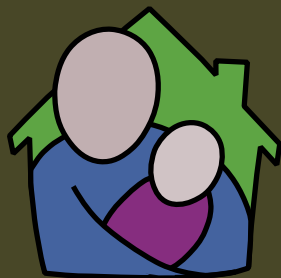




Sick of Dust

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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**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

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
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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 chemical also appears as benzyl-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 environment²⁸ 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 synthetic 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-methyl 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 include 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 plasma⁹⁷ 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 breastmilk¹⁰⁰ 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 octylphenol¹²² and caused the production of an hormone related to milk production (prolactin) both in the adult males¹²³ and after newborn exposure¹²⁴ 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 basal 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 pesticide¹³⁷ 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 lesions¹⁴³ 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's 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 hexachlorobenzene¹⁵¹ 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 in the CDC's upcoming third report.¹⁷⁴

Many other studies have found PBDEs 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 higher²⁰³ than the levels reported previously in Swedish mothers and infants²⁰⁴ 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 lactational 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 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 rats²⁵⁴ 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 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 ten²⁶⁶ 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 been 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 in 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 ≥ 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 urine²⁹⁶

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 U.S., 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 in 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 Korea³⁵⁵ 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 mice³⁶⁶ 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/html/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 their 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. Despite 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-O-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 bioaccumulative 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 Concentrations 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%), transpermethrin (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-n-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 dibutyltin, 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, dibutyltin, 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.

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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 dicyclohexyl 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 ethoxyethoxycarboxylate (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-pyridinol (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 monoethyltin (10 ppb) but did not assay tricyclohexyltin or triphenyltin

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

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

U.K.—Santillo et al.(2003)also assayed monoethyltin (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



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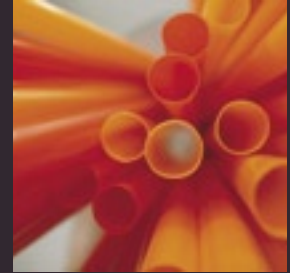
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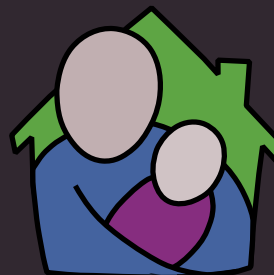


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Pat Costner, Beverley Thorpe & Alexandra McPherson

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