoor air and dust. Environ. Sci. Technol. 37: 4543-4553.

135 World Health Organization. 1990. Permethrin. Environmental Health Criteria 94. Geneva, Switzerland: World Health Organization, United Nations Environment Programme, and International Labor Organization. pp.25–26.

136 Gianessi, L.P. and J.E. Anderson. 1995. Pesticide use in U.S. crop production. National summary report. Washington, D.C.: National Center for Food and Agricultural Policy.

137 Food and Drug Administration. Pesticide program: Residue monitoring 2001. http://www.cfsan.fda.gov/~dms/pes01rep.html#level

138 Eil, C. and B.C. Nisula. 1990. The binding properties of pyrethroids to human skin fibroblast androgen receptors and to sex hormone binding globulin. J. Steroid Biochem. 35:409-414. Quoted in Caroline Cox. Permethrin factsheet. Journal of Pesticide Reform/Summer 1998: Vol.18, No. 214l

139 Stelzer, K.J. and M.A. Gordon. 1984. Effects of pyrethroids on lymphocyte mitogenic responsiveness. Res. Comm. Chem. Pathol. Pharmacol. 46(1):137-150. Quoted in Cox. ibid

140 Cantalamessa, F. 1993. Acute toxicity of two pyrethroids, permethrin and cypermethrin, in neo-natal and adult rats. Arch. Toxicol. 67:510-513.

141 World Health Organization. 1990. Permethrin. Environmental Health Criteria 94. Geneva, Switzerland: World Health Organization, United Nations Environment Programme, and International Labor Organization. Pp.64.

142 Barrueco, C. et al. 1992. Cytogenetic effects of permethrin in cultured lymphocytes. Mutag. 7:433-437.

143 Surrallés, J. et al. 1995. The suitability of the micronucleus assay in human lymphocytes as a new biomarker of excision repair. Mut. Res. 342:43-59.

144 U.S. EPA. 1997. Office of Pesticide Programs list of chemicals evaluated for carcinogenic potential. Memo from W.L. Burnam, Health Effects Division, to Health Effects Division branch chiefs, et al. Washington, D.C., Feb. 19.

145 U.S. EPA. Office of Pesticide Programs. Health Effects Division. 1997. Tox oneliners: Permethrin. Washington D.C., June 24.

146 World Health Organization. 1990. Permethrin. Environmental Health Criteria 94. Geneva, Switzerland: World Health Organization, United Nations Environment Programme, and International Labor Organization. pp.76-78

147 USEPA. Tracking Report. Washington, DC 1997 cited in National Pesticide Telecommunications Network at http://npic.orst.edu/factsheets/pbotech.pdf.

148 United States Environmental Protection Agency, Ground Water and Drinking Water. "Consumer Factsheet on: Pentachlorophenol." Available at http://www.epa.gov/OGWDW/dwh/c-soc/pentachl.html

149 Agency for Toxic Substances and Disease Registry. "ToxFAQs(tm) for Pentachlorophenol CAS# 87-86-5." September 2001. Available at http://www.atsdr.cdc.gov/tfacts51.html accessed 1/16/03.

150 Pesticide Action Network Pesticide Data Base. Chemical Information About Pentachlorophenol." Available at http://www.pesticideinfo.org/PCW/Detail_Chemical.jsp?Rec_Id=PC34087

151 Extension Toxicology Network. "Pesticide Information Profiles: Pentachlorophenol (PCP)." Revised June 1996. Available at http://ace.orst.edu/cgi-bin/mfs/01/pips/pentachl.htm.

152 Agency for Toxic Substances and Disease Registry. "ToxFAQs(tm) for Pentachlorophenol CAS# 87-86-5." September 2001. Available at http://www.atsdr.cdc.gov/tfacts51.html

153 ibid

154 For more information visit: http://www.panna.org/resources/documents/factsPentachlorophenol.dv.html

155 Agency for Toxic Substances and Diseases Registry (ATSDR)/US Public Health Service, Toxicological Profile for 4,4'-DDT, 4,4'-DDE, 4, 4'-DDD (Update). 1994. ATSDR. Atlanta, GA. Cited in http://pmep.cce.cornell.edu/profiles/extoxnet/carbaryl-dicrotophos/ddt-ext.html

156 USEPA: DDT A regulatory History. at http://www.epa.gov/history/topics/ddt/02.htm

157 National Center for Environmental Health, U.S. Centers for Disease Control and Prevention, Second National Report on Human Exposure to Environmental Chemicals, January 2003; http://www.cdc.gov/exposurereport.

158 Cohn, BA, PM Cirillo, MS Wolff, PJ Schwing, RD Cohen, RI Sholtz, A Ferrara, RE Christianson, BJ van den Berg and PK Siiteri. 2003. DDT and DDE exposure in mothers and time to pregnancy in daughters. The Lancet 361: 2205–06.

159 Longnecker, MP, MA Klebanoff, H Zhou, JW Brock. 2001. Association between maternal serum concentration of the DDT metabolite DDE and preterm and small-for-gestational-age babies at birth. The Lancet 358: 110-114.

160 Stapleton, H., Dodder, N., Offenberg, J., Schantz, M., Wise, S., 2005. Polybrominated diphenyl ethers in house dust and clothes dryer lint. Environ. Sci. Technol. In Press.

161 Noren, K.; Meironyte, D., 2000. Certain organochlorine and organobromine contaminants in Swedish human milk in perspective of past 20-30 years. Chemosphere 40: 1111-1123.

162 Lassen, C., Lokke, S. & Hansen, L. I. ( 1999) Brominated Flame Retardants: substance flow analysis and substitution feasibility study. Danish Environmental Protection Agency Environmental Project No. 494, Copenhagen, ISBN 87- 7909- 415- 5: 240 pp.

163 BSEF (Bromine Science and Environmental Forum), 2000. BSEF factsheets: major brominated flame retardants volume estimates, July 27. Available from http://www.bsef.com.

164 Environmental Health Criteria 162: Brominated Diphenyl Ethers; World Health Organization: Geneva, Switzerland, 1994.

165 Lassen, C., Lokke, S. & Hansen, L. I. ( 1999) Brominated Flame Retardants: substance flow analysis and substitution feasibility study. Environmental Project No. 494. Copenhagen: Danish Environmental Protection Agency.

166 Darnerud PO, Eriksen GS, Johannesson T, Larsen PB, Viluksela M., 2001. Polybrominated diphenyl ethers: occurrence, dietary exposure and toxicology.Environ. Health Perspect. 109(Suppl 1):49–68.

167 Hites, R., 2004. Polybrominated diphenyl ethers in the environment and in people: A meta-analysis of concentrations. Environ. Sci. Technol. 38: 945-956.

168 Bocio, A.; Llobet, J. M.; Domingo, J. L.; Corbella, J.; Teixido, A.; Casas, C. Polybrominated diphenyl ethers (PBDEs) in foodstuffs: human exposure through the diet. J. Agric. Food Chem. 2003, 51, 3191- 3195..

169 Butt, C., Diamond, M., Truong, J., Ikonomou, M., Ter Schure, A., 2004. Spatial distribution of polybrominated diphenyl ethers in indoor and outdoor window organic films. Environ. Sci. Technol. 38: 724-731.

170 Stapleton, H., Dodder, N., Offenberg, J., Schantz, M., Wise, S., 2005. Polybrominated diphenyl ethers in house dust and clothes dryer lint. Environ. Sci. Technol. In Press.

171 Birnbaum, L. S., & Staskal, D. F. (2004). Brominated flame retardants: cause for concern? Environ Health Perspect, 112(1), 9-17.

172 Centers for Disease Control and Prevention, 2001. National Report on Human Exposure to Environmental Chemicals.

173 Centers for Disease Control and Prevention. 2003. Second National Report on Human Exposure to Environmental Chemicals. NCEH Pub. No. 02-0716.

174 Centers for Disease Control and Prevention, 2005. Third National Report on Human Exposure to Environmental Chemicals. In Press.

175 Meironyte D, Noren K, Bergman A (1999). Analysis of polybrominated diphenyl ethers in Swedish human milk. A time-related trend study, 1972–1997. J Toxicol Environ Health A;58(6):329–41.

176 Ohta S, Ishizuka D, Nishimura H, Nakao T, Aozasa O, Shimidzu Y, Ochiai F, Kida T, Nishi M, Miyata H (2002). Comparison of polybrominated diphenyl ethers in fish, vegetables, and meats and levels in human milk of nursing women in Japan.Chemosphere;46(5):689–96.

177 Guvenius DM, Aronsson A, Ekman-Ordeberg G, Bergman A, Noren K (2003). Human prenatal and postnatal exposure to polybrominated diphenyl ethers, polychlorinated biphenyls, polychlorobiphenylols, and pentachlorophenol. Environ Health Perspect;111(9):1235–41.

178 Northwest Environment Watch, 2004. Flame Retardants in the Bodies of Pacific Northwest Residents: A Study on Toxic Body Burdens. http://www.northwestwatch.org.

179 Schecter, A. J.; Pavuk, M.; Pa¨pke, O.; Ryan, J. J.; Birnbaum, L.; Rosen, R. Polybrominated Diphenyl Ethers (PBDEs) in U.S. mother's milk. Environ. Health Perspect. 2003, 111, 1723-1729.

180 Lunder, S., Sharp, R., 2003. Mothers' Milk: Record levels of toxic fire retardants found in American mothers' breast milk. http://www.ewg.org/

181 Schroter-Kermani, 2001. Endocrine disrupters in human and environmental samples from the German Environmental Specimen Bank. Proceedings of the Second Status Seminar: Endocrine Disrupters. 2–4 April, 2001, Berlin, Germany.

182 Mazdai, A., Dodder, N., Abernathy, M., Hites, R., Bigsby, R., 2003. Polybrominated diphenyl others in maternal and fetal blood samples. Environ. Health Perspect.111: 1249–52.

183 Guvenius, D., Aronsson, A., Ekman-Ordeberg, G., Bergman, A., Noren, K., 2003. Human prenatal and postnatal exposure to polybrominated diphenyl ethers, polychlorinated biphenyls, polychlorobiphenylols, and pentachlorophenol. Environ. Health Perspect. 111: 1235–41.

184 Mazdai, A., Dodder, N., Abernathy, M., Hites, R., Bigsby, R., 2003. Polybrominated diphenyl ethers in maternal and fetal blood samples. Environ. Health Perspect.111: 1249–52.

185 Hardell L, Lindstrom G, van Bavel B, Wingfors H, Sundelin E, Liljegren G 1998).Concentrations of the flame retardant 2,2',4,4'-tetrabrominated diphenyl ether in human adipose tissue in Swedish persons and the risk for non-Hodgkin's lymphoma. Oncol Res;10(8):429–32.

186 She, J., Petreas, M., Winkler, J., Visita, P., McKinney, M., Kopec, D., 2002. PBDEs in the San Francisco Bay area: Measurements in harbor seal blubber and human breast adipose tissue. Chemosphere 46:697–707.

187 Choi JW, Fujimaki TS, Kitamura K, Hashimoto S, Ito H, Suzuki N, Sakai S, Morita M (2003). Polybrominated dibenzo-pdioxins, dibenzofurans, and diphenyl ethers in Japanese human adipose tissue. Environ Sci Technol;37(5):817–21.

188 Smeds, A., Saukko, P., 2003. Brominated flame retardants and phenolic endocrine disrupters in Finnish human adipose tissue. Chemosphere 53: 1123-1130.

189 Schroter-Kermani, 2001. Endocrine disrupters in human and environmental samples from the German Environmental Specimen Bank. Proceedings of the Second Status Seminar: Endocrine Disrupters. 2–4 April, 2001, Berlin, Germany.

190 Noren, K.; Meironyte´, D., 2000. Certain organochlorine and organobromine contaminants in Swedish human milk in perspective of past 20-30 years. Chemosphere 40: 1111-1123.

191 Betts KS. 2002. Rapidly rising PBDE levels in North America. Environ Sci Technol 36: 50 – 52A.

192 Sjodin, A., Jones, R., Focant, J.-F., Lapeza, C., Wang, R., McGahee, E., Zhang, Y., Turner, W., Slazyk, B., Needham, L., Patterson, D., 2004. Retrospective time-trend of polybrominated diphenyl ether and polybrominated biphenyl levels in human serum from the United States. Environ Health Perspect 112:654–658

193 Ryan JJ, Patry B, Mills P, Beaudoin NG. Recent trends in levels of brominated diphenyl ethers ( BDEs) in human milks from Canada. Organohalog Compd 2002; 58: 173 – 6.

194 Meironyt_e e, D., Nor_e en, K., Bergman, _A A., 1999. Analysis of polybrominated diphenyl ethers in Swedish human milk. A time-related trend study, , 1972–1997. J. Toxicol. Environ. Health Part A 58, 329–341.

195 Hooper K, She J (2003). Lessons from the polybrominated diphenyl ethers (PBDEs): Precautionary principle, primary prevention, and the value of community-based body-burden monitoring using breast milk. Environ Health Perspect;111(1):109–14.

196 Hites, R., 2004. Polybrominated diphenyl ethers in the environment and in people: A meta-analysis of concentrations. Environ. Sci. Technol. 38: 945-956.

197 Northwest Environment Watch, 2004. Flame Retardants in the Bodies of Pacific Northwest Residents: A Study on Toxic Body Burdens. http://www.northwestwatch.org.

198 Schecter, A. J.; Pavuk, M.; Pa¨pke, O.; Ryan, J. J.; Birnbaum, L.; Rosen, R. Polybrominated Diphenyl Ethers (PBDEs) in U.S. mother's milk. Environ. Health Perspect. 2003, 111, 1723–1729.

199 Lunder, S., Sharp, R., 2003. Mothers' Milk: Record levels of toxic fire retardants found in American mothers' breast milk. http://www.ewg.org/

200 She, J., Petreas, M., Winkler, J., Visita, P., McKinney, M., Kopec, D., 2002. PBDEs in the San Francisco Bay area: Measurements in harbor seal blubber and human breast adipose tissue. Chemosphere 46:697–707.

201 Schecter, A. J.; Pavuk, M.; Pa¨pke, O.; Ryan, J. J.; Birnbaum, L.; Rosen, R. Polybrominated Diphenyl Ethers (PBDEs) in U.S. mother's milk. Environ. Health Perspect. 2003, 111, 1723-1729.

202 Schecter, A., Papke, O., Tung, K.-C., Staskal, D., Birnbaum, L., 2004. Polybrominated diphenyl ethers contamination in United States food. Environ. Sci. Technol. 38: 5306-5311.

203 Mazdai, A., Dodder, N., Abernathy, M., Hites, R., Bigsby, R., 2003. Polybrominated diphenyl ethers in maternal and fetal blood samples. Environ. Health Perspect.111: 1249–52.

204 Guvenius, D., Aronsson, A., Ekman-Ordeberg, G., Bergman, A., Noren, K., 2003. Human prenatal and postnatal exposure to polybrominated diphenyl ethers, polychlorinated biphenyls, polychlorobiphenylols, and pentachlorophenol. Environ. Health Perspect. 111: 1235–41.

205 Thomsen, C., Lundanes, E., Becher, G., 2002. Brominated flame retardants in archived serum samples from Norway: A study on temporal trends and the role of age. Environ. Sci. Technol. 36: 1414- 1418

206 She, J., Petreas, M., Winkler, J., Visita, P., McKinney, M., Kopec, D., 2002. PBDEs inthe San Francisco Bay area: Measurements in harbor seal blubber and human breast-adipose tissue. Chemosphere 46:697–707.

207 Schroter-Kermani, 2001. Endocrine disrupters in human and environmental samples from the German Environmental Specimen Bank. Proceedings of the Second Status Seminar: Endocrine Disrupters. 2–4 April, 2001, Berlin, Germany.

208 She, J., Petreas, M., Winkler, J., Visita, P., McKinney, M., Kopec, D., 2002. PBDEs in the San Francisco Bay area: Measurements in harbor seal blubber and human breast adipose tissue. Chemosphere 46:697–707.

209 Mazdai, A., Dodder, N., Abernathy, M., Hites, R., Bigsby, R., 2003. Polybrominated diphenyl ethers in maternal and fetal blood samples. Environ. Health Perspect.111: 1249–52.

210 Darnerud, P., 2003. Toxic effects of brominated flame retardants in man and in wildlife. Environ. Internat. 29: 841-853.

211 Bahn A, Bialik O, Oler J, Houten L, Landau E. Health assessment of occupational exposure to polybrominated biphenyl (PBB) and polybrominated biphenyl oxide (PBBO). Office of pesticides and toxic substances (ISS EPA 560/6-80-001; NTIS No. PB81-159675), US Environmental Protection Agency, Washington, DC; 1980.

212 Hagmar L., Bjork, J., Sjodin, A., Bergman, A., Erfurth E, 2001. Plasma levels of persistent organohalogens and hormone levels in adult male humans. Arch. Environ. Health 56:138– 43.

213 Chen, G., Bunce, N., 2003. Polybrominated diphenyl ethers as Ah receptor agonists and antagonists. Toxicol. Sci. 76: 310-320.

214 Gillner, M.; Jakobsson, E. Structure-affinity relationships for thyroid and dioxin receptor binding of halogenated naphthalenes and diphenyl ethers. Organohalogen Compd. 1996, 29, 220-221.

215 Hallgren, S.; Darnerud, P. O. Effects of polybrominated diphenyl ethers (PBDEs), polychlorinated biphenyls (PCBs) and chlorinated paraffins (CPs) on thyroid hormone levels and enzyme activities in rats. Organohalogen Compd. 1998, 35, 391-394.

216 Meerts, I. A.; Assink, Y.; Cenijn, P. H.; VanDenBerg, J. H.; Weijers, B. M.; Bergman, A°.; Koerman, J. H.; Brouwer, A. Placental transfer of a hydroxylated polychlorinated biphenyl and effects on fetal and maternal thyroid hormone homeostasis in the rat. Toxicol. Sci. 2002, 68, 361-371.

217 Morse, D. C.; Groen, D.; Veerman, M.; van Amerongen, C. J.; Koeter, H. B. W. M.; Smits van Prooije, A. E.; Visser T. J.; Koeman, J. H.; Brouwer, A. Interference of polybrominated biphenyls in hepatic and brain thyroid hormone metabolism in fetal and neonatal rats. Toxicol. Appl. Pharmacol. 1993, 122, 27-33.

218 Meerts, I. A.; Letcher, R. J.; Hoving, S.; Marsh, G.; Bergman, A° .; Lemmen,J. G.; van der Burd, B.; Brouwer, A. In vitro estrogenicity of polybrominated diphenyl ethers, hydroxylated PDBEs, and polybrominated bisphenol A compounds. Environ. Health Perspect. 2001, 109, 399-407.

219 Branchi, I.; Alleva, E.; Costa, L. G. Effects of perinatal exposure to a polybrominated diphenyl ether (PBDE 99) on mouse neurobehavioural development. Neurotoxicology 2002, 23, 375–384.

220 Eriksson, P., Viberg, H., Jakobsson, E., Orn, U., Fredriksson, A., 2002. A brominated flame retardant, 2,2',4,4', 5-pentabromodiphenyl ether: uptake, retention, and induction of neurobehavioral alterations in mice during a critical phase of neonatal brain development. Toxicol. Sci. 2002, 67, 98-103.

221 Birnbaum, L. B.; Staskal, D. F. Brominated flame retardants: Cause for concern? Environ. Health Perspect. 2003, 112, 9-17.

222 Branchi, I.; Capone, F.; Alleva, E.; Costa, L. G. Polybrominated diphenyl ethers: neurobehavioral effects following developmental exposure. Neurotoxicology 2003, 24 (3), 449-462.

223 NTP. Toxicology and Carcinogenesis Studies of Decabromodiphenyl Oxide (CAS No. 1163-19-5) in F344/N Rats and B6C3F1 Mice (Feed Studies). TR-309. Research Triangle Park, NC: National Toxicology Program. 1986. http://ntp-server.niehs.nih.gov/htdocs/LT-studies/tr309.html Last accessed May 15, 2004.

224 Eriksson, P., Johansson, N., Viberg, H., Fischer, C., Fredriksson, A., 2004. Comparative developmental neurotoxicity of flame-retardants, polybrominated flame-retardants and organophosphorous compounds, in mice. Organohalogen Cpds. 66: 3163-3165.

225 Eriksson, P., Jakobsson, E., Fredriksson, A., 2001. Brominated fkame retardants: a novel class of developmental neurotoxicants in our environment? Environ. Health Perspect. 109: 903–908.

226 Eriksson, P., Viberg, H., Jakobsson, E., Orn, U., Fredriksson, A., 2002. A brominated flame retardant, 2,2',4,4',5-pentabromodiphenyl ether: uptake, retention, and induction of neurobehavioral alterations in mice during a critical phase of neonatal brain development. Toxicol. Sci. 2002, 67, 98-103.

227 Eriksson, P., 1997. Developmental neurotoxicity of environmental agents in the neonate. Neurotoxicology 18, 719–726.

228 Darnerud, P., 2003. Toxic effects of brominated flame retardants in man and in wildlife. Environ. Internat. 29: 841–853.

229 Helleday, T., Tuominen, K., Bergman, A., Jenssen, D., 1999. Brominated flame retardants induce intragenic recombination in mammalian cells. Mutat. Res. 439: 137–47.

230 Viberg, H., Fredriksson, A., Jakobsson, E., Orn, U., Eriksson, P., 2003. Neurobehavioral Derangements in Adult Mice Receiving Decabrominated Diphenyl Ether (PBDE 209) during a Defined Period of Neonatal Brain Development. Toxicological Sciences 76: 112-120

231 Eriksson, P., Viberg, H., Jakobsson, E., Orn, U., Fredriksson, A., 2002. A brominated flame retardant, 2,2',4,4',5-pentabromodiphenyl ether: uptake, retention, and induction of neurobehavioral alterations in mice during a critical phase of neonatal brain development. Toxicol. Sci. 2002, 67, 98-103.

232 Eriksson, P., Jakobsson, E., Fredriksson, A., 2001. Brominated fkame retardants: a novel class of developmental neurotoxicants in our environment? Environ. Health Perspect. 109: 903–908.

233 Viberg, H.; Fredriksson, A.; Eriksson, P. Neonatal exposure to polybrominated diphenyl ether ( PBDE 153) disrupts spontaneous behaviour, impairs learning and memory, and decreases hippocampal cholinergic receptors in adult mice. Toxicol. Appl. Pharmacol. 2003, 192, 95- 106.

234 Kuehl, D.W., Haebler, R., 1995. Organochlorine, organobromine, metal, and selenium residues in bottlenose dolphins (Tursiops truncatus) collected during an unusual mortality event in the Gulf of Mexico, 1990. Arch. Environ. Contam. Toxicol. 28, 494–499.

235 Strandman, T., Koistinen, J., Vartiainen, T., 2000. Polybrominated diphenyl ethers (PBDEs) in placenta and human milk. Organohalogen Compd. 47, 61–64.

236 Law, R.J., Allchin, C.R., Bennett, M.E., Morris, S., Rogan, E., 2002. Polybrominated diphenyl ethers in two species of marine top predators from England and Wales. Chemosphere 46, 673–681.

237 She, J., Petreas, M., Winkler, J., Visita, P., McKinney, M., Kopec, D., 2002. PBDEs in the San Francisco Bay area: Measurements in harbor seal blubber and human breast adipose tissue. Chemosphere 46:697–707.

238 Eriksson, P., Jakobsson, E., Fredriksson, A., 2001. Brominated fkame retardants: a novel class of developmental neurotoxicants in our environment? Environ. Health Perspect. 109: 903–908.

239 Viberg, H.; Fredriksson, A.; Eriksson, P. Neonatal exposure to polybrominated diphenyl ether ( PBDE 153) disrupts spontaneous behaviour, impairs learning and memory, and decreases hippocampal cholinergic receptors in adult mice. Toxicol. Appl. Pharmacol. 2003, 192, 95–106.

240 Viberg, H., Fredriksson, A., Jakobsson, E., Orn, U., Eriksson, P., 2003. Neurobehavioral Derangements in Adult Mice Receiving Decabrominated Diphenyl Ether (PBDE 209) during a Defined Period of Neonatal Brain Development. Toxicological Sciences 76: 112-120

241 Fowles, J.R., Fairbrother, A., B.-Steppan, L., Kerkvliet, N.I., 1994. Immunologic and endocrine effects of the flame retardant pentabromodiphenyl ether (DE-71) in C57BL/6J mice. Toxicology 86, 49–61.

242 Eriksson, P., Viberg, H., Jakobsson, E., Örn, U., Fredriksson, A., 1999. PBDE, 2,2',4,4',5-pentabromodiphenyl ether, causes permanent neurotoxic effects during a defined period of neonatal brain development. Organohalogen Compounds 40: 333-336.

243 Eriksson, P., Jakobsson, E., Fredriksson, A., 2001. Brominated fkame retardants: a novel class of developmental neurotoxicants in our environment? Environ. Health Perspect. 109: 903–908.

244 Meerts, I.A.T.M., van Zanden, J.J., Luijks, E.A.C., van Leeuwen-Bol, I., Marsh, G., Jakobsson, E., Bergman, A. A., Brouwer, A., 2000. Potent competitive interaction of some brominated .ame retardants and related compounds with human transthyretin in vitro. Toxicol. Sci. 56, 95–104.

245 Hallgren, S., Sinjari, T., H_a akansson, H., Darnerud, P.O., 2001. Effects of polybrominated diphenyl ethers (PBDEs) and polychlorinated biphenyls (PCBs) on thyroid hormone and vitamin A levels in rats and mice. Arch. Toxicol. 75, 200–208.

246 Zhou, T., Ross, D.G., DeVito, M.J., Crofton, K.M., 2001. Effects of short-term in vivo exposure to polybrominated diphenyl ethers on thyroid hormones and hepatic enzyme activities in weanling rats. Toxicol. Sci. 61, 76–82.

247 Carlson, G.P., 1980a. Induction of xenobiotic metabolism in rats by short-term administration of brominated diphenyl ethers. Toxicol. Lett. 5, 19–25.

248 Carlson, G.P., 1980b. Induction of xenobiotic metabolism in rats by brominated diphenyl ethers administered for 90 days. Toxicol. Lett. 6, 207–212.

249 von Meyerinck, L., Hufnagel, B., Schmoldt, A., Benthe, H.F., 1990. Induction of rat liver microsomal cytochrome P-450 by the pentabromo diphenyl ether Bromkal 70 and half-lives of its components in the adipose tissue. Toxicology 61, 259–274.

250 Fowles, J.R., Fairbrother, A., B.-Steppan, L., Kerkvliet, N.I., 1994. Immunologic and endocrine effects of the flame retardant pentabromodiphenyl ether (DE-71) in C57BL/6J mice. Toxicology 86, 49–61.

251 Helleday, T., Tuominen, K.L., Bergman, A., Jenssen, D., 1999. Brominated .ame retardants induce intragenic recombination in mammalian cells. Mutat. Res. 439, 137–147.

252 Chen, G., Konstantinov, A.D., Chittim, B.G., Joyce, E.M., Bols, N.C., Bunce, N.J., 2001. Synthesis of polybrominated diphenyl ethers and their capacity to induce CYP1A by the Ah receptor mediated pathway. Environ. Sci. Technol. 35, 3749–3756.

253 Meerts, I.A.T.M., Letchere, R.J., Hoving, S., Marsh, G., Bergman, A., Lemmen, J.G., van der Burg, B., Brouwer, A., 2001. In Vitro estragenicity of polybrominated diphenyl ethers, hydroxylated PBDEs, and polybrominated bisphenol A compounds. Environ. Health Perspect. 109, 399–407.

254 Eriksson, P., Viberg, H., Jakobsson, E., Örn, U., Fredriksson, A., 1999. PBDE, 2,2',4,4',5-pentabromodiphenyl ether, causes permanent neurotoxic effects during a defined period of neonatal brain development. Organohalogen Compounds 40: 333-336.

255 Eriksson, P., Jakobsson, E., Fredriksson, A., 2001. Brominated fkame retardants: a novel class of developmental neurotoxicants in our environment? Environ. Health Perspect. 109: 903–908.

256 Kodavanti, P., Ward, T., Burka, T., Ludewig, G., Robertson, L., Birnbaum, L., 2004. Polybrominated diphenyl ether (PBDE) effects in rat neuronal cultures: 14C-PBDE accumulation, biological effect, and structure-activity relationships. Organohalogen Cpds. 66: 3133-3138

257 Viberg, H., Fredriksson, A., Jakobsson, E., Orn, U., Eriksson, P., 2003. Neurobehavioral Derangements in Adult Mice Receiving Decabrominated Diphenyl Ether (PBDE 209) during a Defined Period of Neonatal Brain Development. Toxicological Sciences 76: 112–120

258 Branchi, I.; Alleva, E.; Costa, L. G. Effects of perinatal exposure to a polybrominated diphenyl ether (PBDE 99) on mouse neurobehavioural development. Neurotoxicology 2002, 23, 375–384.

259 Branchi I, Capone F, Alleva E, Costa LG ( 2003). Polybrominated diphenyl ethers: Neurobehavioral effects following developmental exposure. Neurotoxicology; 24 ( 3): 449 – 62.

260 Eriksson, P., Jakobsson, E., Fredriksson, A., 2001. Brominated fkame retardants: a novel class of developmental neurotoxicants in our environment? Environ. Health Perspect. 109: 903–908.

261 Meerts, I. A. T. M., Letcher, R. J., Hoving, S., Marsh, G., Bergman, Å., Lemmen, J. G., van der Burg, B. & Brouwer, A. ( 2001) In vitro estrogenicity of polybrominated diphenyl ethers, hydroxylated PBDEs and polybrominated bisphenol A compounds. Environmental Health Perspectives 109( 4): 399–407

262 Olsen CM, Meussen- Elholm ET, Samuelsen M, Holme JA, Hongslo JK ( 2003). Effects of the environmental oestrogens bisphenol A, tetrachlorobisphenol A, tetrabromobisphenol A, 4-hydroxybiphenyl and 4,4'- dihydroxybiphenyl on oestrogen receptor binding, cell proliferation and regulation of oestrogen sensitive proteins in the human breast cancer cell line MCF- 7. Pharmacol Toxicol; 92( 4): 180–8.

263 Fromme, H., Mattulat, A., Lahrz, T., Ruden, H., 2005. Occurrence of organotin compounds in house dust in Berlin (Germany). Chemosphere 58: 1377-1383.

264 Doering, D., Steckelbroeck, S., Doering, T., Klingmuller, D., 2002. Effects of butyltins on human 5 alpha-reductase type 1 and type 2 activity. Steroids 67: 859-867.

265 Hock, M., 2001. Organotin compounds in the environment—an overview. Appl. Geochem. 16: 719-753.

266 Mercier, A., E. Pelletier and J. Hamel. 1994. Metabolism and subtle toxic effects of butyltin compounds in starfish. Aquat. Toxicol. 28: 259- 273.

267 Graf, G. G., 1996. Tin, tin alloys, and tin compounds. In: H.- J. Arpe ( Ed.), Ullmann's Encyclopedia of Industrial Chemistry, Vol. A27. Wiley- VCH, Weinheim, pp. 49–81.

268 Hock, M., 2001. Organotin compounds in the environment— an overview. Appl. Geochem. 16: 719–753.

269 Rudel, H., 2003. Case study: Bioavailability of tin and tin compounds. Ecotox. Environ. Safety 56: 180-189.

270 Fromme, H., Mattulat, A., Lahrz, T., Ruden, H., 2004. Occurrence of organotin compounds in house dust in Berlin (Germany). Chemosphere. In Press

271 http://www.ortepa.org/stabilizers/pages/applications.htm

272 Gaikema, F., Alberts, P., 1999. Gaschromatografische bepaling van residuen van organotinverbindingen in textileproducten. De Ware(n)-Chemicus 1999: 23-33. As cited in Peters, R., 2003. The determination of selected additives in consumer products. TNO report R 2004/002. Apeldoorn, NHL: TNO.

273 Takehashi, S., Mukai, H., Tanabe, S., Sakayama, K., Miyazaki, T., Masuno, H., 1999. Butyltin residues in livers of humans and wild terrestrial mammals and in plastic products. Environ. Pollut. 106: 213-218.

274 Ohno, H., Suzuki, M., Aoyama, T., Mitani, K., 2003. Determination of organotins in polyvinyl chloride toys. Shokuhin Eiseigaku Zasshi 44: 208–212. [Abstract]

275 Sadiki A, Williams DT. 1996. Speciation of organotin and organolead compounds in drinking water by gas chromatography-atomic emission spectrometry. Chemosphere 32:1983–1992.

276 Sadiki AI, Williams DT. 1999. A study on organotin levels in Canadian drinking water distributed through PVC pipes. Chemosphere 38:1541-1548.

277 Sadiki AI, Williams DT, Carrier R, et al. 1996. Pilot study on the contamination of drinking water by organotin compounds from PVC materials. Chemosphere 32: 2389–2398.

278 Bancon-Montigny, C., Lespes, G., Potin-Gautier, M., 2004. Organotin survey in the Adour-Garonne basin. Water research 38: 933-946.

279 Allsopp, A., Santillo, D. & Johnston, P., 2001. Poison Underfoot: Hazardous Chemicals in PVC Flooring and Hazardous Chemicals in Carpets. ISBN 90- 73361- 68-0. Amsterdam: Greenpeace International.

280 Huang, J.-H., Klemm, O., 2004. Atmospheric speciation of ionic organotin, organolead and organomercury compounds in NE Bavaria (Germany). Atmospheric Environment 38: 5013-5023.

281 Huang, J., Schwesig, D., Matzner, E., 2004. Organotin compounds in precipitation, fog and soils of a forested ecosystem in Germany. Environ. Pollut. 130: 177–186.

282 Bancon-Montigny, C., Lespes, G., Potin-Gautier, M., 2004. Organotin survey in the Adour-Garonne Basin. Water Research 38: 933–946.

283 Maguire, R.J., Tkacz, R.J., Chau, Y.K., Bengert, G.A., Wong, P.T.S., 1986. Occurrence of organotin compounds in water and sediment in Canada. Chemosphere 15, 253–274.

284 Landmeyer, J., Tanner, T., Watt, B., 2004. Biotransformation of tributyltin to tin in freshwater river-bed sediments contaminated by an organotin release. Environ. Sci. Technol. 38: 4106-4112

285 IPCS (1999). Tributyltin oxide. International Programme on Chemical Safety Concise International Chemical Assessment Document: 14. Accessed Sept 2003 at http://www.inchem.org/documents/cicads/cicads/cicad14.htm#PartNumber:

286 Takehashi, S., Mukai, H., Tanabe, S., Sakayama, K., Miyazaki, T., Masuno, H., 1999. Butyltin residues in livers of humans and wild terrestrial mammals and in plastic products. Environ. Pollut. 106: 213–218.

287 Adeeko, Toxicological Sciences 74, 407-415 (2003)

288 Centers for Disease Control and Prevention, 2001. National Report on Human Exposure to Environmental Chemicals.

289 Centers for Disease Control and Prevention. 2003. Second National Report on Human Exposure to Environmental Chemicals. NCEH Pub. No. 02-0716.

290 Centers for Disease Control and Prevention, 2005. Third National Report on Human Exposure toEnvironmental Chemicals. In Press.

291 Kannan K, Senthilkumar K, Giesy JP. 1999. Occurrence of butyltin compounds in human blood. Environ Sci Technol 33: 1776- 1779

292 Rudel, H., Steinhanses, J., 2001. Organische Zinnverbindungen in Biota der Umweltprobenbank—Erganzungsmessungen. Report of Fraunhofer IME for Umweltbundesamt, Berlin. As cited in Rudel, H., 2003. Case study: Bioavailability of tin and tin compounds. Ecotox. Environ. Safety 56: 180–189.

293 Takehashi, S., Mukai, H., Tanabe, S., Sakayama, K., Miyazaki, T., Masuno, H., 1999. Butyltin residues in livers of humans and wild terrestrial mammals and in plastic products. Environ. Pollut. 106: 213-218.

294 Kannan K, Falandysz J. 1997. Butyltin residues in sediment, fish, fish-eating birds, harbour porpoise and human tissues from the Polish coast of the Baltic Sea. Mar Pollut Bull 34: 203–207.

295 J.B. Nielsen, J. Strand, Butyltin compounds in human liver, Environ. Res. 88 (2002) 129–133.

296 R.S. Braman, M.A. Tompkins, Separation and determination of nanogram amounts of inorganic tin and methyltin compounds in the environment, Anal. Chem. 51 (1979) 12– 19.

297 Zuckerman, J. J., Reisdorf, R. J., Ellis, H. V. III, and Wilkinson, R. R. (1978). Organotins in biology and the environment. In Organometals and Organometalloids, Occurrence and Fate in the Environment, American Chemical Society Symposium Series 82. (F. E. Brinckman and J. M. Bellama, Eds.), pp. 388 – 424. American Chemical Society, Washington, DC. As cited in Adeeko, A., Li, D., Forsyth, D., Casey, V., Cooke, G., Barthelemy, J., Cyr, D., Trasler, J., Robaire, B., Hales, B., 2003. Effects of in utero tributyltin chloride exposure in the rat on pregnancy outcome. Toxicol. Sci. 74: 407–415.

298 Jenkins, S., Ehman, K., Barone, S., 2004. Structure-activity comparison of organotin species: Dibutyltin is a developmental neurotoxicant in vitro and in vivo. Develop. Brain Res. 151: 1–12.

299 Norris WR (1994). Triphenyltin Acetate. International Programme on Chemical Safety Poisons Information Monograph: 589. Accessed Sept 2003 at: http://www.inchem.org/documents/pims/chemical/pim589.htm

300 van Heijst APN (1994). Tributyltin compounds. International Programme on Chemical Safety Group Poisons Information Monograph: G018. Accessed Sept 2003 at: http://www.inchem.org/documents/pims/chemical/pimg018.htm

301 Jensen KG, Wiberg K, Klasson-Wehler E, Onfelt A (2000). Induction of aberrant mitosis with PCBs: Particular efficiency of 2, 3,3',4,4'-pentachlorobiphenyl and synergism with triphenyltin. Mutagenesis;15(1):9–15.

302 Cima, F., Ballarin, L., Bressa, G., Martinucci, G., and Burighel, P. 1996. Toxicity of Organotin Compounds on Embryos of a Marine Invertebrate (Styela plicata; Tunicata). Ecotoxicology and Environmental Safety, 35: 174–182.

303 Bouchard, N., Pelletier, E., and Fournier, M. 1999. Effects of Butyltin Compounds on Phagocytic Activity of Hemocytes form Three Marine Bivalves. Environmental Toxicology and Chemistry, 18( 3): 519–522.

304 Al- Ghais, S. M. & Ahmad, A. B. ( 2000) Differential inhibition of xenobioticmetabolizing carboxylesterases by organotins in marine fish. Ecotoxicology and Environmental Safety 46( 3): 258- 264

305 O'Halloran, K., Ahokas, J. T. & Wright, P. F. A. (1998) Response of fish immune cells to in vitro organotin exposures. Aquatic Toxicology 40( 2- 3): 141–156

306 Kannan, K., Senthilkumar, K., Giesy, J.P., 1999. Occurrence of butyltin compounds in human blood. Environ. Sci. Technol. 33, 1776–1779.

307 Kergosien DH, Rice CD. 1998. Macrophage secretory function is enhanced by low doses of tributyltinoxide (TBTO), but not tributyltin- chloride (TBTC1). Arch Environ Contam Toxicol 34( 3): 223–228.

308 Nakata H, Sakakibara A, Kanoh M, Kudo S, Watanabe H, Nagai N, Miyazaki N, Asano Y, Tanabe S (2002). Evaluation of mitogen-induced responses in marine mammal and human lymphocytes by in vitro exposure of butyltins and non-ortho coplanar PCBs. Environ Pollut;120(2):245–53.

309 Stridh H, Cotgreave I, Muller M, Orrenius S, Gigliotti D (2001). Organotin-induced caspase activation and apoptosis in human peripheral blood lymphocytes. Chem ResToxicol;14(7):791–8.

310 Whalen MM, Loganathan BG (2001). Butyltin exposure causes a rapid decrease in cyclic AMP levels in human lymphocytes. Toxicol Appl Pharmacol;171(3):141–8.

311 Whalen MM, Green SA, Loganathan BG (2002). Brief butyltin exposure induces irreversible inhibition of the cytotoxic function on human natural killer cells, in vitro. Environ Res;8 8(1): 19–29.

312 Whalen MM, Walker L, Loganathan BG (2002a). Interleukins 2 and 12 produce signifiant recovery of cytotoxic function in dibutyltin-exposed human natural killer cells. Environ Res; 8 8(2):103–15.

313 Whalen MM, Williams TB, Green SA, Loganathan BG (2002b). Interleukins 2 and 12 produce recovery of cytotoxic function in tributyltinexposed human natural killer cells. Environ Res;8 8(3):199–209.

314 Whalen MM, Wilson S, Gleghorn C, Loganathan BG (2003). Brief exposure to triphenyltin produces irreversible inhibition of the cytotoxic function of human natural killer cells. Environ Res;9 2(3):213–20.

315 Watanabe H, Adachi R, Hirayama A, Kasahara T, Suzuki K (2003). Triphenyltin enhances the neutrophilic differentiation of promyelocytic HL-60 cells. Biochem Biophys Res Commun;306(1):26–31.

316 Miura Y, Matsui H (1991). Inhibitory effects of phenyltin compounds on stimulus-induced changes in cytosolic free calcium and plasma membrane potential of human neutrophils. Arch Toxicol;65(7):562–9.

317 Ema, M., R. Kurosaka, H. Amano and Y. Ogawa. 1995. Comparative developmental toxicity of butyltin trichloride, dibutyltin dichloride and tributyltin chloride in rats. J. Appl. Toxicol. 15( 4): 297–302.

318 de Santiago, A. & Aguilar- Santelises, M. (1999) Organotin compounds decrease in vitro survival, proliferation and differentiation of normal human B lymphocytes. Human and Experimental Toxicology 18(10): 619–624

319 Eskes, C., Honegger, P., Jones-Lepp, T., Varner, K., Matthieu, J.M. & Monnet-Tschudi, F. (1999) Neurotoxicity of dibutyltin in aggregating brain cell cultures. Toxicology In Vitro 13(4-5): 555-560

320 M.M. Whalen, B.G. Loganathan, K. Kannan, Immunotoxicity of environmentally relevant concentrations of butyltins on human natural killer cells in vitro, Environ. Res. 81 (1999) 108–116.

321 Jenkins, S., Ehman, K., Barone, S., 2004. Structure-activity comparison of organotin species: dibutyltin is a developmental neurotoxicant in vitro and in vivo. Developmental Brain Research 151: 1–12.

322 Kergosien DH, Rice CD. 1998. Macrophage secretory function is enhanced by low doses of tributyltinoxide (TBTO), but not tributyltin- chloride (TBTC1). Arch Environ Contam Toxicol 34( 3): 223–228.

323 Adeeko A, Li D, Forsyth DS, Casey V, Cooke GM, Barthelemy J, Cyr DG, Trasler JM, Robaire B, Hales BF (2003). Effects of in utero tributyltin chloride exposure in the rat on pregnancy outcome. Toxicol Sci;74(2): 407–15.

324 Hirose, A., Takagi, A., Nishimura, T., Kanno, J., Ema, M., 2004. Review of reproductive and developmental toxicity induced by organotins in aquatic organisms and experimental animals. Organohalogen Cpds. 66: 3042–3047.

325 Ellis, D., Martin, J., De Silva, A., Mabury, S., Hurley, M., Andersen, M., Wallington, T., 2004. Degradation of fluorotelomer alcohols: A likely atmospheric source of perfluorinated carboxylic acids. Environ. Sci. Technol. 38: 3316-3321.

326 Renner, 2003. Concerns over common perfluorinated surfactant. Environmental Science & Technology, Science News. http://pubs.acs.org/subscribe/journals/esthag-w/2003/may/science/rr_surfactant.html.

327 Ring, K.-L., Kaelin, T., Kishi, A., 2002. Fluoropolymers: CEH Report. [Abstract] http://www.sriconsulting.com/CEH/Public/Reports

328 Ring, K.-L., Loechner, U., 2003. Fluoroelastomers: CEH Report. [Abstract] http://www.sriconsulting.com/CEH/Public/Reports

329 Ring, K.-L., Kaelin, T., Kishi, A., 2002. Fluoropolymers: CEH Report. [Abstract] http://www.sriconsulting.com/CEH/Public/Reports

330 Ring, K.-L., Kaelin, T., Kishi, A., 2002. Fluoropolymers: CEH Report. [Abstract] http://www.sriconsulting.com/CEH/Public/Reports

331 Ring, K.-L., Loechner, U., 2003. Fluoroelastomers: CEH Report. [Abstract] http://www.sriconsulting.com/CEH/Public/Reports

332 Richardson, S., 2004. Environmental mass spectrometry: emerging contaminants and current issues. Anal. Chem. 76: 3337-3364.

333 Shoeib, M., Harner, T., Ikonomou, M., Kannan, K., 2004. Indoor and outdoor air concentrations and phase partitioning of perfluoroalkyl sulfonamides and polybrominated diphenyl ethers. Environ. Sci. Technol. 38: 1313-1320.

334 Shoeib, M., Harner, T., Wilford, B., Jones, K., Zhu, J., 2004. A survey of perfluoroalkyl sulfonamides in indoor and outdoor air using passive air samplers. Organohalogen Cpds. 66" 3999–4003.

335 Tittlemier, S., Edwards, L., Pepper, K., 2003. Concentrations and temporal trends of two perfluorooctyl sulfonamides in fast food composites collected during the Canadian total diet study. Organohalogen Cpds. 62: 315-318.

336 Giesy, J. P., and Kannan, K. (2001). Global distribution of perfluorooctane sulfonate in wildlife. Environ. Sci. Technol. 35, 1339–1342.

337 Kannan, K., Choi, J.W., Iseki, N., Senthilkumar, K., Kim, D. H., and Giesy, J.P. (2002a). Concentrations of perfluorinated acids in livers of birds from Japan and Korea. Chemosphere 49, 225–231.

338 Kannan, K., Corsolini, S., Falandysz, J., Oehme, G., Focardi, S., and Giesy, J.P. (2002b). Perfluorooctanesulfonate and related fluorinated hydrocarbonsin marine mammals, fishes, and birds from coasts of the Baltic and theMediterranean Seas. Environ. Sci. Technol. 36, 3120–3126.

339 Kannan, K., Franson, J. C., Bowerman, W.W., Hansen, K.J., Jones, P. D., and Giesy, J.P. (2001a). Perfluorooctane sulfonate in fish-eating water birds including bald eagles and albatrosses. Environ. Sci. Technol. 35, 3065–3070.

340 Kannan, K., Hansen, K. J., Wade, T. L., and Giesy, J.P. (2002c). Perfluorooctane sulfonate in oysters, Crassostrea virginica, from the Gulf of Mexico and the Chesapeake Bay, USA. Arch. Environ. Contam. Toxicol. 42, 313–318.

341 Kannan, K., Koistinen, J., Beckmen, K., Evans, T., Gorzelany, J. F., Hansen, K.J., Jones, P. D., Helle, E., Nyman, M., and Giesy, J. P. (2001b). Accumulation of perfluorooctane sulfonate in marine mammals. Environ. Sci. Technol. 35, 1593–1598.

342 Corsolini, S., Kanna, K., 2004. Perfluorooctanesulfonate and related fluorochemicals in several organisms including humans from Italy. Organohalogen Cpds. 66: 4079-4085.

343 Guruge, K., Taniyasu, S., Miyazaki, S., Yamanaka, N., Yamashita, N., 2004. Age dependent accumulation of perfluorinated chemicals in beef cattles. Organohalogen Cpds. 66: 4029-4034.

344 Centers for Disease Control and Prevention, 2001. National Report on Human Exposure to Environmental Chemicals.

345 Centers for Disease Control and Prevention. 2003. Second National Report on Human Exposure to Environmental Chemicals. NCEH Pub. No. 02-0716.

346 Centers for Disease Control and Prevention, 2005. Third National Report on Human Exposure to Environmental Chemicals. In Press.

347 Dominiak, M., 2004. PFOS/PFOA: EPA update on perfluorinated compounds. U.S. Environmental Protection Agency. GCRC 2004. March 9, 2004

348 Olsen, G. W., Church, T. R., Miller, J. P., Burris, J. M., Hansen, K. J., Lundberg, J. K., Armitage, J. M., Herron, R. M., Medhdizadehkashi, Z., Nobiletti, J. B., O'Neill, E. M., Mandel, J. H., Zobel, L. R., 2003a. Perfluorooctanesulfonate and other fluorochemicals in the serum of American Red Cross adult blood donors. Environ. Health Perspect. 111, 1892–1901.

349 Olsen, G. W., Burris, J. M., Lundberg, J. K., Hansen, K. J., Mandel, J. H.,, Zobel, L. R., 2002. Identification of Fluorochemicals in Human Sera: III. Pediatric Participants in a Group A Streptococci Clinical Trial Investigation. US EPA Docket AR- 226–1085.

350 Olsen, G. W., Church, T. R., Larson, E. B., van Belle, G., Lundberg, J. K., Hansen, K. J., Burris, J. M., Mandel, J. H., Zobel, L. R., 2004. Serum concentrations of perfluorooctanesulfonate and other fluorochemicals in an elderly population from Seattle, Washington. Chemosphere. 54, 1599–1611.

351 Olsen, G. W., Burris, J. M., Mandel, J. H., and Zobel, L. R. (1999). Serum perfluorooctane sulfonate and hepatic and lipid clinical chemistry tests in fluorochemical production employees. J. Occup. Environ. Med. 41, 799–806.

352 Olsen, G. W., Schmickler, M. N., Tierens, J. M., Logan, P. W., Burris, J. M., Burlew, M. M., Lundberg, J. K., Mandel, J. H., 2001. Descriptive summary of serum fluorochemical levels among employee participants of the year 2000 Antwerp fluorochemical medical surveillance program. St. Paul ( MN), 3M Company. U. S. EPA Docket AR- 226- 1030- 0206

353 Olsen, G. W., Burris, J. M., Burlew, M. M., Mandel, J.H., 2003b. Epidemiologic assessment of worker serum perfluorooctanesulfonate ( PFOS) and perfluorooctanoate ( PFOA) concentrations and medical surveillance examinations. J. Occup. Environ. Med. 45, 260–270.

354 Olsen, G. W., Logan, P. W., Hansen, K. J., Simpson, C. A., Burris, J. M., Burlew, M. M., Vororath, P. P., Venkateswarlu, P., Schumpert, J. C., Mandel, J. H., 2003c. An occupational exposure assessment of a perfluorooctanesulfonyl fluoride production site: biomonitoring. Am. Ind. Hyg. Assoc. J. 64, 651–659.

355 Kannan, K., Corsolini, S., Falandysz, J., Fillmann, G., Kumar, K., Loganathan, B., Mohd, M., Olivero, J., Van Wouwe, N., Yang, J., Aldous, K., 2004. Perfluorooctanesulfonate and related fluorochemicals in human blood from several countries. Environ. Sci. Technol.2004, 38,4489-4495

356 Karrman, A., van Bavel, B., Jamberg, U., Hardell, L., Lindstrom, G., 2004. Levels of perfluoroalkylated compounds in whole blood from Sweden. Organohalogen Cpds. 66: 4058-4062.

357 Lindstrom, G., Karrman, A., van Bavel, B., Hardell, L., Hedlund, B., 2004. Levels of persistent fluorinated, chlorinated and brominated compounds in human blood collected in Sweden in 1997-2000. Organohalogen Cpds. 66: 2639-2642.

358 Masunaga, S., Kannan, K., Doi, R., Nakanishi, J., Giesy, J., 2002. Levels of perfluorooctane sulfonate (PFOS) and other related compounds in the blood of Japanese people. Organohalogen Cpds. 59: 319-322.

359 Tittlemier, S., Ryan, J., Van Oostdam, J., 2004. Presence of anionic perfluorinated organic compounds in serum collected from northern Canadian populations. Organohalogen Cpds. 66:4009-4014.

360 Olsen, G., Hansen, K., Stevenson, L., Burris, J., Mandel, J., 2003. Human donor liver and serum concentrations of perfluorooctanesulfonate and other perfluorochemicals. Environ. Sci. Technol. 37: 888-891.

361 Tittlemier, S., Ryan, J., Van Oostdam, J., 2004. Presence of anionic perfluorinated organic compounds in serum collected from northern Canadian populations. Organohalogen Cpds. 66:4009-4014.

362 Inoue, K., Okada, F., Ito, R., Kato, S., Sasaki, S., Nakajima, S., Uno, A., Saijo, Y., Sata, F., Yoshimura, Y., Kishi, R., Nakazawa, H., 2004. Perfluorooctane Sulfonate (PFOS) and Related Perfluorinated Compounds in Human Maternal and Cord Blood Samples: Assessment of PFOS Exposure in a Susceptible Population during Pregnancy. Environ Health Perspect 112:1204–1207.

363 Inoue, K., Okada, F., Ito, R., Kato, S., Sasaki, S., Nakajima, S., Uno, A., Saijo, Y., Sata, F., Yoshimura, Y., Kishi, R., Nakazawa, H., 2004. Perfluorooctane Sulfonate (PFOS) and Related Perfluorinated Compounds in Human Maternal and Cord Blood Samples: Assessment of PFOS Exposure in a Susceptible Population during Pregnancy. Environ Health Perspect 112:1204–1207

364 Kuklenyik, Z., Reich, J., Tully, J., Needham, L., Calafat, A., 2004. Automated solid-phase extraction and measurement of perfluorinated organic acids and amides in human serum and milk. Environ. Sci. Technol. 38: 3698-3704.

365 Calafat, A., Kuklenyik, Z., Reich, J., Butenhoff, J., Needham, L., 2003. Quantitative analysis of serum and breast milk for perfluorochemical surfactants. Organohalogen Cpds. 62L 319-322,

366 Hoff, P., Scheirs, J., Van de vijver, K., Van Dongen, W., Esmans, E., Blust, R., De Coen, W., 2004. Biochemical effect evaluation of perfluorooctane sulfonic acid-contaminated wood mide (Apodemus sylvaticus). Environ. Health Perspect. 112: 681–686.

367 Organisation for Economic Co-operation and Development (OECD), 2002. Hazard Assessment of Perfluorooctane Sulfonate (PFOS) and Its Salts. ENV/JM/RD(2002)17/FINAL. http://www.oecd.org/dataoecd/23/18/2382880.pdf. Accessed 2 Jan 2005.

368 Thayer, K., Klein, J. Gray, S., Houlihan, J., Wiles, R., Greenleaf, T., " PFCs: A Family of Chemicals that Contaminate the Planet," Part 6: PFCs in Animals Worldwide. Environmental Working Group, 2003. http:// www. ewg. org/ reports/ pfcworld/ part8. php. Accessed 3 January 2005.

369 Organization for Economic Cooperation and Development ( OECD). ( 2002) Hazard Assessment of perfluorooctane sulfonate ( PFOS) and its salts ( November, 21, 2002). ENV/JM/RD( 2002) 17/FINAL. Available online http://www. oecd. org/dataoecd/23/18/ 2382880.pdf.

370 Austin, M. E., Kasturi, B. S., Barber, M., Kannan, K., MohanKumar, P. S., MohanKumar, S. M. J. Neuroendocrine effects of perfluorooctane sulfonate in rats. Environ Health Perspect 2003: 111: 12: 1485–1489.

371 Lau, C., Butenhoff, J., Rogers, J., 2004. The developmental toxicity of perfluoroalkyl acids and their derivatives. Toxicol. Appl. Pharmacol. 198: 231–241.

372 Thibodeaux, J., Hanson, R., Rogers, J., Grey, B., Barbee, B., Richards, J., Butenhoff, J., Stevenson, L, Lau, C., 2003. Exposure to perfluorooctane sulfonate during pregnancy in rat and mouse. I: Maternal and prenatal evaluations. Toxicol. Sci. 74: 369-381.

373 Lau, C., Thibodaux, J., Hanson, R., Rogers, J., Grey, B., Stanton, M., butenhoff, J., Stevenson, L., 2003. Exposure to perfluorooctane sulfonate during pregnancy in rat and mouse: II: Postnatal evaluation. Toxicol. Sci. 74: 382–392.

374 Hoff, P., Scheirs, J., Van de Vijver, K., Van Dongen, W., Esmans, E., Blust, R., De Coen, W., 2004. Biochemical effect evaluation of perfluorooctane sulfonic acid-contaminated wood mice (Apodemus sylvaticus). Environ. Health Perpect. 112: 681–686.

375 Thayer, K., Klein, J. Gray, S., Houlihan, J., Wiles, R., Greenleaf, T., PFCs: A Family of Chemicals that Contaminate the Planet. Environmental Working Group 2003. http://www. ewg.org/reports/pfcworld/part4. php. Accessed January 5, 2005.

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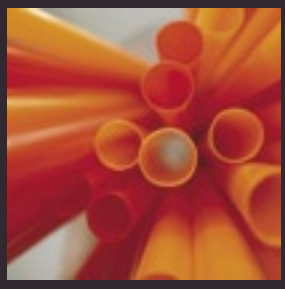

# Sick of Dust

## Chemicals in Common Products— A Needless Health Risk in Our Homes

Pat Costner, Beverley Thorpe & Alexandra McPherson

**The American people deserve to be safe in our own homes and should be able to purchase products without unwittingly exposing ourselves and our children to substances that can cause cancer and disrupt normal and healthy development. This study provides solid evidence that the federal government, U.S. states and U.S. industry must take immediate action to replace harmful chemicals with safe substitutes.**

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