Pollution

in people

A Study of Toxic Chemicals in Washingtonians

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Pollution in People

A Toxic-Free Legacy Coalition Report

May 2006

Acknowledgements

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The Toxic-Free Legacy Coalition is an alliance of organizations across Washington state that rejects the trespass of toxic chemicals in our environment and our bodies. Our vision is to leave our children a legacy of fresh air, clean water, thriving wildlife, and healthy bodies — a Toxic-Free Legacy.

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Pollution in People

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Executive Summary

Toxic chemicals from consumer products, food, and industrial pollution contaminate our bodies. Every person tested had at least 26 and as many as 39 toxic chemicals in his or her body.

Study participant Deb Abrahamson has her blood drawn for testing.

Pollution in People

Last year, ten Washington residents agreed to testing of their hair, blood, and urine for the presence of toxic chemicals as part of an investigative study by the Toxic-Free Legacy Coalition. The Coalition was seeking to determine which chemicals were getting into our bodies, and at what levels, to better understand the potential harm posed by poor regulation of chemicals, and to develop better solutions.

For decades, toxic chemicals in soil, water, air, and sediment have made front-page news. These chemicals range from pesticides like DDT, banned more than thirty years ago but still polluting our soil, to the flame-retarding PCBs and PBDEs building up in salmon and orca whales.

Scientists are now finding these same chemicals in people. The computers we use every day, the cars we drive, and the pans we cook on are leaching toxic chemicals into our homes and into our bodies.

We tested ten Washington residents for six groups of chemicals: phthalates; PBDEs; the heavy metals arsenic, lead, and mercury; perfluorinated chemicals such as those used to make Teflon; pesticides; and the banned but persistent chemicals PCBs and DDT. Our findings reveal that under the current regulatory system, toxic chemicals from consumer products and industrial pollution contaminate each of us and threaten our health.

Key Findings

1. Toxic chemicals from consumer products, food, and industrial pollution contaminate our bodies. Every person tested had at least 26 and as many as 39 toxic chemicals in his or her body. This pollution came from food; everyday household dust; direct contact with products such as personal care items, consumer electronics, and stain-resistant furniture; and from

contaminated soil, air, and water. Many of the chemicals do not break down or do so slowly, and therefore build up in human bodies and breastmilk.

2. The toxic chemicals in our bodies are cause for concern because they can lead to health problems. For some chemicals, the levels we found are at or near those believed to be capable of causing serious problems, such as infertility and learning deficits. Many of these problems can result from being exposed to chemicals at critical points of child development, which can cause permanent damage.

- Every participant was contaminated with phthalates, found in myriad everyday products. The same is true for perfluorinated chemicals, used to make Teflon and stain-protection treatments for paper and textiles.
- Every participant had PCBs in his or her blood, despite a decades-old ban on the chemicals. PCBs from everyday exposures have been shown to cause learning deficits.
- Every participant had PBDEs in his or her blood. Dr. Patricia Dawson had PBDEs in her body at levels close to those that cause reproductive problems in laboratory animals.
- We found a marker for the pesticide carbaryl, considered a carcinogen by the EPA, in five of ten participants: Rev. Ann Holmes Redding, Sen. Lisa Brown, Sen. Bill Finkbeiner, Deb Abrahamson, and Allyson Schrier.
- Three of our ten participants — Denis Hayes, Sen. Bill Finkbeiner, and Karen Bowman — had mercury exposures above the Environmental Protection Agency's "safe" levels.
- Even Laurie Valeriano, toxic chemical expert and regular organic shopper, tested positive for more than two dozen chemicals.

3. State and federal government have failed to prevent the use of harmful chemicals in consumer products, manufacturing processes, and food production. Most chemicals are virtually unregulated, because federal law does not require testing for harmful effects before chemicals are allowed for use in products or manufacturing. Once chemicals are in use, it is extremely difficult for the Environmental Protection Agency (EPA) to restrict them. The law does require pesticide testing, but at the same time it permits the ongoing use of pesticides that can cause cancer, nervous system harm, and other health problems. At the state level, Washington lacks the regulatory structure needed to prevent harmful chemicals from turning up in consumer products, air, water, and people.

Recommendations

Washington state, already a leader in phasing out some dangerous chemicals such as mercury, should take immediate steps to protect the health of its residents by developing a common-sense chemicals policy that ensures only the safest chemicals are used in consumer products, manufacturing, and food production.

Governor Gregoire, the legislature, and agencies should take the following steps:

Come clean with the facts. Require companies to provide data on the health effects caused by the chemicals they produce or use in production. Companies must also be required to make this information available to the public.

Take out the toxics. Develop immediate plans to phase out of products and manufacturing chemicals that can damage children's intellectual

development, harm reproduction, cause cancer, or build up in our bodies.

Switch to safer substitutes. Assist companies in replacing hazardous chemicals with safer substances and practices, using requirements, incentives, and technical assistance.

A Real Solution Is Emerging

A growing number of companies are already switching to safer chemicals and practices in response to mounting scientific evidence and growing consumer demand. Microsoft has switched to safer packaging plastics, the health care community has taken strides to reduce its use of mercury and phthalates, and food companies like Campbell's Soup Company are marketing organic alternatives, produced without harmful pesticides. In the regulatory arena, the European Union has led the way by establishing a forward-thinking chemicals policy that requires testing and moves companies toward safer materials and processes.

This study's findings show that toxic chemicals which can cause cancer, learning problems, and infertility are likely already in all Washingtonians. The Toxic-Free Legacy Coalition calls on Gov. Gregoire, the state Legislature, and state agencies to lead our state into a healthy future with real reform to ensure that our consumer products and food are made in the safest ways possible.

About This Study

In 2005, the Toxic-Free Legacy Coalition and the Washington Toxics Coalition invited ten Washingtonians on an unusual journey: to submit their hair, blood, and urine for toxic chemicals testing. We and our participants sought to uncover the chemical secrets in their bodies—to find out whether the computers, cars, and cosmetics they use could in fact be the source of hidden dangers. We submitted their samples to accredited laboratories to test for heavy metals; pesticides; toxic flame retardants; the plasticizers known as phthalates; the "Teflon chemicals" (perfluorinated compounds); and the banned but persistent DDT and PCBs.

Our participants:

Rev. Dr. Ann Holmes Redding, Episcopal priest, St. Mark's Cathedral

Dr. Patricia Dawson, breast cancer surgeon, Swedish Providence Medical Center

Pam Tazioli, breast cancer survivor and Washington State Coordinator, Breast Cancer Fund

Denis Hayes, Earth Day founder and president, Bullitt Foundation

Senator Lisa Brown, Washington State Senate Majority Leader

Senator Bill Finkbeiner, Washington State Senate

Laurie Valeriano, toxics policy expert, Washington Toxics Coalition

Deb Abrahamson, member, Spokane Tribe and director, Society for Sovereignty, Health, Air, Water, and Land

Allyson Schrier, children's book author

Karen Bowman, registered nurse; consultant, Washington State Nurses Association; and faculty member, University of Washington Nursing Department

We submitted blood, urine, and hair samples to three laboratories that specialize in highly sensitive chemical analysis. For some chemicals,

the laboratories analyzed the samples for the parent compound; for others, such as phthalates and some pesticides, the analysis was for metabolites, or breakdown products. The laboratories reported the results to us in varying units of measurement. For ease of understanding, we have converted the results in most cases to parts per billion (ppb).

We used several methods to determine the implications of the chemical levels found in our participants. Statistician Abbe Rubin analyzed the resulting data. For chemicals that were detected in most or all participants' samples, medians were calculated.[1] Where possible, we compared levels in our participants with values for the U.S. population at large obtained by the Centers for Disease Control and Prevention (CDC). For chemicals not studied by the CDC, we used values from independent scientific studies for comparison purposes.

In addition to determining whether levels in our participants were above or below national averages, we examined studies on the chemicals' toxicity to assess health hazards posed by the levels we found. In some cases, such as with lead, scientists have extensively researched and documented the chemicals' health effects in humans. As a result, we were able to compare levels found in our study directly to levels known to have caused harm in people. In other cases, such as with toxic flame retardants, most available health effects information comes from laboratory animal experiments, not human studies. In these cases, we used animal testing levels to assess potential impacts in people.

Animals and people can vary significantly in their response to toxic chemicals, and either group can be much more sensitive than the other, depending on the chemical. People can also differ from each other in their ability to detoxify harmful chemicals in the body. For these reasons, regulatory agencies typically apply a safety factor when using data from laboratory animals to set regulatory limits. For example, EPA typically applies a safety factor of ten to account for differences between animals and humans, and an additional factor of ten for differences among people. The agency may also apply a safety factor of up to ten to account for other uncertainties.

For both people and other animals, the most sensitive time of life is generally during development: before birth and in early childhood. In this study, we compared our participants' results to the lowest levels in human or animal studies where health effects have been seen. In some cases, such levels are those that result in harm to the offspring when the mother is exposed during pregnancy. None of our participants was pregnant at the time of sampling. To protect public health, however, it is necessary to maintain levels in all individuals that are below levels that would harm a developing child. Therefore, we use the levels we detected in our ten participants as a barometer of the degree of danger faced by the rest of the residents of Washington.

Report reviewers included David Cosman, Ph.D., Nancy Dickeman, Philip Dickey, Ph.D., Steve Gilbert, Ph.D., Kim Hooper, Ph.D., Kristin Hyde, Jen Lamson, Sharyle Patton, Abbe Rubin, Ph.D., Ivy Sager-Rosenthal, Dr. Ted Schettler, Margaret Shield, Kristin Schafer, Gregg Small, Pam Tazioli, Heather Trim, and Laurie Valeriano. Elena Conis edited the manuscript.

[1] In order to be consistent with methods used by the CDC, to calculate medians, values for samples in which the chemical was not detected were set at the detection limit divided by the square root of two.

From Toys to Body Lotion:

Phthalates, the Everywhere Chemicals

Pam is encountering phthalates in her daily life: from the vinyl wallpaper in her apartment, the food she eats, her follow-up medical care, and the cosmetics and personal-care products she uses.

Pam Tazioli, Washington State Coordinator, Breast Cancer Fund.

Pam Tazioli is the consummate Washington woman. Raised in Seattle, she grew up swimming in Puget Sound, digging clams on Northwest beaches, and hiking in the Cascades. She went on to start two day care centers and help children with special needs. But Pam was forced to examine her life at age 47, when she was diagnosed with two forms of breast cancer that required extensive treatment. A double mastectomy and six rounds of chemotherapy later, Pam is now passionate about her health and diligent about getting the care she needs to keep the cancer from coming back. As the Washington State Coordinator for the Breast Cancer Fund, Pam also educates women about how to reduce breast cancer by addressing environmental causes like pesticides and other toxic chemicals.

Pam has particular concerns about a class of plasticizing chemicals known as phthalates (pronounced THAL-ates), used widely in consumer products like cosmetics, vinyl flooring, and shower curtains. Phthalates are nearly ubiquitous in the medical devices Pam got to know intimately during her treatment. Her fears about the chemicals were realized when she received her Pollution in People study results: Pam had some of the highest levels of phthalates in the study, with a total of 467 ppb in her urine. Since phthalates don't build up in the body, it's unlikely that this was a remnant of her cancer treatment. Instead, her levels indicate Pam is encountering phthalates in her daily life: from the vinyl wallpaper in her apartment, the food she eats, her follow-up medical care, and the cosmetics and personal-care products she uses.

Our results show that Pam is not the only one unable to avoid phthalate exposure. We tested for seven forms of phthalates, which vary in their toxicity and use.[2] Most forms were

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[2] *We tested for seven phthalate monoesters, which are breakdown products of five phthalate diesters used in products.*

found in all participants; five participants tested positive for all seven forms. Because the CDC tests for phthalates, we were able to compare the levels in our participants with levels found in a large number of people nationwide. For most of the forms, we found levels much higher than those in the population at large.

Figure 1: Levels of DEHP Metabolites in Ten Washingtonians

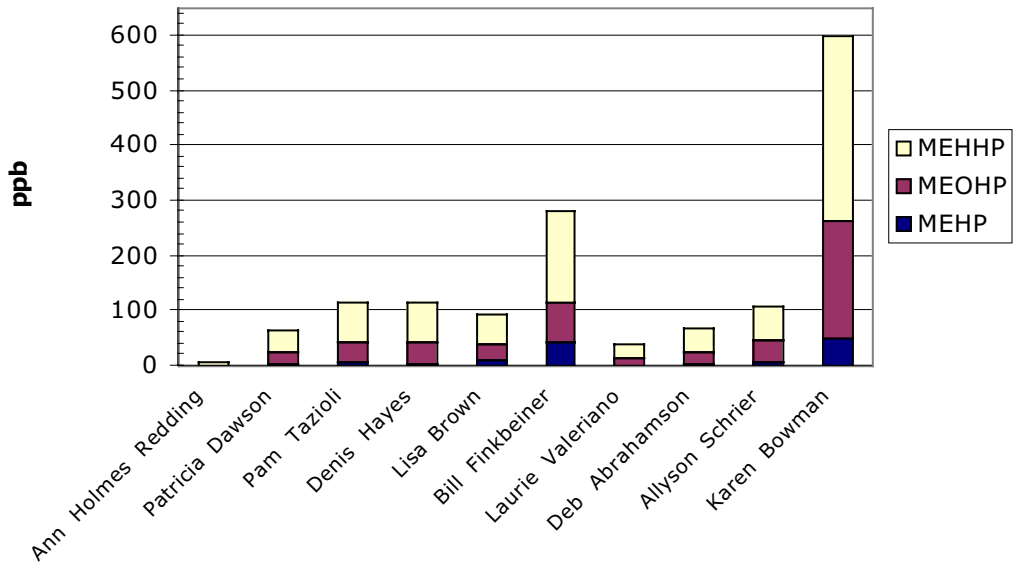


Figure 1: Three breakdown products of the phthalate DEHP were measured in urine: MEHP, MEOHP, and MEHHP.

Figure 1 shows our participants' exposures to the phthalate known as DEHP, which is widely used and, among phthalates, appears to be most toxic at low levels of exposure. Median levels among our participants for the three DEHP metabolites (MEHP, MEOHP, and MEHHP) were 7.7, 31.9, and 58.6 ppb; median levels in the CDC study were lower, at 4.1, 17.7, and 12.2 ppb (CDC 2005). We can't be sure why the levels in our participants were higher, but there are two possibilities. Our samples were all taken at the first morning void, while the CDC took samples throughout the day, potentially creating a difference. Our participants or Washingtonians in general may, however, have greater exposures than others in the United States.

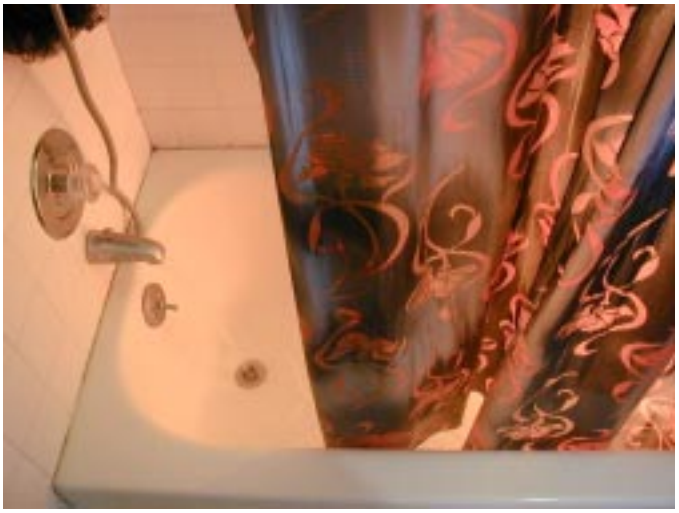
The Pollution in People participant at the top of the list for DEHP metabolites is an occupational and environmental nurse whose job takes her to foundries, shipyards, biotech companies, and a medical supplies warehouse. The chemicals she picks up at these workplaces include heavy metals, solvents—and apparently phthalates. Karen Bowman's career in nursing spans more than two decades, and the tools of her trade make heavy use of phthalate-containing plastics. Medi-

Karen Bowman, registered nurse, Washington State Nurses Association.

cal devices made of polyvinyl chloride (PVC), such as gloves, tubing, and intravenous bags, contain 20 to 40% DEHP. Karen's overall DEHP metabolite levels (603 ppb) were more than twice those of any other participant.

Phthalates: How We Get Exposed

The widespread exposures among our study participants are not a surprise. Phthalates are found in an array of consumer products most of us use every day: cosmetics and personal-care



products, plastic toys, and vinyl flooring, wallpaper, and shower curtains. The chemicals are often an unlisted ingredient in personal-care products such as lotions, shampoo, perfume, and aftershave, where they are used to carry fragrances and can thus simply be labeled "fragrance," despite the fact that they may make up 20% or more of the product (ATSDR 1995). In nail polish, phthalates are included to prevent chipping (EWG 2000).

In plastic, phthalates can make up as much as 80% of a product. Since they're not chemically bound to the plastic, up to 50% of the phthalates can leach from a product over its lifetime (DiGangi 2002). Phthalates are used most often as plasticizers, or softeners, in PVC products; in fact, such use accounts for 90% of all phthalate use. PVC, the second-most commonly used plastic worldwide (CHEJ 2004), is found in an incredible range of products, from pipes and building materials to packaging and toys.

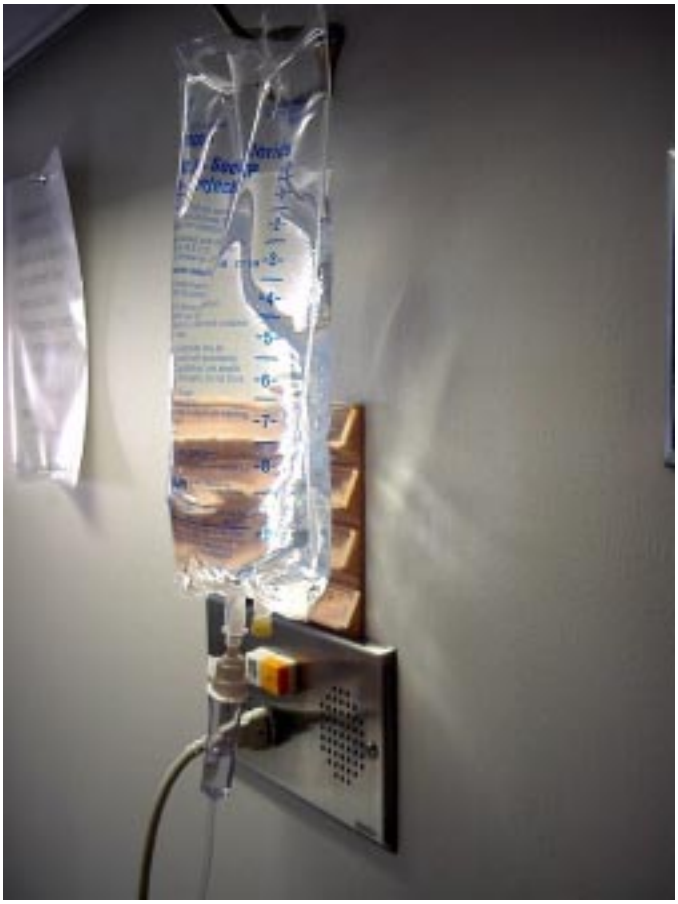
Scientists have shown that phthalates leach from medical devices, such as tubing and blood bags. In a 2005 study, Harvard University researchers measured phthalate levels in newborns at two Boston-area hospitals to determine whether those who received feedings, respiratory therapy, and other treatments with PVC products received higher phthalate exposures than those treated with non-PVC products. The study found that the newborns in the hospital where PVC was used had higher phthalate levels than newborns in the hospital using predominantly other plastics (Green 2005). Newborns that had the most intensive treatment with phthalate-containing products had levels five times those of newborns with less-intensive treatment.

Male Reproductive Problems Top Health Concerns

For years, evidence that exposure to phthalates may be leading to health problems has been accumulating. Animal tests have revealed a wide array of phthalate-related health effects, most of them reproductive: small or otherwise abnormal testes, hypospadias (abnormal urinary openings), and undescended testes (Gray 2000). These effects occur at exposure levels higher than those

expected for people today; however, some of the most highly exposed people have phthalate levels greater than the no-effect, or safe, level in animal tests. For example, some children in neonatal intensive care have DEHP doses greater than levels considered safe by the FDA or EPA. In humans, phthalates cross the placenta to reach the growing fetus. They're also present in breastmilk.

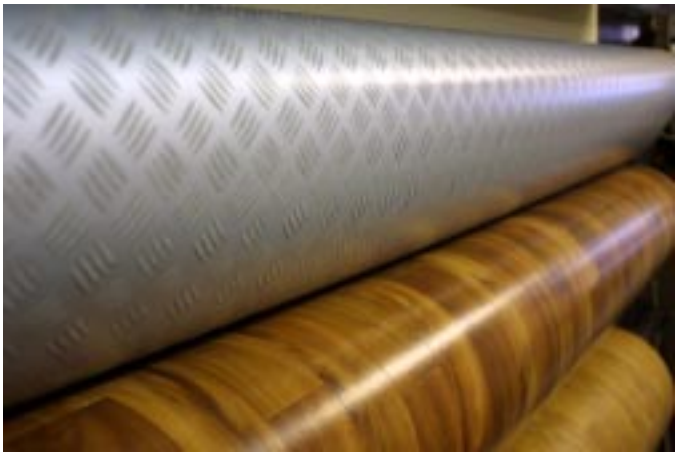
Researchers believe that the phthalate forms that have these reproductive effects, such as DEHP and DBP, act by reducing levels of testosterone and important growth factors in young males. Damage occurs when males are exposed in utero — that is, when mothers come into contact with phthalates during pregnancy. Phthalates are not all equally toxic in this regard, but CDC testing indicates that exposure patterns are of concern. In particular, women have higher exposure to DBP, the harmful phthalate form found in nail polish and other cosmetics, than do men. These women are also exposed to other phthalates, with similar effects, creating the potential that a significant segment of the population may have total phthalate levels high enough to cause harm.



A 2005 study that looked at mothers' phthalate exposure and reproductive organs in their baby boys provides evidence that current exposure levels may indeed be having an impact on boys' health. University of Rochester researcher Shana Swan and colleagues found that baby boys whose mothers had greater exposure to phthalates were more likely to have altered genital development. These boys had a changed penis location and a smaller average penis size and were more likely to have undescended testicles (Swan 2005). These effects are consistent with a "phthalate syndrome" observed in male rodents with phthalate-induced feminized traits. Future studies will shed more light on the extent to which phthalates are already affecting baby boys' reproductive development.

Phthalate exposure has also been linked to lower sperm counts, reduced sperm motility, and damaged sperm in men (Duty 2003). The plasticizers may also affect women's fertility; animal studies show that females exposed to the chemicals experience more difficulty becoming pregnant (Hauser 2005).

Other phthalate-related health concerns include liver and kidney damage as well as asthma (ATSDR 2002). Researchers have found that children in homes with greater levels of phthalates are more likely to have asthma (Bornehag 2004). In adults, phthalate exposure has been associated with reduced lung capacity, with the magnitude of effects similar to that of tobacco smoke (Hoppin 2004).



Policy Changes Needed

The reality of the reproductive effects caused by phthalates at today's exposure levels highlights the urgent need to eliminate the plasticizers from products. Addressing two types of products containing phthalates—PVC and cosmetics—would have a major impact in reducing exposure.

A number of companies, hospitals, and government agencies have taken steps to switch to alternative materials and phase out PVC use. Microsoft, for example, has now completely ended the use of PVC in its packaging material, and Kaiser Permanente has pledged to reduce PVC wherever possible in new construction. The health care provider has also worked with vendors to develop PVC-free carpeting and wall coverings. Evergreen Hospital in Kirkland has eliminated most PVC products from its neonatal intensive care unit, as has the Special Care Nursery at Group Health Cooperative in Seattle. Seattle and Olympia have both passed resolutions committing them to seeking alternatives to PVC for city operations.

Based on existing evidence, the European Union passed legislation banning some phthalates in cosmetics in 2003, and has kept three phthalates out of toys since 1999. Although many uses of phthalates are essentially ungoverned in the United States, cosmetic and medical uses are regulated by the Food and Drug Administration (FDA). The FDA has not taken steps to ban phthalates. It has, however, urged medical providers to switch to alternative products that do not contain phthalates. Cosmetics companies, on the other hand, have taken action on their own: Revlon, L'Oreal, and other major companies are phasing phthalates out of nail polish. Three hundred companies, including The Body Shop and Burt's Bees, have pledged to eliminate phthalates from their products in response to requests from the Campaign for Safe Cosmetics.

Reducing Your Exposure to Phthalates

You can reduce your and your family's exposure to phthalates by avoiding PVC and purchasing products from companies that have eliminated phthalates.

PVC-free building products. Steer clear of vinyl windows and doors and choose wood instead. For flooring, choose linoleum, cork, bamboo, or wood instead of vinyl. Adhesives, caulk, grout, and sealants may also contain phthalates. You can check for phthalate ingredients in these products using the National Institutes of Health's Household Products Database: www.householdproducts.nlm.nih.gov/ (search for 'phthalate' as an ingredient).

PVC-free shower curtains. Avoid vinyl shower curtains in favor of natural fibers, polyester, or nylon.

PVC-free packaging. Look at the recycling symbol on products you purchase in plastic packaging. Plastics marked with the #3 symbol contain PVC.

PVC-free toys. Toymakers Brio, Chicco, Early Start, Evenflo, Gerber, Lego, Prime Time, Sassy, and Tiny Love have pledged to stop using PVC.

PVC-free food storage. Buy plastic wrap and bags made from polyethylene, such as GLAD. For food storage, use glass containers or plastic containers marked with recycling symbols other than the #3.

Phthalate-free cosmetics. Check ingredient lists and avoid products listing 'fragrance' or phthalates. A wide variety of personal-care products may contain phthalates, including perfume, cologne, after-shave, deodorant, soap, hair and skin-care products, and makeup. Choose products from companies that have signed the Compact for Safe Cosmetics: a list is available at www.safecosmetics.org.

Burning Problem:

Toxic Flame Retardants in People and Wildlife

Somehow, her body has absorbed enough of the toxic flame retardants known as PBDEs to make her levels three times the national average.

Dr. Patricia Dawson, breast cancer surgeon, Swedish Provedence Medical Center.

Every day for the past twelve years, Dr. Patricia Dawson has risen at 5 a.m. to help women face breast cancer. Women from throughout the Northwest, hoping to purge their bodies of cancer and put their lives back together, come to Swedish Medical Center's Comprehensive Breast Center, where Patricia is a surgeon. In Patricia's line of work there is no denying disease, and she grapples every day with questions about the blame that can be placed on toxic chemicals and other environmental causes of cancer.

By submitting her hair, urine, and blood for chemical testing, Patricia sought to learn more about how our daily decisions and our government's policies on toxic chemicals directly affect our lives. She was surprised by what her test results revealed: she is ingesting pesticides along with the nutrients from her otherwise healthful diet. Her body carries DDT and PCBs decades after these chemicals were banned. But perhaps most disturbing was the fact that somehow, her body has absorbed enough of the toxic flame retardants known as PBDEs to make her levels three times the national average.

Each of the ten Pollution in People participants tested positive for PBDEs, with levels ranging from 29 to 147 ppb (as measured compared to total fat in blood samples). With 147 ppb PBDEs in her blood, Patricia had the highest level in the group. The CDC has not included PBDEs in its ongoing program to test U.S. residents for toxic chemicals, but Tom McDonald, at the time a California EPA scientist, recently compiled six studies (with a total of 191 tested individuals) and found a median level of 47.9 ppb in tested women nationally (McDonald 2005), a level comparable to the median in our study, 47.5 ppb.

McDonald also back-calculated from the measured levels to estimate daily exposures for these women and compare them to

exposure levels that caused harm in laboratory animals. His findings indicate that current levels in U.S. women are at or approaching those that could harm a developing fetus. Levels of PBDEs that caused behavioral problems in mice were just 4 to 11 times those of the most exposed U.S. women (those in the top five percent of tested women, with levels of 302 ppb and above). Rats suffer fertility problems—reduced sperm counts and changes to ovary cells—at levels at or *lower* than those of the most exposed women. Patricia's level, at 147 ppb, is uncomfortably close to the approximately 230 ppb in affected rats. Generally, agencies seek to ensure a safety margin of at least 100, a margin that is much greater than that which exists today for many women.

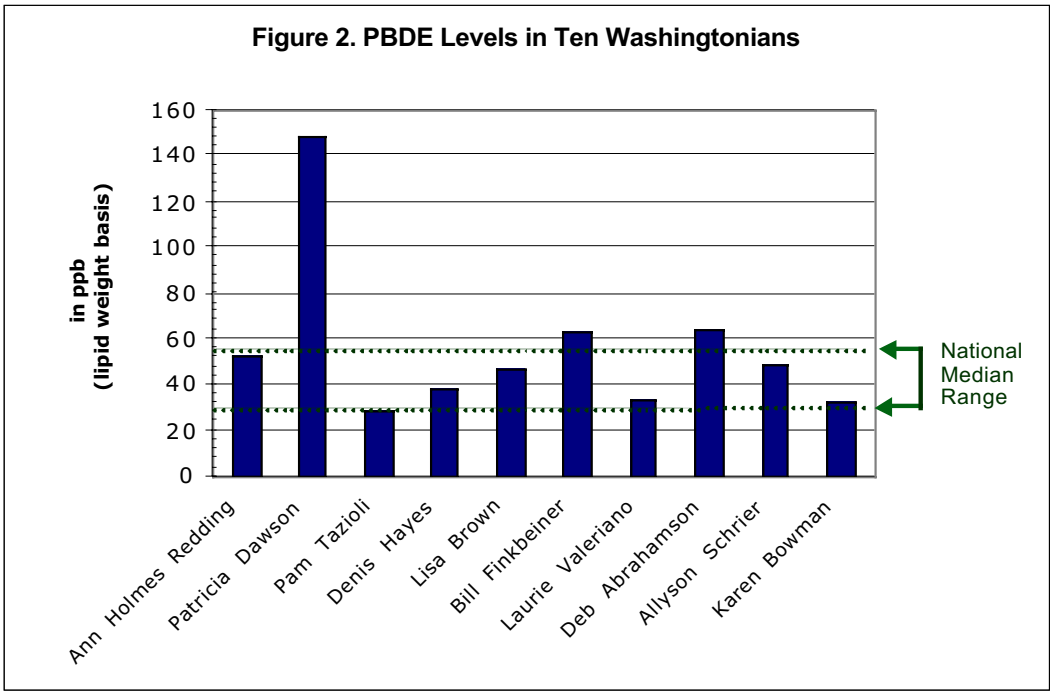


Figure 2: PBDE levels, measured in blood serum and expressed on a lipid weight basis.

Figure 2 shows PBDE levels in Pollution in People participants as compared to the national median.

Out of Our Mattresses, Into Our Bodies

Although PBDEs are used around the world, the largest volumes are used in the Americas: an estimated 33,100 metric tons in 2001 alone (WDOE 2005). The flame retardants, developed 30 years ago, have been used heavily in the production of furniture, textiles, and electronics. Two of the commercial formulations of PBDEs, known as penta and octa, were once widely used in foam and plastic products from upholstered furniture to kitchen appliances. But in 2004, industry voluntarily ended production in the United States in response to new information on high levels in breastmilk. EPA subsequently issued a rule requiring companies to notify the agency before beginning any significant new uses of penta or octa.

Still in production, however, is the deca formulation of PBDEs, long the most widely used, with 50 million pounds going into products each year. Deca is employed primarily in plastics for electronics, such as television and computer housings, as well as in textiles. Its use may in-

crease with the introduction of new, more rigorous standards for fire resistance for upholstered furniture.

The first hint that the chemicals were building up in the environment came in 1981, when PBDEs were found in Sweden's River Viskan (Sjödin 2003). Subsequent studies found that environmental levels were rising at an alarming rate across the globe. Between 1981 and 2000, levels in Arctic seals increased tenfold (Ikonomou 2000). From 1988 to 1999, levels in Beluga whales in the St. Lawrence Estuary increased exponentially, doubling every three years or less (Lebeuf 2004). And during roughly the same period (1989 to 1998), levels in San Francisco Bay harbor seals doubled every 1.8 years (She 2002).

Puget Sound appears to have an especially dire PBDE problem. Recent measurements have found that harbor seals, particularly those that live near Seattle, have elevated levels of PBDEs (Ross 2006). They also found that Puget Sound's Chinook salmon—the key food source for endangered orca whales—have the highest levels among tested fish, which included herring, sole, rockfish, and lingcod (O'Neill 2006).

Because of their presence in such a wide variety of consumer products, each of us encounters PBDEs daily. Many products made with PBDEs, such as furniture, are used for many years and shed the chemicals over the course of their lifetimes. A number of studies have found PBDEs in house dust as well as indoor air, which is considerably more contaminated with these chemicals than outdoor air (Stapleton 2005, Sjödin 2004, Butt 2004). We're also likely consuming the flame retardants with every meal: studies in the U.S., Europe, and Asia have found PBDEs in fish, meat, eggs, fruits, vegetables, and infant formula (Schecter 2004, Bocio 2003).

PBDEs made the headlines in 2003, when an Environmental Working Group study found previously unheard of levels in U.S. women's breastmilk. Recent studies have estimated that

the largest percentage of PBDE exposures in children, particularly infants and toddlers who are not breastfed, comes from house dust (Jones-Otazo 2005). Breastfed infants, however, have the

highest exposure of any age group (Health Canada 2004). Scientists have even found PBDEs in umbilical cord blood, revealing that today's newborns are exposed even before they are born (Health Canada 2004, Mazdai 2003).

Allyson Schrier, children's book author.

That PBDEs were in breastmilk was news to Allyson Schrier, who had never even heard of the chemicals when she was pregnant with and nursing her two sons. But when her son Aidan was diagnosed as having learning disabilities, she began a quest for environmental chemicals that could interfere with brain development. Allyson was outraged to learn years later that breastmilk was contaminated with toxic flame retardants linked to learning and memory problems. In 2006, she brought her son and three other children to meet with her legislators and ask for a ban on PBDEs. Now, laboratory results have confirmed what she suspected: PBDEs are in her body, at a level of 48.3 ppb.

Slow to Learn

Our daily dose of PBDEs may be enough to keep our children from learning at their potential. Studies in laboratory animals have found that PBDEs profoundly and permanently affect the developing brain at levels frighteningly close to those in today's most exposed women. In a series of studies on rodents, rats and mice exposed to a single dose of PBDEs 10 days after birth had difficulty adjusting to new environments and negotiating mazes, indicating effects on learning, behavior, and memory (Ericksson 2001). A 2003 study found similar effects in mice exposed to deca (Viberg 2003).

While long-term studies on PBDEs' effects in humans have not been conducted, animal studies suggest their effects are eerily similar to those of PCBs, their close chemical cousins. Long-term studies of children exposed to PCBs show that

Breastfeeding is Still the Best for Babies

While researchers have found PBDEs and other chemicals in breastmilk, mothers should not be discouraged from breastfeeding. Breastmilk is the best nutrition for babies. Infants who do not breastfeed or do so for only a short time have more acute illness such as ear, lung, and urinary infections. Exposure to foods other than human milk in the first few months of life can increase the risk of life-long autoimmune illnesses. Without breastfeeding, infants do not receive optimal nutrition, important hormones, protective immune factors, and promoters of brain development.

Formula feeding does not eliminate children's exposure to toxic chemicals. Children are exposed to toxic chemicals through other food, the household environment, and from contaminants that cross the placenta while a fetus is still developing.

For more information, see *Why Breast-Feeding is Still Best for Baby* by Physicians for Social Responsibility at http://psr.igc.org/BFeasyeng2pg.10.18.pdf.

early exposure leads to deficits in learning (Schantz 2003).

PBDE exposure may also affect thyroid hormone, which is essential for proper brain development in the fetus. In animal studies, both penta and octa have been shown to reduce levels of thyroid hormone (Zhou 2002, Zhou 2001) and liver toxicity (Darnerud 2001). They've also been shown to cause bone malformations and reduced weight gain as a result of prenatal exposure (Darnerud 2003).

Scientists are beginning to study the effects of PBDEs on wildlife. Recent research by the National Marine Fisheries Service has found that PBDEs alter fish thyroid hormone levels, delay hatching, and retard growth (Lema 2006). Scientists have expressed concern that PBDEs may threaten the health of orca whales, particularly when combined with effects from PCBs (Ross 2006).

Many of these toxicity studies have been conducted on the phased-out PBDE formulations. But researchers have produced considerable evidence that, once in the environment, the still widely used deca formulation gets broken down into chemicals that, like those in penta and octa, accumulate in human and animal tissue. Four studies, examining the breakdown of deca by sunlight and by living organisms, found that deca degrades into some of the PBDEs found in the penta and octa formulations (Söderstrom 2004, Bezares-Cruz 2004). A study of the degradation of deca in house dust found rapid breakdown and concluded that 83% of the deca converted to other PBDEs, some of which are more persistent and toxic than deca itself (Stapleton 2005). Use of deca continues at very high levels, and recent testing has typically detected more deca than the other formulations in the indoor and outdoor environment (Sharp 2004, Song 2004).

Policy Changes Needed

Electronics, furniture, and other companies have proven that these products can be made fire-safe without PBDEs. Furniture-maker IKEA has found ways to design its furniture so that flame retardants are not needed. Major U.S. electronics companies, such as HP and Dell, have turned away from PBDEs and use alternate flame retardants or non-flammable materials.

Eight U.S. states have passed legislation to ban penta and octa PBDEs, and several states have passed laws to study deca. In 2003, the European Union issued a directive to phase out PBDEs by July 2006; however, deca is currently exempted from the phaseout until 2010, although the European Union may rescind the exemption at any time.

Washington state agencies, at the direction of an executive order signed in 2004, have undertaken a major effort to study PBDEs and develop a phase-out plan. The Washington State Departments of Health and Ecology published a final plan in 2005 that recommended phasing out all forms of PBDEs, including deca, as long as safer alternatives are available. The plan identified several alternatives that, unlike PBDEs, do not persist in the environment or build up in people and wildlife.

In Washington, the Departments of Ecology and Health have called for a phaseout of all forms of PBDEs. The agencies, together with members of the Toxic-Free Legacy Coalition, supported legislation in 2006 that would have ended the manufacture and sale of all products containing penta and octa after 2007; computers and televisions made

with deca after 2010; and residential furniture made with deca after 2012. The legislation would also have required state agencies to purchase PBDE-free products and allow for the Department of Ecology to study actions needed to address existing PBDE contamination. Bills with these provisions passed the Senate and House Rules Committees, but legislative leaders did not bring them to a vote.

Along with the legislation, the Toxic-Free Legacy Coalition is calling for an end to the use of PBDEs in all consumer products and for manufacturers to disclosure the use of any chemical flame retardants. Legislation is planned for 2007 to phase out PBDEs.

Reducing Your Exposure to PBDEs

You can take the following steps to reduce your family's exposure to PBDEs:

Buy PBDE-free furniture. Choose furniture that does not contain PBDEs, which are often used in furniture upholstery and foam. IKEA does not use PBDEs in its products, and Serta states that their mattresses produced after 2005 do not contain PBDEs. Other retailers offering PBDE-free products include:

Greener Lifestyles (couches and chairs):
www.greenerlifestyles.com

Soaring Heart (mattresses and futons):
www.soaringheart.com

Furnature (couches and chairs):
www.furnature.com

Bean Products (couches and chairs):
www.beanproducts.com

For more information on companies offering PBDE-free products, see:

Safer Products Project: www.safer-products.org

Smart Shopper's PBDE Card:
www.thegreenguide.com

If you cannot find information on whether a manufacturer uses PBDEs, contact the company directly.

If you already own furniture that contains PBDEs, cover and seal any rips in upholstery, and consider replacing old items where foam is exposed, loose, and crumbling. Cover mattresses with allergen-barrier casings to reduce the amount of PBDE-laden dust that they release.

Make electronics PBDE-free. Choose electronics made with alternatives to PBDEs, available from Canon, Dell, HP, Intel, Erickson, Apple, and Sony.

Avoid farmed fish. European and U.S. farmed salmon have particularly high levels of PBDEs. Choose wild salmon instead.

Reduce animal fats. Choose lean meat and poultry cuts and low-fat dairy products. Cut visible fat off meat and poultry before cooking, and choose lower-fat cooking methods, such as broiling, grilling, roasting, or pressure-cooking.

Heavy Metals:
A Centuries-Old Story

Denis generously donated enough hair for the Pollution in People study to discover that he has accumulated a significant amount of mercury in his body—in fact, the highest level in our group.

Denis Hayes, president, Bullitt Foundation.

Since 1970, when he coordinated the first Earth Day, Denis Hayes has been dedicated to making our planet a healthy place. Raised in Camas, Washington, marked by the grandeur of the Columbia River and the pollution of a major pulp mill, he grew up with an appreciation for nature and an understanding of how humans can degrade it.

Today, Denis is the chair of the International Earth Day Network; he also presides over the conservation-minded Bullitt Foundation, and in various posts has spent decades promoting renewable energy sources around the world. His accolades are impressive: he's been named a "Hero of the Planet" by Time magazine, awarded the Sierra Club's John Muir Award, and dubbed one of the twentieth century's environmental heroes by the National Audubon Society.

Although he recently began sporting a buzz cut to please his wife, Denis generously donated enough hair for the Pollution in People study to discover that he has accumulated a significant amount of mercury in his body—in fact, the highest level in our group. At 2020 ppb, his hair mercury level is more than three times the median for his age and gender (Environmental Quality Institute 2005).[3] Most likely his high level results from his fish-rich diet, not uncommon in the Pacific Northwest. Figure 3 shows our study participants' levels, which ranged from 59.5 to 2020 ppb, with a median of 887 ppb.

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[3] *Participants in this study were self-selecting volunteers, and may not reflect the age and racial/ethnic background distribution in the general population.*

We also tested our participants for lead and arsenic. Four tested positive for arsenic; one, nurse Karen Bowman, tested positive for lead.[4]

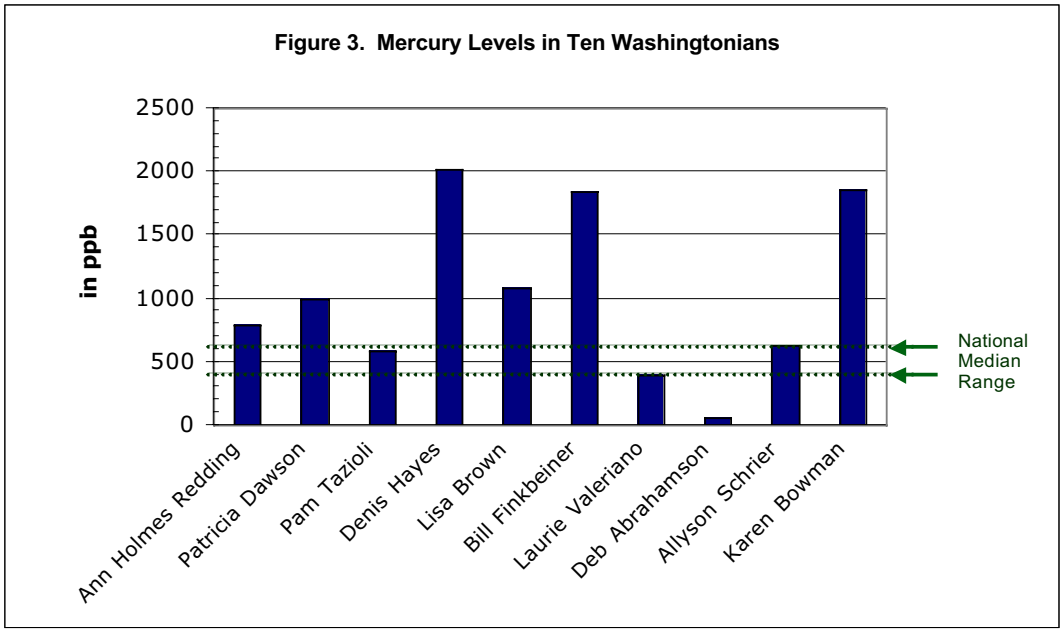

Figure 3: Mercury levels in participant hair.

Karen also had the second-highest mercury level, and the highest among the study's women. Her level of 1880 ppb puts her above EPA's reference dose, or "safe" level, for women of childbearing age (1100 ppb) (NAS 2000). The National Academy of Sciences has concluded that exposures above this level may harm neurological development in the developing fetus. Bill Finkbeiner's mercury also exceeded the safe level, and Lisa Brown's mercury, at 1080 ppb, is just below the benchmark.

For centuries, humans have known that heavy metals such as mercury, lead, and arsenic can seriously harm our health. Lead's effects on the brain were noted as early as the second century B.C.E., and arsenic has been notorious as a poison since the Middle Ages. The ancient Romans noted mercury's harmful effects when slaves mined the metal in Spain. The Incas used mercury to extract gold in the 1500s, and despite a well-developed reputation for harming the brain and causing birth defects, mercury continues to be used in mining, in addition to other industries and consumer products. Mercury, lead, and arsenic are naturally occurring elements, but our bodies don't need them in any way. And at very low levels, all three can be toxic.

A Fish Story

Today, mercury is found in electrical fixtures, switches, medical equipment, and amalgam fillings; the metal is also used in battery, chemical, and paper production, and, in many countries, gold extraction (Gilbert 2004). In Washington, mercury is emitted by the coal-burning power plant in Centralia, and by manufacturers, oil refineries, medical waste disposal facilities, dental offices, and cremation facilities (Ecology 2003). The largest amount of Washington's environmental mercury from in-state sources likely comes from the combustion of diesel, jet fuel, and heating oil. Mercury from these sources often circulates in the atmosphere and deposits on land

[4] *The laboratory detection limit for lead was 3 µg/dL, and 10 ppb for arsenic.*

and water, where it increases in concentration as it moves up the food chain.

Bacteria in water convert mercury to toxic organic mercury, which builds up in fish. When we eat fish—particularly long-lived fish that have accumulated mercury from a lifetime of eating other fish—the mercury tags along and settles in our bodies. The Washington State Department of Health has issued warnings about eating fish from

Lake Whatcom, Lake Roosevelt, Sinclair Inlet, the Duwamish River, and Eagle Harbor because of mercury contamination (WDOH 2006). The department has also warned women of childbearing age and children under six not to eat any shark, swordfish, tilefish, king mackerel, or tuna steaks (WDOH 2001).

Getting the Lead Out

More so than mercury, lead turns up in a frighteningly large array of consumer products, from art supplies and automobile components to PVC clothing, building materials, toys, lunchboxes, and even candy. Gasoline and paint are now lead-free in the United States and many other countries, but lead continues to be added to certain hair dyes and specialty paints (FDA 2002). And despite a 1978 ban, lead paint on the walls of old homes and buildings continues to be a primary source of lead exposure for children. Because of its slightly sweet taste, children commonly eat peeling lead-paint chips if they have access to them.

Most of us are exposed to lead through direct contact with lead-containing products, drinking lead-contaminated water, and through house dust into which lead from indoor and outdoor sources has settled. In certain areas and homes, contaminated soil and dust from lead paint are a significant source, particularly for children. Two of Washington's now-shuttered smelters (facilities that process metals) emitted lead that deposited in soil. Soils are also contaminated by past use of the pesticide lead arsenate in orchards. Lead then enters our homes when we track it in on the bottom of our shoes.

Lead in the workplace can cause elevated levels both in the worker and in the worker's family members (MMWR 2001). Workers can bring lead home on clothing and shoes. In our study, because the test was only sensitive enough to pick up relatively high levels, the only participant who tested positive was nurse Karen Bowman. Karen has regular occupational exposure through her nursing visits to workplaces such as machine shops and metal fabricating companies.

Arsenic and Old Wood

Arsenic-treated wood is a fixture in many of our homes, where it has commonly been used to construct tough, long-lasting decks, fences, and play structures. But arsenic leaches from treated lumber and rubs off the wood when people or animals come in contact with it.

In 2002, after various risks assessments indicated elevated childhood cancer risk from arsenic exposure, EPA reached an agreement with manufacturers to phase out the use of arsenic for residential wood treatment. However, many homes still have arsenic-treated wood in place, as well as soil contaminated from this lumber. As with lead, Washington's soils are tainted in many areas with arsenic from smelters and pesticides. Arsenic continues to be used in other products, too, including paints, dyes, metals, soaps, and drugs (USEPA 2005). Other major sources of arsenic, depending on diet and location, are drinking water and seafood. While seafood may contain large amounts of arsenic, most of its arsenic is believed to be in the less-toxic inorganic form. The test in our study analyzed for the toxic, inorganic form found in drinking water and other sources.

How Metals Threaten Our Health

Lead, mercury, and arsenic may occur naturally in the earth, but just because they're natural doesn't mean they're harmless. And because we have found such countless uses for these metals throughout the ages, today's children, adults, and wildlife must contend with their effects on health.

Like many other chemicals, lead and mercury exact their most devastating toll on the developing brain. Children exposed to lead at a young age are more likely to suffer from shorter attention spans and are less able to read and learn than their peers (Gilbert 2004). A recent analysis of multiple studies by scientists at the Cincinnati Children's Hospital found that lead has a significant effect on brain development at blood levels below 10 μg/dL, the current level at which public health agencies take action. In fact, they found

no level that did not have an impact on intellect as measured by a decrease in IQ scores (Lanphear 2005).

Research on mercury shows similar effects. Mass poisoning episodes, like that in Japan's Minamata Bay community in the 1950s, have proven that mercury can cause birth defects including mental retardation and deformed limbs, and studies on children with above-average mercury exposures show that the metal can affect their ability to learn. In a study published in 2005, Harvard Medical School researchers tested mercury in new mothers' hair at the time of childbirth and found that infants exposed to less mercury in the womb scored better in intellectual tests (Oken 2005). Arsenic may also affect the developing brain: a recent study found that children with greater exposure to arsenic had deficits in intelligence compared to their less-exposed peers (Wasserman 2004).

Lead, mercury, and arsenic do not stop at harming the developing brain. Their health effects have been well-studied, and are summarized in Table 1. It should be noted that many of these effects occur only at relatively high exposure levels.

Table 1: Common Health Effects of Lead, Mercury, and Arsenic

Metal	Health Effects
Lead	behavioral problems
	high blood pressure, anemia
	kidney damage
	memory and learning difficulties
	miscarriage, decreased sperm production
	reduced IQ
Mercury	blindness and deafness
	brain damage
	digestive problems
	kidney damage
	lack of coordination
	mental retardation
Arsenic	breathing problems
	death
	decreased intelligence
	lung and skin cancer
	nausea, diarrhea, vomiting
	peripheral nervous system problems

Sources: ATSDR, Gilbert 2004

Policy Changes Needed

Heavy metals have a long history of industrial and personal use—and just as long a history of harming human health. Only relatively recently have people begun to take action to curtail their use. Decades of evidence on lead's health effects were amassed before the metal was banned in paint and gasoline, and lead is still allowed in many consumer products. Arsenic-treated wood was extremely widely used before manufacturers agreed to phase it out. Recently, a number of states, including Washington and Oregon, have passed legislation to address mercury use in products such as thermometers and thermostats, but major sources like coal burning continue.

Local advocacy and legislation have made a difference in reducing continued pollution with these metals. In 2005, an independent consumer watchdog group found high levels of lead in children's soft vinyl lunchboxes. In response to a request by the Washington Toxics Coalition, the Washington State Department of Ecology took action in late 2005 to prevent the sale of such lunchboxes in the state.

King County has had tremendous success in reducing mercury pollution from dental offices by

cracking down on dentists to keep mercury out of their wastewater. Mercury in dental offices comes from amalgam fillings, which are about half mercury by weight. State law requires dentists to use devices called separators, which remove mercury from wastewater, but compliance in the past has been poor. By conducting inspections and threatening fines, King County was able to achieve 97% compliance and a 50% reduction in mercury in wastewater between 2000 and 2003 (King County 2005).

The following actions would reduce ongoing exposure to these toxic heavy metals:

- Lead, mercury, and arsenic should be phased out of products.
- Coal burning should be replaced with conservation and cleaner sources of fuel for energy production. In the meantime, existing coal-fired power plants should be required to install the best technology to limit mercury emissions.
- Contaminated sites should be cleaned up promptly and fully. Where a large geographic area is contaminated, state government should take measures to ensure facilities such as schools and day care centers are not sited on contaminated soil.
- Solid-waste and medical-waste incinerators should be shut down and replaced with waste and toxicity reduction, reuse, recycling, and composting programs.
- Health care facilities, including hospitals and dental offices, should phase out mercury-containing products in favor of safer alternatives.
- Government agencies should expand programs to remove, collect, and safely store mercury from thermostats, thermometers, and switches.
- School districts should take remedial action to eliminate lead exposure to children from school drinking water.

Reducing Your Exposure to Heavy Metals

We come into contact with lead, mercury, and arsenic in many aspects of our lives, but there are some steps we can take to reduce our exposure.

Remove treated wood. Remove wood treated with the preservatives CCA or ACZA, which contain arsenic. If removing arsenic-treated wood is not an option, you can paint or seal the wood to reduce leaching and contact exposure. Choose semi-transparent deck stains for deck surfaces and play structures, and latex paint for fences, tables, and other furniture. Reapply the coating when it shows signs of deterioration.

Avoid fish high in mercury. Avoid fish high in mercury, such as king mackerel, tilefish, swordfish, orange roughy, and marlin. Limit consumption of tuna, especially steaks and canned 'white' albacore. Lower-mercury choices include wild salmon, sardines, anchovies, Atlantic herring, Dungeness crab, Pacific cod, Alaskan black cod, farmed striped bass, tilapia, farmed catfish, clams, mussels, and Pacific oysters. If you eat sport-caught fish, check the Department of Health's fish advisories for specific guidance on Washington water bodies or coastal waters. Fish

and shellfish consumption advisories are available at www.doh.wa.gov/ehp/oehas/EHA_fish_adv.htm.

You can find additional guidance on fish choices at the following websites:

Environmental Defense's Oceans Alive: Best and Worst Seafood:
www.oceansalive.org/eat.cfm

Environmental Working Group: Mercury in Seafood (includes Tuna Calculator):
www.ewg.org/issues/mercury/index.php

Fish are an excellent source of nutrients, including protein, omega-3 fatty acids, and vitamin D, and we encourage people to continue eating fish following these precautions. Limiting

mercury intake from fish is especially important for young children and women who are pregnant, nursing, or of child-bearing age.

Watch for lead paint. If you live in a home built before 1978, it is likely to contain lead-based paint. If the paint is chipping, peeling, or otherwise deteriorating, or if you want to remodel, hire a certified abatement worker to remove or contain contaminated paint. Also, use door mats, remove shoes at the door, and vacuum and clean regularly to reduce lead that accumulates in house dust.

Protect drinking water. Flush your cold water pipes (run water until it becomes as cold as it will get) before drinking, and only use cold water for drinking or cooking, to reduce exposure to lead that may be leaching from plumbing.

Avoid PVC. Choose alternatives to products made of PVC, which often contain lead. This is especially important for items that are likely to come into direct contact with children's hands and mouths, such as toys, teethers, and lunchboxes. Also, check to make sure that you don't own children's products that have been recalled due to high levels of lead. Old toys and furniture made prior to 1978 may contain lead-based paint. For consumer product safety information, visit the Consumer Product Safety Commission's website at www.cpsc.gov.

Watch for lead in dishware. Do not use old, imported, or homemade ceramic dishware, unless you know that the glazes do not contain lead. Avoid leaded crystal, as well as imported food cans, which can contain lead solder.

Make sure medicines are free of toxic metals. Some home remedies, as well as drugs and cosmetics, can contain these metals. Look at ingredient lists, talk to your doctor, and avoid folk remedies and other medicines that contain lead, arsenic, or mercury.

Be cautious with mercury-containing products. When possible, choose products without mercury,

such as digital thermostats and thermometers. Be careful not to break fluorescent light bulbs, mercury thermometers, or other household items that contain liquid mercury. These products release harmful mercury vapors when broken. If they do break, use appropriate clean-up methods, found at www.epa.gov/epaoswer/hazwaste/mercury/faq/spills.htm.

Check paints and art supplies. Avoid paints containing mercury compounds, which were used in the past as fungicides and are still found in some paints as pigments. Also avoid lead solder and artists' paints and glazes that contain lead. Information on some products containing these ingredients is available from the Household Products Database: www.householdproducts.nlm.nih.gov. Otherwise, ask the manufacturer.

Skip herbicides with arsenic. Avoid arsenic-containing herbicides, which have ingredients listed as monosodium methanearsonate (MSMA), calcium acid methanearsonate, or cacodylic acid.

Consider composite fillings. Consider choosing composite dental fillings rather than mercury-containing amalgam fillings.

Stain-Protectors Leave an Indelible Mark

"The monkey and the rat test results were scary. The monkeys all died, and with the rats, the pups died."

—Anonymous EPA official (Renner 2003)

Senator Bill Finkbeiner, Washington State Senate.

Senator Bill Finkbeiner is the picture of health. At thirty-seven, he has the energy to maintain a seat in the legislature, run the family property-development business, and play with his two young children. He keeps in shape running, biking, and coaching his daughter's soccer team, and doesn't expect unpleasant news from routine medical tests. But when he donated his hair, urine, and blood for toxic chemical testing, he was a bit apprehensive about what he might learn. It turned out his apprehension was warranted.

Bill topped the list of Pollution in People study participants for levels of perfluorinated compounds (PFCs) and the pesticide carbaryl. He had the second-highest levels of the toxic flame retardants PBDEs, the second-highest level of phthalates, and the third-highest level of mercury. Some of these results are understandable—the carbaryl, for example, probably comes from the conventionally-grown fruits and vegetables he eats. Others are more difficult to explain, particularly his level of PFCs.

Deb Abrahamson, member, Spokane Tribe, and director, Society for Sovereignty, Health, Air, Water, and Land.

At the far eastern edge of the state, Deb Abrahamson's lifestyle doesn't have too much in common with Bill Finkbeiner's. In Wellpinit, Washington, on the Spokane Tribe Reservation, Deb's family eats rainbow trout caught in nearby Turtle Lake, digs camas roots, and gathers huckleberries and serviceberries for dessert. But their study results show Deb and Bill have more in common

than they bargained for: toxic chemicals they knew about, like mercury, and some they never suspected, like PFCs.

PFCs — unique chemicals that are possibly best known for their use in the manufacture of Teflon cookware and Scotchgard — are incredibly resistant to breakdown and are turning up in unexpected places around the world. Though they've been used for more than fifty years in countless familiar products, from fire extinguishing foam to microwave popcorn bags, they've been subjected to little government testing.

PFCs come in many forms, but two have received considerable attention in recent years. PFOS, or perfluorooctane sulfonate, is a member of a family of chemicals once used in treatments for paper food containers, fire-fighting foams, and pesticides, as well as for preventing stains in textiles. Until 2002, the Minnesota company 3M was the major global producer, using PFOS-related chemicals to make Scotchgard, used to treat carpet, furniture, and clothing.

DuPont is currently the major manufacturer of the PFC called PFOA, or perfluorooctanoic acid. The company uses PFOA in the manufacturing of Teflon non-stick cookware. PFOA may also be generated by the breakdown of related chemicals that DuPont uses to make stain-protection treatments for paper products and textiles.

Bill Finkbeiner tested positive for five of the twelve PFCs in our study. PFOS was the highest PFC in each participant. PFOS does not break down under normal environmental circumstances, and builds up in people and wildlife. PFOS levels in our participants ranged from 3.3 to 49.4 ppb, with a median of 21.3 ppb. Bill topped the list for PFOS levels with 49.4 ppb in his blood.

Extensive information on PFC levels in the general population has been lacking, but the CDC recently published data from samples taken in 2001 and 2002, in which blood from 1,832 individuals was pooled into 54 samples for testing. These data revealed that non-Hispanic white males had the highest levels of PFOS, with a mean of 40.2 ppb, while women had somewhat lower levels, with an average of 17.9 ppb for non-Hispanic black women and 24 ppb for non-Hispanic white women. Mexican-American women had the lowest levels, at 10.4 ppb (Calafat 2006). Reasons for the gender and racial/ethnic differences are not known but may be due to varying use of PFC-treated consumer products.

PFOA is also commonly found in people. In the CDC study, levels varied by gender and racial/ethnic background, from 2.1 ppb for Mexican-American women to 7 ppb for non-Hispanic white males.

Previous studies have documented PFCs in specific populations. A 3M-funded study of Red Cross blood donors in Maryland found PFOS at a

median level of 34.7 ppb and PFOA at 5.6 ppb (Olsen 2005). A global look that included individuals from the United States, Colombia, Brazil, Belgium, Italy, Poland, India, Malaysia, and Korea found considerably higher levels in residents from the United States and Poland, with the lowest levels in India (Kannan 2004). The wide variation is likely due to greater use of PFC-treated products in some countries. A 2002 3M study of 599 children revealed that children have unexpectedly high concentrations, with PFOS at a mean of 37.5 ppb but up to 515 ppb in some children (Olsen 2002).

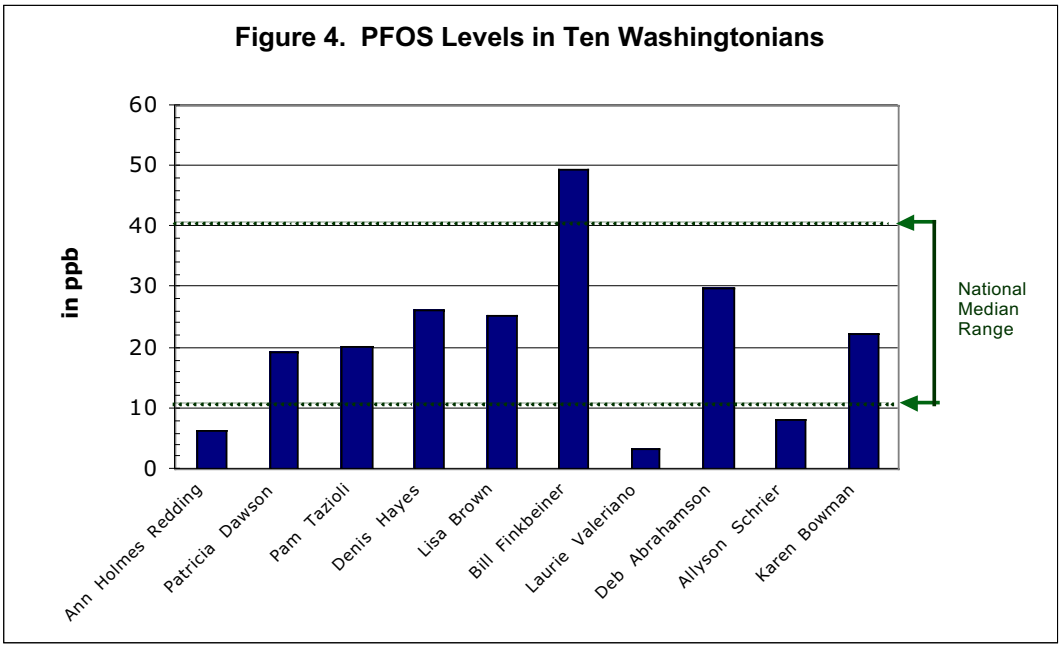

Figure 4: Levels of PFOS measured in participant blood serum.

Bill Finkbeiner's PFOS level (49.4 ppb) is somewhat higher than the national average for white men (40.2 ppb). The women in our study ranged from well below the national average, at 3.3 ppb, to somewhat above, at 29.8 ppb.

PFOA levels in our participants ranged from 0.7 to 7.4 ppb, with a median of 3.6 ppb. While these levels are lower than our participants' levels of PFOS, they may well be on the rise as other PFCs continue to break down into PFOA, which does not degrade.

Although not as well studied, two other PFCs, known as PFDA and PFHxS, are developing a reputation for toxicity. We detected at least one of these chemicals in six of our participants.

Figures 4 and 5 show our participants' levels of the two compounds most commonly found, PFOS and PFOA.

The Post-war Revolution in Stain Protection

PFCs have been in use since the 1950s and have made possible such revolutionary products as stain-resistant furniture and non-stick pans. Even that Northwest emblem, the Gore-Tex jacket, contains PFCs. Chemically, PFCs repel both oil and water. This property has made them irresistible for a wide variety of applications, from stain-resistant couches, leathers, and carpets, for which about 5 millions pounds are used each year (Renner 2001), to grease-resistant food packaging and paper products, for which 2.7 million pounds are used annually.

PFCs are also included in cleaning and personal-care products like shampoo and denture cleaners, and have numerous industrial applications, from semiconductor production to coatings for imaging films and printing plates. But exactly how each of us ends up with these chemicals in our bodies is somewhat of a mystery, though it is likely a combination of direct contact with products that contain PFCs together with exposure from our food, water, air, and house dust.

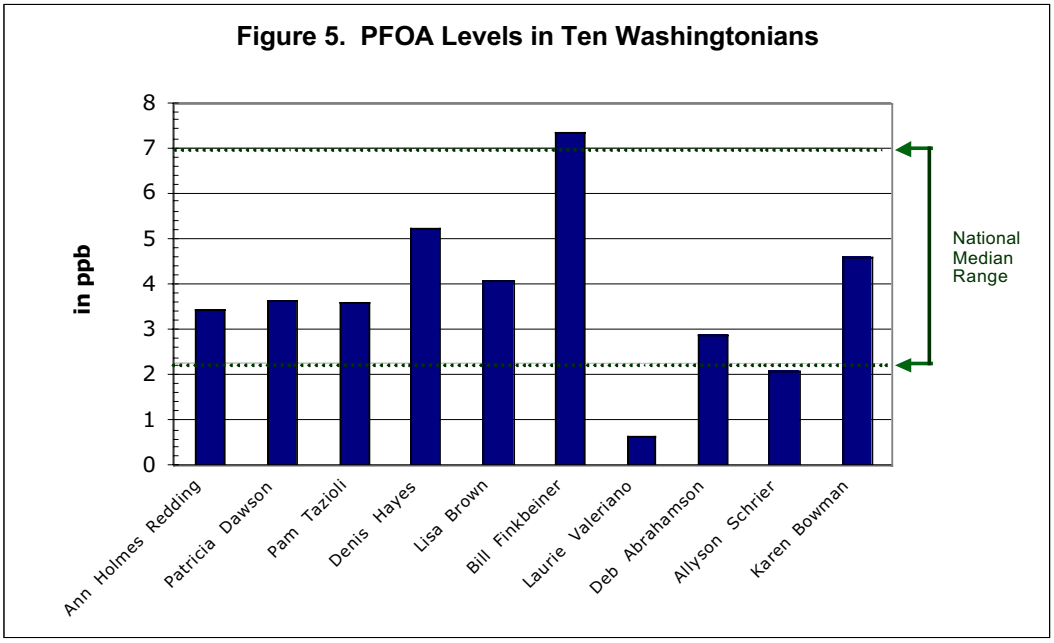

Figure 5: Levels of PFOA measured in participant blood serum.

Perhaps most disturbing about these chemicals is their extreme persistence. Even if production were to end today, levels of the breakdown product PFOA would continue to increase in the environment for many years to come. 3M, which manufactured PFCs prior to 2002, has disclosed that "perfluorinated compounds are extremely resistant to biodegradation" (3M 2000). PFOA is particularly resistant to breakdown processes: it has been found not to degrade at all—even when boiled in nitric acid for an hour (Renner 2001). Once PFOA enters our bodies, it remains in our blood and liver, and it takes years to get rid of it (USEPA 2003). Researchers have estimated PFOA's half-life in our bodies, or the time it would take to expel half of a dose, at more than four years (Kudo 2003). PFOS's half-life has been estimated at more than eight years (OECD 2002).

Researchers have found PFCs in wild animals around the world (Giesy 2001). Predatory animals such as mink, bald eagles, and polar bears displayed the highest levels, indicating that these chemicals increase in concentration as they move up the food chain.

PFOA a Likely Carcinogen

For 50 years, PFCs were used in consumer products without government scrutiny to ensure their safety. But industry-led laboratory studies indicate that our study participants—and the public at large—should be concerned that their PFC levels may be harming their health. In animal tests, male rats with a blood serum PFOA level of approximately 40 ppb had symptoms of kidney and liver damage (USEPA 2002). Female rats with the same level had smaller offspring with reduced growth in later life. Other effects, including increased number of dead offspring and altered size of the liver and pituitary in surviving pups, were seen at higher doses. No one knows exactly what this means for people, but there is considerable cause for concern when, pound for pound, levels in ordinary people like Bill Finkbeiner approach the levels shown to harm laboratory animals.

PFOA also causes liver, pancreatic, testicular, and mammary gland tumors in laboratory animals (USEPA 2002). Studies by 3M to see whether workers exposed to PFOA were more likely to die of cancer have found a possible link to prostate and testicular cancer (Gilliland 1993, Alexander 2001). In 2004, EPA asked an advisory panel of 17 independent scientists to consider the evidence on PFOA's carcinogenicity. In February 2006, the verdict came in: the panel declared PFOA "likely to be carcinogenic."

PFOS has its own problems. As long ago as the 1970s, scientists obtained disturbing results when they exposed monkeys to the chemical. In the first study, no exposed rhesus monkeys survived past three weeks (Goldenthal 1979 as described in OECD 2002). Before they died, the monkeys were weak and lethargic and suffered twitching, trembling, and convulsions. A follow-up study, using lower doses, caused anorexia, diarrhea, convulsions, and harm to the pancreas. Even at the lowest dose, monkeys were listless and had gastrointestinal troubles.

PFOS also causes cancer and reproductive problems in laboratory animals. A two-year study in rats found increases in liver and thyroid cancer (OECD 2002). When pregnant rats were exposed to PFOS, many of the offspring died shortly after birth. When the survivors reproduced, their pups were smaller at birth than the pups of unexposed animals. In rabbits, offspring of exposed mothers had more skeletal abnormalities and lower birth weight.

Policy Changes Needed

PFCs have been produced, used, and disposed of essentially without regulation for the last half-century. Under current federal law, the EPA can require studies on the potential health effects of a chemical only when it already has evidence that the chemical is causing harm. Rigorous evaluation of these chemicals therefore did not start until the late 1990s, following 50 years of use. Rising levels of PFCs in the environment and increasing governmental pressure, however, have led to voluntary actions to reduce PFC production and use. In 2002, 3M ceased using PFCs for its signature product, Scotchgard, because of concerns over release of PFOS and PFOA during manufacture and use. In early 2006, the EPA, Teflon manufacturer DuPont, and seven other companies announced an agreement to reduce PFOA in emissions from manufacturing plants and in consumer products by 95% by the year 2010.

While these actions are a step in the right direction, they do not adequately protect public health from the dangers posed by PFCs. The Washington State Department of Ecology should begin in 2007 by completing a chemical action plan under its program on persistent toxic chemicals to phase out PFOA. The remaining PFCs should undergo expedited review, and, if necessary, be eliminated from products.

Reducing Your Exposure to PFCs

Avoid purchasing or, at a minimum, limit use of products containing PFCs.

Watch for packaged foods. Stay away from greasy or oily packaged and fast foods, as the packages often contain grease-repellent coatings. Examples include microwave popcorn bags, french fry boxes, and pizza boxes.

Avoid stain-resistance treatments. Choose furniture and carpets that aren't marketed as "stain-resistant," and don't apply finishing treatments such as Stainmaster to these or other items. Where possible, choose alternatives to clothing that has been treated for water or stain resistance, such as outerwear and sportswear. Other products that may be treated include shoes, luggage, and camping and sporting equipment.

Check your personal-care products. Avoid personal-care products made with Teflon or containing ingredients that include the words "fluoro" or "perfluoro." PFCs can be found in dental floss and a variety of cosmetics, including nail polish, facial moisturizers, and eye makeup.

Avoid Teflon or non-stick cookware. If you choose to continue using non-stick cookware, be very careful not to let it heat to above 450°F. Do not leave non-stick cookware unattended on the stove, or use non-stick cookware in hot ovens or grills. Discard products if non-stick coatings show signs of deterioration.

Choice is Clear on Pesticides

Over fifteen years as a community organizer and toxic chemicals policy expert, Laurie Valeriano has learned a thing or two about avoiding products that could harm her health. This knowledge, unfortunately, has not fully protected Laurie or her family.

Laurie Valeriano, toxics policy expert, Washington Toxics Coalition.

Over fifteen years as a community organizer and toxic chemicals policy expert, Laurie Valeriano has learned a thing or two about avoiding products that could harm her health. You won't find vinyl shower curtains or toys at her house, and she limits her use of personal-care products that could contain toxic additives such as phthalates. She knows which plastics are relatively safe, and which ones to steer clear of.

This knowledge, unfortunately, has not fully protected Laurie or her family. Laurie's body contains mercury, PFCs, toxic flame retardants, PCBs, and phthalates, albeit at lower levels than in other participants. Moreover, it is likely that she passed significant amounts of these chemicals to her three children in the womb and while breastfeeding.

But what we did *not* find in Laurie's body shows that her choices are—in at least one significant way—having a positive effect on her and her family's health. The Pollution in People study, which tested for metabolites of such commonly used pesticides as malathion, chlorpyrifos, azinphos methyl, and carbaryl, did not find any sign of pesticides in Laurie. Why? Well, for starters, Laurie and her family use alternatives to pesticides in their home and garden, go to a pesticide-free park, and eat organically grown food. Her decisions are backed by good evidence, too: University of Washington research has found that children who eat an organic diet are much less likely to be exposed to pesticides (Lu 2005).

Senator Lisa Brown has made different choices. While she's been a long-time advocate for government action to protect people and wildlife from toxic chemicals, she makes consumer decisions that most any well-educated woman in the United States would: her home includes electronics likely embedded with toxic flame retardants, and she drives a car with an inte-

rior made with phthalates. Most of the food she and her son eat is conventionally grown, not organic. And Lisa, along with five other participants, tested positive for pesticides.

We tested for a series of pesticide breakdown products, or metabolites, that indicate exposure to organophosphate pesticides, as well as the metabolite of the insecticide carbaryl. The carbaryl metabolite was most commonly found, turning up in five participants.[5] We also found the organophosphate metabolites known as DMTP (in four participants) and DMP (in two), indicating exposure to the widely used insecticides azinphos methyl and malathion, among other pesticides.[6] These insecticides are very commonly used in agriculture, and malathion is also found in some home-use products. One participant had the metabolite DEP, suggesting she had been exposed to the organophosphates diazinon or chlorpyrifos, or other less commonly used pesticides.

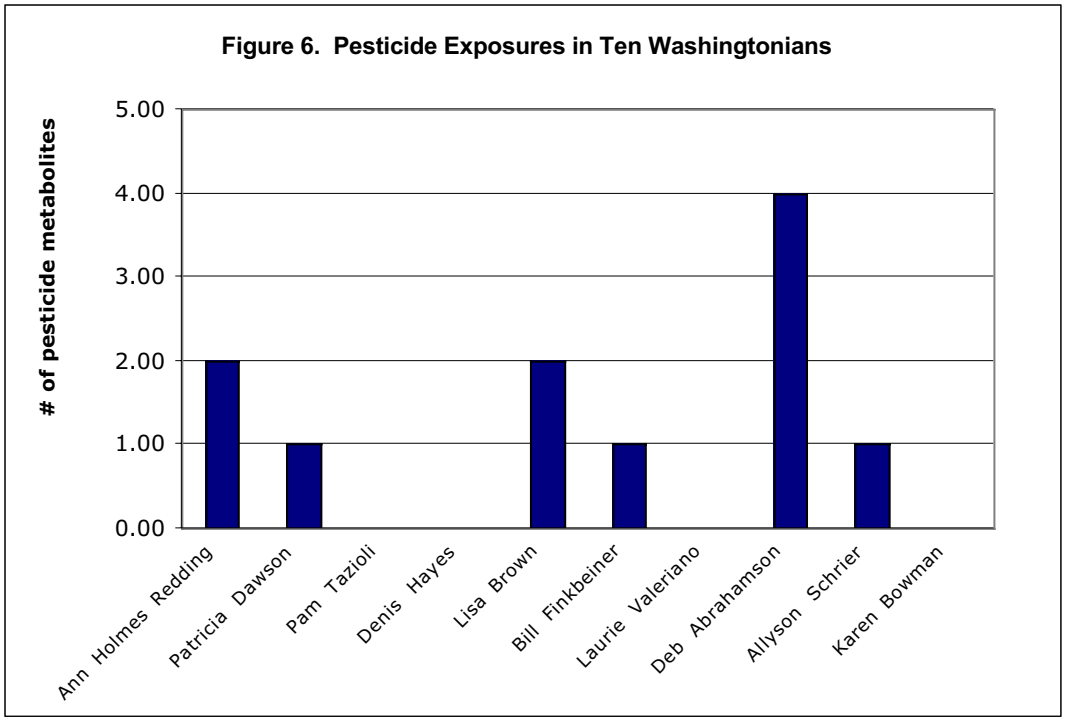

Figure 6: The number of organophosphate and carbaryl pesticide metabolites detected in participant urine.

Senator Lisa Brown, Washington State Senate Majority Leader.

We also tested for a metabolite specific to chlorpyrifos exposure but did not find it in any study participants, possibly reflecting the phase-out of this pesticide's residential uses, which began in 2000.[7] However, pesticide levels in two

[5] The presence of 1-napthol (the carbaryl metabolite tested) in urine may also be the result of exposure to naphthalene, tobacco smoke, or fires. None of our study participants is a smoker or lives with a smoker.

[6] DMTP, DMP, and DEP are "non-specific" metabolites of organophosphate pesticides, meaning they may result from exposure to more than one pesticide.

[7] We also tested for the herbicide 2,4-D, but the test was not sensitive enough to detect levels commonly found in the population at large.

of our participants, Deb Abrahamson and Ann Holmes Redding, suggest that the two are more highly exposed to organophosphate pesticides than 90% of people nationally when compared against the results of a 2005 CDC study (CDC 2005). Deb had levels of DMP, DMTP, and DEP that put her in the top 10% nationally; Ann's levels of DMP and DMTP were also in the top 10%. Organophosphates do not persist in the body, so these levels reflect recent exposures. Many of us are exposed to these pesticides regularly over our lifetimes, which often leads to consistently detectable levels.

Figure 6 (previous page) shows participants' exposures to organophosphates and carbaryl. The chart shows the number of metabolites of these pesticides detected, out of a total of seven tested.

Along for the Ride

Most of our ten participants don't spray these insecticides in their home or garden, so the most likely source of the pesticides in their bodies is their food, especially since most of their diets are not organic. The organophosphate pesticides diazinon and chlorpyrifos, once the most widely used insecticides in U.S. homes and gardens, are now primarily used in agriculture (with a few exceptions, they are not used in urban areas). Azinphos methyl is highly acutely toxic and is used only in agriculture, on such crops as apples, cherries, and pears (USEPA 1999). Malathion is used in both settings, but most commonly in agriculture. Carbaryl is sprayed on about half of Washington's apple crop and much of its grape acreage (WSDA 2003).

The U.S. Department of Agriculture (USDA) conducts a yearly "market basket" survey to test for residues of pesticides in produce. The most-contaminated fruits and vegetables include apples, bell peppers, celery, cherries, grapes, nectarines, peaches, pears, potatoes, raspberries, spinach, and strawberries. Purchasing some foods in particular comes with a near-guarantee of pesticides along for the ride. USDA's most recent testing found that 98% of apples and 97% of bell peppers are contaminated (USDA 2006). All of the apples and pears tested by the USDA were contaminated with 1-napthol, the breakdown product of carbaryl. The agency found up to nine pesticides on a single apple and eight in a single grape sample.

We found evidence in our study that suggests eating organic provides some protection from pesticide exposure. We tested for a correlation between pesticide detections in our participants and the number of organic meals each person reported eating. We found a statistically significant correlation, indicating that the more organic food each participant ate, the less likely he or she was to have pesticides in his or her body.[7]

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[7] *Spearman's correlation coefficient was calculated as -0.69, and it was significantly different from zero at p=0.028, indicating a possible negative correlation of the number of organophosphate and/or carbaryl metabolite detections with the number of organic meals per week.*

Farm workers and farm worker families are exposed to the most agricultural pesticides by far. While mixing and applying pesticides, they come into contact with significant amounts of chemicals, later tracking them into their homes on their shoes and clothing. Their homes, which are often near the farms they work on, become further contaminated when pesticides drift from nearby fields.

From Convulsions to Cancer

It's no secret that these pesticides can harm the nervous system. Organophosphate pesticides block an enzyme, acetylcholinesterase, that is critical for proper transmission of signals from one nerve to the next. Shortly after being exposed to a high dose of organophosphates, a person will suffer weakness, cramps, breathing trouble, nausea, and vomiting (USEPA 1999). Worse, some effects may persist long after exposure. In one study, farm and pest-control workers tested months to years after a pesticide-poisoning incident had poorer memory and damaged motor skills, as well as anxiety, depression, and confusion (Eskenazi 1999). Carbaryl has similar immediate effects on the nervous system. Inhaling or ingesting large amounts can cause nausea, stomach cramps, and diarrhea, as well as sweating, blurred vision, loss of coordination, and convulsions (USEPA 2003).

Increasing evidence from animal studies also indicates that the brain development of fetuses and children may be impaired by exposure to organophosphates. Studies in young mice found that a single dose of an organophosphate caused permanent damage to brain function, including hyperactivity (Ahlbom 1995). Similarly, mice with prenatal exposure to diazinon had poorer coordination and endurance, as well as delayed sexual development (Schettler 2000). Rats with prenatal exposure to chlorpyrifos had smaller brain weights and decreased reflexes (Chanda 1996, Schettler 2000).

Recent studies in farmworker populations indicate that developmental damage from these pesticides is occurring in people. University of California researchers have studied a group of children born to farmworkers in California's Salinas Valley, where 500,000 pounds of organophosphate pesticides are used each year. In a 2005 study, the group reported that infants with greater exposure to organophosphates had more abnormal reflexes (Young 2005). They also found that mothers with higher exposures are at increased risk for preterm birth (Eskenazi 2004). In Oregon, researchers compared neurological performance of farmworkers with that of an immigrant community living on the coast with little exposure to pesticides (Rothlein 2006). Their findings: adults with greater exposures to organophosphates scored more poorly in tests of attention

spans and motor function.

There's also powerful evidence that carbaryl may cause cancer; EPA considers carbaryl a likely human carcinogen (USEPA 2003). Several studies have reported greater incidence of childhood brain cancer in homes using carbaryl (Davis 1993) or types of products that may contain carbaryl (Pagoda 1997). Other studies have found an elevated risk of non-Hodgkins lymphoma (NHL) among farmers who handled carbamate insecticides in general and carbaryl in particular. A study of Canadian farmers showed that those using carbaryl had twice the incidence of NHL (McDuffie 2001). A reanalysis of pooled data from three separate studies in several Midwestern states found a 60% increase in NHL among farmers who reported using carbaryl (Zheng 2001). While these studies don't prove an association, their sample sizes were large and their findings correlate with evidence from animal studies linking carbaryl to immune suppression, a known risk factor for NHL.

Policy Changes Needed

The presence of these pesticides in our participants' bodies demonstrates the extreme flaws in today's system for regulating pesticides. People cannot fulfill the most basic of needs—nutrition—without risking harm from pesticides that can damage brain function and cause cancer. Although the EPA requires manufacturers to test pesticides for harmful effects, national rules do not prevent continued use of pesticides that test positive for cancer or harm to brain development.

The federal pesticide law, the Federal Insecticide, Fungicide and Rodenticide Act, makes no guarantee that pesticides allowed for use will not cause harm to people and other living things. Rather, the law protects a pesticide's uses unless the chemical poses "unreasonable risk to man or the environment, taking into account the economic, social, and environmental costs and benefits of the use of any pesticide." That is, as long as a pesticide's perceived economic benefits outweigh its health risks, the law allows for its use.

The Food Quality Protection Act of 1996 aimed to improve this standard somewhat for pesticides used on food, but EPA has yet to implement many of the law's important provisions. For example, the law requires EPA to consider the cumulative effects of different pesticides that have the same health effect. A decade after its passage, EPA is still in the process of determining how to implement this requirement.

Under the same act, EPA has developed agreements with the pesticide industry to nearly eliminate home use of two important organophosphates, chlorpyrifos and diazinon. As noted above and as demonstrated in our study, however, the widespread agricultural use of organophosphates, including of these two pesticides, means that U.S. residents continue to be regularly exposed to the chemicals. The EPA has also placed some restrictions on carbaryl, but its use continues in the home and garden setting and in agriculture. And EPA continues to allow other carbamate pesticides, chemical relatives of carbaryl, which have similar effects on the nervous system.

To eliminate exposure to these pesticides, EPA should phase out the use of all organophosphate and carbamate pesticides. The Washington State Department of Agriculture can also take action to phase out these and other toxic pesticides. At the state level, the legislature should

continue to provide funding to Washington State University's Center for Sustaining Agriculture and Natural Resources to develop alternatives for both conventional and organic growers. Cities and counties can eliminate their own use of toxic pesticides on public property, and educate residents about replacing pesticides with healthier practices in the home and garden. Laurie Valeriano's family has a pesticide-free park to use because of a community effort to make it one of Seattle's 22 pesticide-free parks. This effort, together with a campaign led by local residents and organizations, ultimately led the City of Seattle to establish a precedent-setting policy ending its use of the most toxic pesticides, which other cities and counties can emulate.

Reducing Your Exposure to Pesticides

Although some exposure to pesticides is difficult to avoid, you can significantly reduce pesticides in your diet and your surroundings with a few simple steps.

Buy organic. Organically-grown food is produced without the use of toxic pesticides. It's especially important to buy organically grown apples, bell peppers, celery, cherries, grapes, nectarines, peaches, pears, potatoes, raspberries, spinach, and strawberries. Ask your grocer to start carrying organic food if it doesn't already.

Use alternatives to pesticides in your home and garden. There are many non-chemical methods of pest control that are safe and effective, such as using traps and barriers and removing pests physically. Focus on preventive techniques, which are most effective in the long run. For example, plug holes to keep insects from coming in the house, and grow healthy grass so that weeds don't thrive in the lawn. Extensive resources for preventing and addressing pest problems are available at www.watoxics.org.

Advocate for pesticide reduction in your school and park. Many school districts, cities, and counties have policies to replace toxic pesticides with safer practices. The Washington Toxics Coalition can help you secure such a policy in your district, city, or county.

The Chemicals That Came to Stay

The histories of DDT and PCBs are both success stories and cautionary tales. Since these chemicals were banned 30 years ago, levels in our bodies have declined. And yet, we still face levels that could be causing harm—decades after regulatory action.

Rev. Dr. Ann Holmes Redding, Episcopal priest, St. Mark's Cathedral.

Some chemicals just won't go away.

Rachel Carson taught us that lesson more than 40 years ago, when she issued a wake-up call in her book *Silent Spring*. Pesticides like DDT, then in common use for everything from mosquito control to orchard spraying, were not breaking down into harmless chemicals after their use. Instead, as Carson pointed out, the chemicals were building up in soil and sediment, fish and wildlife, threatening to destroy the very fabric of life.

For the Pollution in People study, we tested for DDT and PCBs, two chemicals that were long ago banned but continue to haunt us. These chemicals persist in the environment and our bodies. Women pass them on to their children in the womb and through breastmilk. And as each of us ages, our load of these chemicals increases.

Rev. Ann Holmes Redding sees this chemical contamination as a corruption of the sanctity of life. As an Episcopal priest and New Testament scholar, Ann believes our bodies are a gift from God and that we have a responsibility to care for them. But for nearly her entire life, Ann's body has been home to unwelcome chemical imposters like DDT and PCBs. Her Pollution in People study results showed that she has 8.7 ppb DDT, a level high enough to put her in the top 25% of people nationwide. She also carries 1.5 ppb PCBs, again at the high end of national exposures.

DDT

DDT was first developed as an insecticide in the 1940s, and it was widely used during World War II to combat insect-borne diseases, such as malaria and typhus. After the war, DDT's effectiveness, persistence, and low cost made it popular for

agricultural and commercial uses. In 1959, at the height of its popularity, 80 million pounds of the chemical were applied to forests, fields, and gardens (USEPA 1972). Over DDT's 30-year history in the United States, more than a billion pounds were used (USEPA 1975).

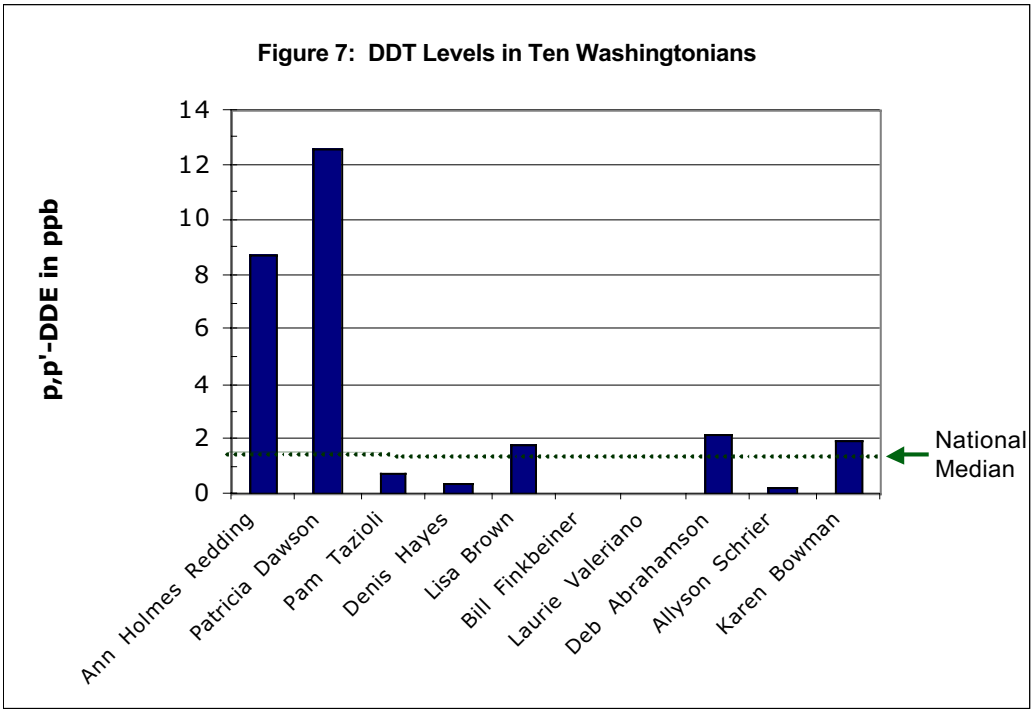


Figure 7: DDT exposure was measured as the breakdown product p,p'-DDE in blood serum.

EPA banned nearly all domestic uses of DDT in 1972. Today, its use is limited to malaria control programs in some developing countries. But most of us are exposed to DDT every day because it is in so much of the food we eat (USDA 2006). Vegetables, meat, fish, and dairy products all contain DDT, but animal and fatty foods contain the highest levels because the chemical is stored in fat and increases in concentration as it moves up the food chain (ATSDR 2002). Children, breastfeeding infants, and people living in the eastern Arctic have the greatest ongoing exposures to DDT from food.

Exposure to DDT is harmful to the nervous system, with high levels causing dizziness, tremor, irritability, and convulsions (ATSDR 2002). Animal studies have found that low levels can affect nervous system development. In addition, people who applied DDT in occupational settings have suffered lasting neurological problems, performing tasks more slowly and displaying delayed reaction times, less dexterity and strength, and reduced cognitive function (van Wendel de Joode 2001).

DDT is also considered a hormone disrupting chemical because of its estrogen-like properties,

and researchers have found disturbing effects in this regard. Mothers with greater exposure to DDT are more likely to have premature or small-for-gestational-age babies than mothers less exposed to the chemical (Longnecker 2001). Mothers with more DDT also breastfeed for a shorter period, possibly because DDT mimics hormones that inhibit milk production (ATSDR 2002). Animal studies have found that DDT causes cancer, and EPA ranks DDT as a probable human carcinogen.

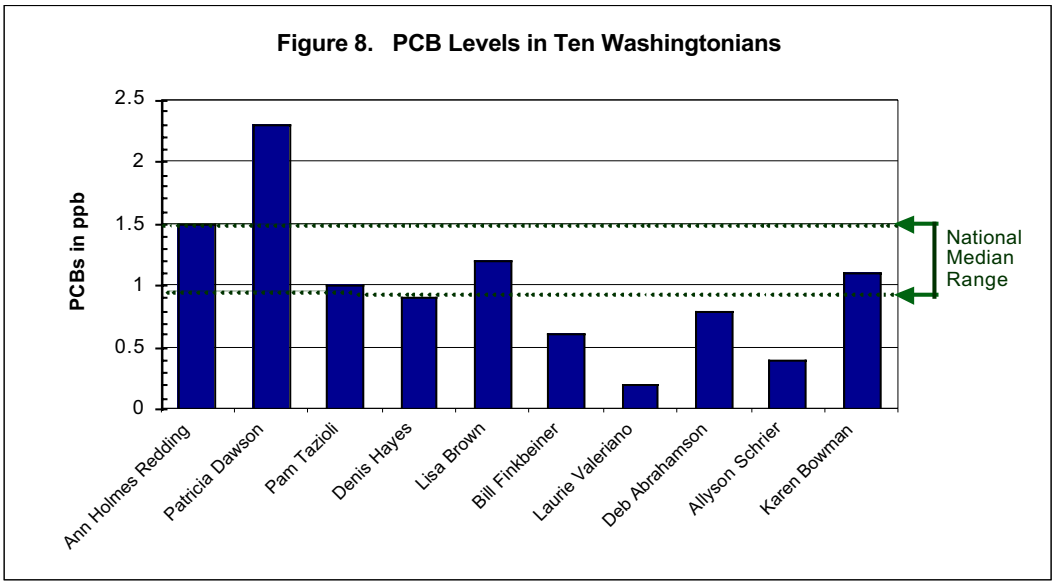


Figure 8: PCBs were measured in participant blood serum.

PCBs

PCBs had a more obscure purpose, but the chemicals made a name for themselves nonetheless. Between 1929 and the mid-1980s, PCBs were popular as cooling fluids in electrical equipment and machinery because of their reputation for durability and fire resistance (USEPA 1979).

Concerns about PCBs' health effects and persistence surfaced in the 1970s, and Monsanto, the major U.S. manufacturer of the chemicals, stopped producing them in 1977. The EPA phased out most uses of PCBs shortly thereafter. Levels of the chemical in people and wildlife have since declined, but three decades later we continue to ingest PCBs when we eat fish, meat, or dairy products (ATSDR 2002).

Because PCBs accumulate in sediment in rivers, lakes, and coastal areas, fish contain particularly high levels of the chemicals. Levels in fish can be 2,000 to more than a million times higher than levels in surrounding waters (USEPA 1999). Because of PCB contamination, the Washington State Department of Health recommends limiting consumption of fish and shellfish from many of the state's water bodies (WDOH 2006).

Women who consume PCBs in their diet readily pass them to their children in breast milk: infants may get 6 to 12% of their lifetime exposure to PCBs from breastfeeding alone (ATSDR 2002). At levels typically found in women and children around the world, PCBs can have profound effects on intellectual development.

In studies of large numbers of children in the U.S., Germany, and the Netherlands, those with

greater prenatal exposures (measured by levels in umbilical cord blood or the mother's blood) performed worse on tests of brain development than children with lower exposures (Shantz 2003). The same body of research also revealed lower birth weights and slowed growth in children with higher PCB levels. In each of these studies, the mothers of the most-exposed children obtained PCBs from fish or other common sources.

Researchers who followed children in Michigan from birth to age 11 found that these effects persist (Jacobson 2002). They compared children from sport-fishing families, whose mothers ate above-average amounts of Lake Michigan fish, with children whose mothers ate no Lake Michigan fish. The sport-fishers' children, who had greater prenatal exposures, showed intellectual deficits as infants, at age 4, and again at age 11, when they displayed attention deficits, lower IQs, and poorer reading comprehension. While the mothers of the most-exposed children in this study had PCB levels several times those of our participants, other studies have found similar effects at lower levels (Shantz 2003).

In addition to cognitive damage, PCBs cause tumors in laboratory animals (Ross 2004) and have been classified by the EPA as probable human carcinogens. Studies suggest the chemicals are also toxic to the immune system, reproductive organs, and thyroid.

PCBs are a major contaminant in Puget Sound, and evidence is accumulating that they are a serious threat to the Sound's wildlife, too. Puget Sound's endangered orca whales have accumulated PCBs to the point that they rank among the most contaminated marine mammals in the world (Ross 2006). Levels in orcas already

exceed those needed to cause health effects such as immune system depression.

Policy Changes Needed

The histories of DDT and PCBs are both success stories and cautionary tales. Since these chemicals were banned 30 years ago, levels in our bodies have declined. And yet, we still face levels that could be causing harm—decades after regulatory action.

To this day, runoff from agricultural lands transports DDT-containing sediment to rivers and streams, where it is taken up by fish. PCBs persisting in river and bay sediment cause astonishingly high levels in orca whales and salmon. As a result, both chemicals persist in our diets. With both DDT and PCBs, the EPA allowed production and use to go on far too long, to the point where our air, water, land, and bodies became so contaminated that decades of cleanup efforts have yet to eliminate their threats to our health. And the incredible sums expended by state, federal,

and tribal governments are all too often resources that could have been put to other uses had these chemicals been adequately tested and analyzed before their widespread production.

Governor Christine Gregoire has launched a major initiative to restore and protect Puget

Sound, with the goal of solving the Sound's biggest pollution problems by 2020. Because cleaning up contaminated sediment is incredibly expensive, the 2006 Washington State Legislature appropriated $44 million for just a single year of Puget Sound cleanup and restoration activities. This initiative is a bold step toward addressing the problem of historical pollution in Puget Sound. However, to fully restore the health of Puget Sound and other toxic sites, state government must place equal or greater priority on preventing the Sound's recontamination with these banned chemicals—as well as other persistent toxic chemicals, like PBDEs and perfluorinated compounds.

Reducing Your Exposure to DDT and PCBs

Unless you live near an industrial or agricultural site contaminated with PCBs or DDT, your greatest source of exposure to these chemicals is likely to be food. While you cannot completely avoid these chemicals in your diet, you can make some choices that will help reduce your exposure to them.

The most important actions you can take to reduce the PCBs and DDT in your diet are to cut back on animal fats and watch the type of fish you eat.

Choose fish wisely. Check with state advisories before eating sport-caught fish or shellfish, which are often high in PCBs and DDT. Commercial fish that are high in PCBs include Atlantic or farmed salmon, bluefish, wild striped bass, white and Atlantic croaker, blackback or winter flounder, summer flounder, and blue crab. Commercial fish that contain higher levels of pesticides, including DDT, are bluefish, wild striped bass, American eel, and Atlantic salmon.

When preparing fish, remove the skin, trim the fat, and broil, bake, or grill the fish so that

the fat drips away; this will reduce your exposure to PCBs and other toxic chemicals that have accumulated in fatty tissue. Fish are an excellent source of nutrients including protein, omega-3 fatty acids, and vitamin D, so don't remove fish from your diet—but do be selective about the fish you eat.

Make your meat lean. When it comes to meat, choose lean meat cuts, and buy organic meats if possible. Cut off visible fat before cooking meat and choose lower-fat cooking methods: broiling, grilling, roasting or pressure-cooking. Avoid frying meat in lard, bacon grease, or butter.

Limit dairy fat. Opt for low-fat, organic options when it comes to dairy products, too.

For more information on pollutants in fish, meats, and dairy, see:

Environmental Defense's Oceans Alive: Best and Worst Seafood
www.oceansalive.org/eat.cfm

Washington State Fish and Shellfish Consumption Advisories:
www.doh.wa.gov/ehp/oehas/EHA_fish_adv.htm

IATP Smart Meat and Dairy Guide for Parents and Children
www.iatp.org/foodandhealth

The Laws that Fail Us, and a Better Way

Washington should adopt common-sense regulations that ensure that industries use only the safest chemicals and materials to manufacture products.

There is no denying that toxic chemicals have set up shop in our bodies and homes—without our permission. Teflon chemicals that can cause cancer. Pesticides that harm the nervous system. Phthalates that threaten fertility. Flame retardants that accumulate in our tissues. And persistent chemicals, banned for 30 years, that still contaminate some of our otherwise most nutritious foods.

Companies like DuPont, 3M, and Monsanto undoubtedly chose these chemicals for their effectiveness and durability. They discovered that they could use fluorine, for example, to create products that make red wine run off of a couch instead of staining it, or let pancakes slide from the pan instead of sticking to it. Such products and others—plastic toys for infants, insect spray for our yards, and the fire-resistant PCBs that once cooled machines—seem to work great. And the average consumer is justified in believing that government agencies have tested and approved all of these products, and that their use is safe.

These companies did not, however, create products that serve a purpose without leaving a toxic legacy in their wake.

Neither manufacturers nor government agencies took the time to ensure that these products are safe for our bodies and the environment. Government agencies rarely require toxicity testing, and even when they do, they do not reject chemicals linked to serious health problems. In the end, we end up saddled with toxic problems, like fish we can't eat and flame retardants in our breastmilk and blood.

Don't Ask, Don't Tell

In this laissez-faire system, government action to protect the public is extremely rare, even when things go awry. The EPA

intervened to ban DDT and PCBs in the 1970s, but since then has stopped only five chemicals under the weak law that governs toxic chemicals in the United States (Wilson 2006). Under this law, the Toxic Substances Control Act (TSCA), EPA considers the vast majority of chemicals safe until they are proven dangerous. New chemicals do not go through a regular screening process to determine health threats, and the law does not give EPA authority to require toxicity testing to produce the information it would need to assess health threats.

In many ways, little progress has been made since Congress passed TSCA in 1976. Of the 81,600 chemicals used in industry today, 62,000 were already in production in 1979 when the law was implemented. Congress included measures in TSCA to protect chemicals already in use when the law was implemented. These exempt chemicals make up 92% of today's high-use chemicals, those produced or imported at more than one million pounds each year (Wilson 2006). EPA faces substantial legal hurdles if it attempts to restrict any of these 62,000 chemicals.

The situation is only slightly better for chemicals introduced after 1979. Manufacturers don't need permission from EPA to produce a new chemical, but they are required to notify the agency and provide any available information on harmful effects. If EPA finds that a chemical presents an unreasonable risk to health, the agency can take action to restrict the chemical before it goes into production. EPA placed restrictions on 3,500 chemicals between 1979 and 2004, a figure that represents fewer than 10% of the chemicals that came to market during that time.

Once a chemical is in production, EPA may only restrict its use when a number of conditions are met. First, the agency must demonstrate the chemical's negative effects on human health and the environment. Second, it must assess the chemical's benefits and demonstrate that substitutes for its uses are available. Third, EPA's action must constitute the least burdensome requirement that will be sufficiently protective. Finally, EPA may restrict a chemical only if no other law can adequately address the hazard. Under this system, top protections don't go to people—they go to the chemical.

EPA may require health-effects testing only when it has already demonstrated that a chemical poses an unreasonable risk to health or the environment, or that it will be produced in quantities likely to result in substantial human or environmental exposure (GAO 2005). EPA has used this authority for only 200 of the 81,600 chemicals currently registered for use (GAO 2005, Wilson 2006). Even when it has information on a chemical's potential health effects, EPA cannot share it publicly or with state government agencies because the law allows companies to claim such data as confidential business information. This secrecy creates stumbling blocks for state agencies seeking to safeguard health and for manufacturers making decisions on which chemicals to use.

The federal law governing pesticides takes a different approach, but in many ways arrives at the same destination. EPA has historically regulated pesticides under a risk/benefit standard, under which the agency must allow a pesticide for use if its economic benefits outweigh its health risks. In 1996, Congress passed the Food Quality Protection Act, which established different re-

quirements for pesticides used in food production. Under this system, pesticide manufacturers submit voluminous reports on pesticide toxicity, generally including tests on the chemical's acute toxicity, likelihood to cause cancer, and ecological effects, which EPA then uses to assess the level of risk to health posed by the chemical's use.

Although this process often places restrictions on pesticides, it has not eliminated the use of many pesticides linked to serious health problems, such as carbaryl. Despite the fact that carbaryl is known to harm the nervous system and is considered likely to cause cancer, EPA's recent assessment allowed the pesticide to remain on the shelves. Some of its uses, such as flea treatment, have been banned, but the pesticide is still sold in 10-pound bags to be sold and spread on lawns, and it's used extensively in agriculture. The result is that we continue to be exposed to this dangerous pesticide, despite its subjection to a process intended to weed out harmful chemicals.

An Uphill Battle for States

With federal regulations failing, Washington state agencies face significant challenges in their efforts to protect health and the environment from toxic chemicals. When they have attempted to reduce or eliminate exposures to a specific chemical, such as the toxic flame retardants PBDEs, they've found that they have neither the information nor the power they need to take prompt action. When these agencies have been successful in restricting a chemical, they've found that they lack the resources necessary to help businesses adopt safer practices and choose safer materials.

Insufficient Information

In 1998, Washington state adopted a groundbreaking policy to eliminate persistent toxic chemicals. To carry out this policy, the state Departments of Health and Ecology have developed "chemical action plans," which evaluate the exposures, uses, and toxic effects of a chemical, and its potential alternatives. This process has brought important information gaps to light. While attempting to determine the availability of safer alternatives to deca-PBDE, for example, the departments had no access to basic data on persistence, bioaccumulation, and toxicity of alternative chemicals. The state resorted to computer modeling to predict the alternative chemicals' behavior, but this solution was far from perfect.

State agencies have been further frustrated by the lack of information about which companies use what chemicals, and which chemicals end up in a company's final products. This information gap is highlighted when agencies attempt to control specific chemicals, such as PBDEs or lead, and find that they must either conduct their own testing or beg companies for information. Without access to this information, agencies are limited in their ability to assist industry in reducing pollution or switching to safer materials.

Unwieldy Regulatory Framework

While Washington state has a policy and rule to phase out persistent toxic chemicals, the state regulatory agencies, with limited exception, lack the regulatory framework to act quickly to ban the sale of products containing these chemicals or others that threaten human health. State agencies derive most of their authority from federal laws that focus not on

preventing pollution but on *regulating* it by limiting discharges into water and controlling emissions from smokestacks.

Because they are not set up to eliminate dangerous chemicals in products, state agencies have been forced to seek assistance from the state legislature to ban specific chemicals, such as PBDEs. The Departments of Ecology and Health, through Ecology's program on persistent toxic chemicals, have sought restrictions on mercury and PBDEs. The program's record so far: despite an eight-year history, it has won legislative approval to restrict only one chemical, mercury. This chemical-by-chemical approach is clearly not the road to timely solutions for toxic problems.

The Washington State Department of Agriculture does have clear authority to ban pesticides that harm health or the environment, and it has used this authority in the past to address particularly hazardous situations. What it lacks, however, is a framework to assess the potential hazards of pesticides and eliminate those that are likely to cause harm. The agency requires registration of pesticides used in the state but does not conduct its own assessment of toxicity unless there is already evidence of severe harm.

Taken as a whole, Washington's agencies have no clear path forward for assessing what chemicals are too dangerous to appear in products. The Department of Ecology's persistent toxic chemicals program and the Department of Agriculture's pesticide registration program are a far cry from the regulatory framework needed to keep toxic chemicals out of products.

Lack of Resources and Incentives

Washington state also lacks the technical resources and incentives needed to assist local companies in developing safer processes and products. While the Department of Ecology has a limited technical assistance team, its focus has been on reducing energy and water use rather than on toxic chemicals. To effectively assist businesses in reducing pollution, the state needs a comprehensive program to help local companies develop safer products and reduce toxic chemical use. An effective program would require companies to develop and implement plans to reduce pollution; provide incentives, such as tax cuts, for companies that reduce their use of toxic chemicals; and institute disincentives for pollution, such as fees based on chemical use and releases.

The Path Forward

Industry leaders are demonstrating that change is possible, by responding to an evolving global marketplace that is demanding safer products. In many cases, European regulations have driven companies to reformulate their products and adopt new standards. In other cases, companies see a market advantage in meeting consumer demand for less-toxic, conscientiously produced products. These stories are becoming more and more common as companies of all sizes recognize that the future lies in developing products that will last both because they are effective and because they use the safest materials.

- Health care provider Kaiser Permanente's chemical use policy helps the company reduce its reliance on chemicals that build up in our bodies or are linked to cancer or reproductive problems. Kaiser has also embraced organic food and has farmers

markets offering organic food at 25 of its medical facilities (HCWH 2005).

- Furniture maker Herman Miller has pledged to generate zero hazardous waste and zero air and water emissions by 2020.

The company's innovative design processes now include assessments of the hazards of potential materials. The company's Mirra chair, for example, replaces PVC and PBDEs with safer alternatives.

- Dell, the largest computer manufacturer in the world, acted to phase out all PBDEs when the European Union adopted a phaseout of most PBDEs by 2006. Dell has also gone farther, developing a list of chemicals it has chosen to reduce or eliminate from its products, including lead, PVC, PBDEs, and mercury.

- Cascadian Farm, which began as a small farm in the Skagit Valley in the 1970s, has grown to become a leading grower and processor of organic foods with products sold nationwide. General Mills obtained the company in 2000, recognizing the growth potential for this market.

- Cosmetics company The Body Shop has pledged to eliminate phthalates from its products. The company joins a group of 300 that have agreed to take this step in response to a request from the Campaign for Safe Cosmetics.

Change Coming From Europe

Having recognized the flaws in its previous regulatory system, which was very similar to that of the United States, the European Union is now undertaking a top-to-bottom change in the way it regulates chemicals. Its REACH (Registration, Evaluation, and Authorization of Chemicals) initiative, which takes effect in 2007, will close the information gap by requiring companies to register and supply basic information on up to 30,000 chemicals already in use. Companies will be required to develop and submit more extensive information on 5,000 higher-use chemicals so that regulatory agencies can evaluate them. The legislation also bans chemicals that cause cancer, mutations, or reproductive harm, or that are persistent and bioaccumulative, unless these risks are adequately controlled.

Washington State Can Lead the Way

In some cases, innovative businesses and industry leaders, as well as state and local government agencies, have already joined with scientists and public health experts to phase out some

dangerous chemicals. But to effectively manage the more than 80,000 chemical substances currently in use, we need a new approach. We simply cannot wait for people to get sick and then go after problem chemicals one at a time. As Washington state's population grows and industry expands, we need a policy framework that will marry economic growth with solutions to health and environmental problems.

Washington state has proven itself as a leader when it comes to addressing the serious threat of toxic chemicals. In 1998, the Department of Ecology established the first program in the nation to phase out persistent toxic chemicals. Since that time, Ecology has developed and implemented plans to phase out mercury and PBDEs, and has supported legislation to address these threats.

But the department and others have seen the limitations of a chemical-by-chemical approach that takes action only when threats have been demonstrated. Like the current federal regulatory system, such an approach is destined to lag behind harm to people and wildlife. Washington should adopt common-sense regulations that ensure that industries use only the safest chemicals and materials to manufacture products.

Washington state can be a leader in protecting health with the following reforms:

1. Ensure that only the safest chemicals and materials are used to create consumer goods, grow food, or support industries.

Companies all over the world are using innovative practices to develop consumer goods that are safe and effective. Major computer manufacturers, such as Apple and Dell, have replaced the toxic flame retardants PBDEs with safer substitutes. IKEA redesigned its foam mattresses so that they no longer need any chemical flame retardant. More and more food companies, such as Campbell's Soup Company, are producing organic versions of their products. Right now, these success stories are the exception. For Washington to have a vibrant economy *and* a high quality of life, they must become the rule.

Washington needs new chemicals policy that rewards innovative companies and moves the rest toward safer materials and practices. State government should show leadership by developing policies and programs that will get toxic chemicals out of our bodies and secure us a healthy place to live.

2. Require that companies provide full information on the chemicals they use in products and in manufacturing.

We won't achieve safer products and practices as long as we don't ask questions or get answers about whether chemicals can harm our

health and build up in our bodies. Chemicals should be tested for safety before they are ever allowed for use—just like pharmaceuticals are. And companies must make testing results available to government agencies and the public.

Washington should require companies to provide data on the health effects caused by the chemicals they produce or use in production, and make this information available to the public. Washington should start by requiring companies to submit existing health effects information as well as reports on what chemicals they use in their manufacturing processes and products. The state should also conduct a thorough analysis to consider requiring additional tests to develop solid information about the toxicity of chemicals used in Washington and sold in our products.

3. Prevent chemicals that build up in our bodies, cause cancer, or harm our fertility from being used in commerce or released from factories in our communities.

The surprising truth is that chemicals that can damage children's intellectual development, harm reproduction, cause cancer, or build up in our bodies are commonly used in everyday products like cologne and non-stick pans. Products must be free of chemicals that build up in our bodies or are linked to serious health problems.

Washington state should develop immediate plans to phase these chemicals out of products and manufacturing. The plans must take on the most toxic chemicals in a comprehensive way, rather than one at a time. Safer materials are available, and governments can drive the marketplace toward safer substitutes by restricting these hazardous chemicals.

4. Invest in the development of safer chemicals, materials, and processes.

Forward-thinking companies have taken steps to develop products with ingredients known to be safe and produce food without pesticides. To further enable such innovation, however, we need investment from government agencies and major industries. Much of Washington's history has been shaped by pioneering spirit, innovation, and a strong commitment to develop new technologies. Now it's time for Washington state to support development of production practices that no longer rely on toxic chemicals, and invest in green chemistry—processes that substitute safer and biologically-based methods for production. Research on alternatives to pesticides should also be a priority.

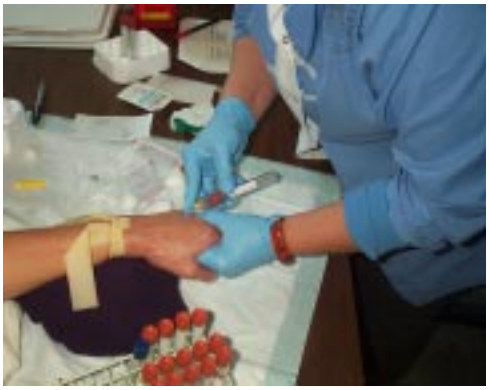
Universities are a key player in several arenas. They have a significant role to play in developing the basic technology for green chemistry practices, and the state legislature should support them in doing so. Washington State University has established a startup program on Biological and Organic Agriculture to develop

alternatives to toxic pesticides for growers. This program should be substantially expanded to provide resources for many crops across Washington. To provide resources for other industries, Washington should develop an institute modeled on Massachusetts' Toxics Use Reduction Institute, which conducts and disseminates research on safer substitutes and helps companies develop and implement plans to reduce pollution. The research and assistance should be coupled with a requirement to create and fulfill such plans. The state's tax structure should also provide incentives for companies to reduce their use and release of toxic chemicals.

Time for Strong Leadership

These reforms will not be completed overnight, but it is critical that we start down the path to a healthier future now. Leadership from Governor Gregoire, the Washington State Legislature, and the Departments of Ecology, Health, and Agriculture is essential to initiate lasting change. It is time to create a new common-sense chemicals policy so that we can move from today's toxic reality to a legacy of health for tomorrow.

Appendix 1: Materials and Methods



The ten participants in our sample were selected for diversity in occupations, geography, age, gender, and race. Each participant was asked to complete an exposure assessment questionnaire and provide information about their residences, occupations, diet, and potential toxic exposures. Samples were taken primarily in September and October of 2005. Nurses and a certified phlebotomist collected blood samples into vacutainers; after clotting, serum was obtained by centrifuging tubes and pouring off or pipetting serum into storage vials. One vacutainer of whole blood was maintained for each participant for lead testing. Participants provided first morning void urine samples for phthalate testing and mid-morning urine samples for pesticide and arsenic testing. Hair samples for mercury analysis were cut from the base of the scalp (or beyond, if necessary); in one case, chest hair was collected due to insufficient head hair.

PFC, PBDE, and Phthalate Analysis

AXYS Analytical Services (Victoria, BC) analyzed serum samples for perfluorinated compounds and PBDEs and urine samples for phthalates. Below are the laboratory's methods, in brief.

PBDEs: EPA Method 1614, an HRGC/HRMS method using an isotope dilution internal standard quantification.

Phthalate monoesters: Each separate urine sample was spiked with a suite of ^{13}C mono-ester surrogate standards and with an enzyme to release the mono-esters from their glucoronated form. The sample was extracted on NEXUS SPE cartridges, eluted, and analyzed by liquid chromatography tandem mass spectrometry (LC/MS/MS).

PFCs: For each individual, a 2 mL serum sample was homogenized with 0.25M sodium carbonate and 0.5M tetrabutylammonium hydrogen sulfate to disrupt cells and form respective ion pairs. The aqueous homogenate was then extracted with methyl tert-butyl ether (MTBE). The MTBE solution was reduced to dryness and reconstituted in 50:50 water/methanol. Analysis was performed using LC/MS/MS with a Genesis C8 (2.1 mm x 50 mm, 4 μm) chromatographic column. Quantification was performed by standard curve using perfuorononanoic acid or perfluorododecanoic acid as the internal standard.

Pesticide, Arsenic, Lead, and PCB Analysis

Pacific Toxicology Labs (Los Angeles, CA) and National Medical Services (Willow Grove, PA) analyzed urine samples for organophosphate pesticides, 1-napthol, 2,5,6-TCP, 2,4-D, and arsenic; serum for organochlorine pesticides and PCBs; and whole blood for lead. Below are the laboratories' methods, in brief.

Organochlorine pesticides: A hexane extract of serum was concentrated and analyzed by electron capture capillary gas chromatography using the internal standard method.

PCBs: The Webb-McCall method was used, in which PCBs were extracted from de-proteinized serum with 1:1 hexane/ethyl ether. PCBs were separated from organochlorine pesticides and biogenic material by chromatography on silica gel using hexane as eluent. PCB concentrations in the eluent were determined by electron capture gas chromatographic analysis using Webb-McCall mean weight percent factors and the internal standard method.

Organophosphate pesticide metabolites: Freeze-dried urine samples were derivatized with a benzyltoyltriazine reagent to produce benzyl derivatives of alkylphosphate metabolites. A saturated salt solution was added to the tubes and the benzyl derivatives were extracted with cyclohexane and analyzed by gas chromatography with flame photometric detection.

Arsenic: Arsenic was reduced to the trivalent form with potassium iodide and extracted into toluene. After a back-extraction into aqueous HNO_3, the arsenic was measured by graphite furnace atomic absorption spectophotometry.

Lead: Lead in whole blood was measured on graphic furnace atomic absorption spectroscopy. Blood samples were mixed with antifoam agent and Triton X-100 prior to analysis. The sample was atomized at 2800° C in a graphite tube and light absorbed was measured by passing monochromatic light from a cathode tube. A calibration curve was generated from known standards and the values for unknown samples were extrapolated.

3,5,6-TCP and 2,4-D: These compounds were measured in urine by gas chromatography/mass spectrometry (GC/MS). The samples were hydrolyzed in acid to release the compounds from conjugates. After extraction in butyl chloride, the compounds were derivatized to form trimethylsilyl derivatives prior to analysis in the GC/MS in the SIM mode. A calibration curve was generated from known standards.

Two samples were analyzed by National Medical Services due to changes in procedures at Pacific Toxicology.[8] For 3,5,6-TCP, urine samples

were made acidic with HCl and heated to convert any conjugated compound into the un-conjugated form. Samples were extracted with an organic solvent that was tested on an Agilent 5890 gas chromatograph with an electron capture detector. The analytical column was a 30-meter x 0.32 mm ID with a 0.25 micron DB-1 film, and it ran on a temperature program from 100° C to 300° C at a rate of 20° C per minute. Calibrators at concentrations of 50, 100, 500 and 1000 ppb and controls at 75 and 750 ppb were run to calibrate and control each analytical batch.

For 2,4-D, urine samples were made acidic with HCl and extracted with an organic solvent, back-extracted into a sodium hydroxide solution that was made acidic and extracted with an organic solvent. Extractions were analyzed on an Agilent 6890 gas chromatograph with an electron capture detector. The analytical column was a 30-meter x 0.32 mm ID with a 0.25 micron DB-5 film, and it ran on a temperature program from 140° C to 280° C to at a rate of 10° C per minute. Calibrators at concentrations of 20, 50, 100, 200, 400, and 600 ppb, and controls at 50 and 300 ppb were run to calibrate and control each analytical batch.

Carbamates: Carbaryl in urine was measured by GC/MS as the metabolite 1-napthol. The samples were hydrolyzed in acid to release the compounds from conjugates. After extraction in butyl chloride, the compounds were derivatized to form trimethylsilyl derivatives prior to analysis in the GC/MS in the SIM mode. A calibration curve was generated from known standards.

Again, two samples were analyzed by National Medical Services due to changes in procedures at Pacific Toxicology. A 0.5-mL aliquot of urine was buffered with saturated ammonium chloride and an internal standard (methyl-phenobarbital) was added. This was extracted with a mixed organic solvent that was evaporated to dryness and reconstituted with the HPLC mobile phase. Reconstituted extractions were analyzed on a Zorbax Stable Bond C-18, 4.6 x 150 mm analytical column with an Optimize 3mm cartridge C-18 precolumn. The mobile phase consisted of 24% CH_3CN in 0.05 M KH_2PO_4, 0.007 M 1-heptanesulfonate sodium, and 0.01 M triethylamine (adjusted to pH 3.0 with H_3PO_4). Detection was by UV at 210 nm. Calibrators for both carbaryl and 1-naphthol at concentrations of 100, 400, 1000, and 4000 ppb, and controls at 400 and 2000 ppb, were run to calibrate and control each analytical batch.

Mercury Analysis

Mercury analysis in hair was conducted by Brooks Rand (Seattle, WA). Hair samples were homogenized by cutting the hair into small pieces. The hair was transferred to clean glass jars. Sufficient acetone to cover each sample was added, and the jars were capped and shaken for 30 seconds. After allowing the hair to settle, the acetone was pipetted off. The samples were then cleaned three times with Triton TX-100 washes, followed by rinses with deionized water and filtration. The hair samples were then transferred to clean, dry, 40 mL glass vials and dried in a 40° C oven overnight. Two aliquots of quartz wool were homogenized according to the same procedure and analyzed along with the samples. Ho-

[8] *Pacific Toxicology ceased offering certain analyses during the course of our study; some of the analyses for two samples, those of Karen Bowman and Denis Hayes, were performed by National Medical Services.*

mogenized and cleaned hair samples were prepared and analyzed in accordance with the Appendix to EPA Method 1631. Hair samples were digested with a nitric/sulfuric acid solution and further oxidized with bromine monochloride. All samples were then analyzed with stannous chloride reduction, gold amalgamation, and cold vapor atomic fluorescence spectroscopy (CVAFS) detection using a BRL Model III CVAFS Mercury Analyzer. All sample results for low-level mercury analysis were blank corrected.

Data Analysis

For phthalates, PFCs, PBDEs, DDE, and PCBs, medians were calculated setting non-detectable values at the detection limit divided by the square root of two. Total PBDEs were calculated in the same manner. Medians were not calculated for arsenic, lead, organophosphate pesticides, or carbaryl because of the relatively high number of participants with undetectable levels. The influence of diet on pesticide levels was analyzed by computing the Spearman correlation between (1) the number of organophosphate and/or carbaryl metabolite detections per week and (2) the number of organic meals per week. Spearman's correlation coefficient was calculated, and it was significantly different from zero at $p=0.028$, indicating a statistically significant correlation.

Appendix 2: Chemical Information and Test Results

Table 2: Chemicals and Breakdown Products Tested

Chemical Group	Specific Chemical Tested	Chemical Name or Explanation
Phthalates	mMeP	Metabolite of DMP (dimethyl phthalate) – used in hair-care products, solid rocket propellant, insect repellants, and plastics
	mEtP	Metabolite of DEP (diethyl phthalate) – found in personal care products such as perfume, cologne, aftershaves, deodorants, shampoo, and hand lotion
	mBuP	Metabolite of DBP (dibutyl phthalate) – found in personal care products such as nail polish and in pharmaceuticals
	mBzP	Metabolite of BzBP (benzylbutyl phthalate) – found in vinyl flooring, car-care products, personal-care products, adhesives, and sealants
	mEHP	Metabolites of DEHP (di-(2-ethylhexyl) phthalate) – found in PVC products including medical products such as tubing; auto interiors; consumer products such as clothing, diaper covers, shower curtains, and furniture
	mEOHP	
	mEHHP	
PBDEs		Measurement of PBDEs (polybrominated diphenyl ethers); value reported is sum of levels of 40 congeners, adjusted for lipid content of blood.
Metals	Lead	
	Arsenic	Measurement of speciated arsenic, which is a summation of inorganic arsenic, demethylarsinic acid, and monomethylarsonic acid. Does not include organic arsenic
	Mercury	
Perfluorinated Compounds	PFOA	Perfluorooctanoic acid
	PFNA	Perfluornonananoic acid
	PFDA	Perfluorodecanoic acid
	PFUnA	Perfluoroundecanoic acid
	PFHxS	Perfluorohexanoic sulfonate
	PFOS	Perfluorooctane sulfonate
	PFOSA	Perfluorooctane sulfonamide
Pesticides	1-napthol	Metabolite of carbaryl pesticide
	DMP	Metabolites of organo-phosphate pesticides: Dimethylphosphate
	DMTP	Dimethylthiophosphate
	DMDTP	Dimethyldithiophosphate
	DEP	Diethylphosphate
	DETP	Diethylthiophosphate
	DEDTP	Diethyldithiophosphate
Persistent Toxic Chemicals (banned)	PCBs	Total PCBs tested in blood
	p,p'-DDE	Metabolite of DDT

Table 3: Results

Chemical Class	Testing Medium	Chemical Tested	Ann Holmes Redding	Patricia Dawson	Pam Tazioli	Denis Hayes	Lisa Brown	Bill Finkbeiner	Laurie Valeriano	Deb Abrahamson	Allyson Schrier	Karen Bowman
Phthalates (shown as ppb)	urine	mMeP	<1.07	<6.08	3.49	1.8	<3.40	11.2	<5.00	4.27	<8.00	8.05
		mEtP	<1.96	85.9	302	52.3	73.2	234	15.2	163	38.1	189
		mBuP	<0.900	24.8	16.4	14.9	158	78.1	17.1	9.13	68.8	134
		mBzP	6.48	12.4	25.7	16.9	59.3	99	23.9	9.2	37.2	96.7
		mEHP	<3	3.8	7.52	3.3	10.3	43.7	<3	3.7	7.8	51.9
		mEOHP	1.68	21.3	35.7	39.8	28.1	73.6	13.5	22.7	38.9	211
		mEHHP	5.16	40	74.1	72	56.7	165	24.5	42.5	60.4	338
Chemical Class	**Testing Medium**	**Chemical Tested**	**Ann Holmes Redding**	**Patricia Dawson**	**Pam Tazioli**	**Denis Hayes**	**Lisa Brown**	**Bill Finkbeiner**	**Laurie Valeriano**	**Deb Abrahamson**	**Allyson Schrier**	**Karen Bowman**
PBDEs (shown as ppt on a lipid weight basis)	blood serum	Br2-DPE-7	< 5.15	< 6.21	< 9.46	< 6.90	< 6.33	< 12.6	< 5.16	< 5.90	< 7.05	< 6.68
		Br2-DPE-8/11	< 5.01	< 5.40	< 8.36	< 5.58	< 5.19	< 10.2	< 5.16	< 4.75	< 5.67	K 5.84
		Br2-DPE-10	6.19	K 8.10	K 14.3	< 8.08	< 7.46	< 14.5	< 5.49	K 7.71	< 8.31	< 7.63
		Br2-DPE-12/13	< 5.01	< 5.40	< 8.36	< 5.14	< 4.81	< 8.49	< 5.16	< 4.75	< 5.67	< 4.53
		Br2-DPE-15	692	278	68.3	417	286	89.8	406	272	94.5	145
		Br3-DPE-17/25	72.9	159	K 36.7	58	65.3	145	35.8	45.3	37.5	92.2
		Br3-DPE-28/33	3030	2540	668	1440	1290	960	759	2180	741	995
		Br3-DPE-30	< 6.92	< 8.64	< 12.7	< 8.37	< 7.72	< 13.2	< 6.16	< 7.05	< 7.94	< 8.58
		Br3-DPE-32	< 5.30	< 6.62	< 9.87	< 6.46	< 5.95	< 10.2	< 5.16	< 5.41	< 6.17	< 6.56
		Br3-DPE-35	8.69	< 5.40	< 8.36	< 5.14	K 5.57	< 8.68	< 5.16	< 4.75	< 5.67	< 5.60
		Br3-DPE-37	10.3	K 14.0	< 8.36	9.55	11.3	K 10.6	K 5.82	18.4	< 5.67	9.78
		Br4-DPE-47	28000	83000	9900	17200	23800	20900	15000	27200	19000	15900
		Br4-DPE-49	82.6	177	95.5	259	190	215	109	203	125	178
		Br4-DPE-51	K 10.5	15.9	< 15.9	< 10.6	12	30.4	< 9.98	< 11.1	< 12.7	14.5
		Br4-DPE-66	197	670	119	142	228	219	108	216	134	142
		Br4-DPE-71	< 12.8	35.6	< 22.8	< 15.1	< 17.1	< 43.4	< 14.3	< 15.9	< 18.1	< 17.6
		Br4-DPE-75	33.7	119	< 18.5	20.7	27.8	< 35.3	16.3	26.1	23.8	20.5
		Br4-DPE-77	< 7.22	< 7.02	< 13.4	< 8.96	< 10.0	< 27.7	< 8.49	< 9.51	< 10.6	< 11.2
		Br4-DPE-79	K 79.4	K 212	K 46.7	K 69.0	K 80.1	K 47.6	K 44.3	K 117	K 53.3	K 32.1
		Br5-DPE-85	439	1490	163	151	392	494	181	885	300	201
		Br5-DPE-99	4550	34200	1860	1970	4520	6380	2100	4490	4020	2600
		Br5-DPE-100	4040	8660	1110	1720	3060	3170	1650	7740	3640	1930
		Br5-DPE-105	< 10.2	< 15.5	< 16.2	< 8.96	< 44.7	< 18.9	< 66.9	< 61.8	< 8.06	< 18.2
		Br5-DPE-116	< 13.8	< 20.9	< 22.3	< 12.5	< 54.8	< 23.0	< 82.4	< 75.7	< 11.1	< 22.4
		Br5-DPE-119/120	24.7	66.8	< 14.8	20.9	< 29.5	< 12.5	< 48.3	< 40.8	< 7.31	18.1
		Br5-DPE-126	< 5.01	K 12.2	< 8.36	< 5.14	< 15.7	< 7.74	< 23.3	K 22.0	< 5.67	< 7.15
		Br6-DPE-128	< 42.4	< 46.4	< 89.9	< 27.5	< 34.3	< 66.6	< 44.3	< 32.3	< 55.2	< 44.3
		Br6-DPE-138/166	79.2	232	< 84.9	K 32.9	63.6	K 140	30.5	177	68.3	45.8
		Br6-DPE-140	33.1	96.1	< 52.6	33.5	35.7	< 51.9	16.5	105	81	21.5
		Br6-DPE-153	1590	2960	1030	6240	2490	17300	1380	7920	11700	1410
		Br6-DPE-154	409	2030	144	176	354	381	151	723	402	197
		Br6-DPE-155	48.5	207	< 32.9	K 38.0	K 55.4	38.3	K 26.0	83.3	K 79.0	K 34.6
		Br7-DPE-181	< 19.3	< 30.5	< 27.0	< 20.7	< 21.5	< 62.1	< 23.0	< 21.8	< 26.6	< 38.5
		Br7-DPE-183	249	1660	149	223	183	K 153	331	213	249	169
		Br7-DPE-190	36.1	102	< 40.3	68.6	K 41.0	< 96.2	52.6	K 43.1	39.7	< 59.7
		Br8-DPE-203	118	124	K 84.7	151	175	K 179	156	152	K 116	K 173
		Br9-DPE-206	< 801	< 735	< 1220	< 799	< 688	< 1030	< 905	1000	< 685	< 649
		Br9-DPE-207	885	1230	< 1150	< 756	2280	1120	894	1570	679	K 1100
		Br9-DPE-208	< 524	< 481	< 799	< 523	543	808	< 592	620	< 448	736
		Br10-DPE-209	< 9720	< 8910	< 14800	< 9690	< 8350	< 12500	< 11000	< 10800	< 8310	< 7870
Chemical Class	**Testing Medium**	**Chemical Tested**	**Ann Holmes Redding**	**Patricia Dawson**	**Pam Tazioli**	**Denis Hayes**	**Lisa Brown**	**Bill Finkbeiner**	**Laurie Valeriano**	**Deb Abrahamson**	**Allyson Schrier**	**Karen Bowman**
PFCs, or perfluorinated compounds (shown as ppb)	blood serum	PFBA	< 0.548	< 0.548	< 0.548	< 0.548	< 0.548	< 0.548	< 0.548	< 0.548	< 0.548	< 0.548
		PFPeA	< 0.518	< 0.518	< 0.518	< 0.518	< 0.518	< 0.518	< 0.518	< 0.518	< 0.518	< 0.518
		PFHxA	< 0.506	< 0.506	< 0.506	< 0.506	< 0.506	< 0.506	< 0.506	< 0.506	< 0.506	< 0.506
		PFHpA	< 0.508	< 0.508	< 0.508	< 0.508	< 0.508	< 0.508	< 0.508	< 0.508	< 0.508	< 0.508
		PFOA	3.45	3.64	3.6	5.24	4.1	7.35	0.65	2.88	2.07	4.6
		PFNA	0.712	1.51	1.53	0.953	1.83	1.96	< 0.522	0.761	0.598	1.34
		PFDA	< 0.518	0.648	< 0.518	< 0.518	0.78	0.711	< 0.518	< 0.518	< 0.518	< 0.518
		PFUnA	< 0.524	< 0.524	0.553	0.538	< 0.524	0.779	< 0.524	< 0.524	< 0.524	< 0.524
		PFDoA	< 0.530	< 0.530	< 0.530	< 0.530	< 0.530	< 0.530	< 0.530	< 0.530	< 0.530	< 0.530
		PFBS	< 0.996	< 0.996	< 0.996	< 0.996	< 0.996	< 0.996	< 0.996	< 0.996	< 0.996	< 0.996
		PFHxS	1.7	1.33	< 1.02	5.32	1.69	< 1.02	< 1.02	3.57	< 1.02	1.59
		PFOS	6.36	19.4	20.2	26.3	25.2	49.4	3.27	29.8	8.15	22.3
		PFOSA	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500

Table continued on next page

Table 3: Results (continued from previous page)

Chemical Class	Testing Medium	Chemical Tested	Ann Holmes Redding	Patricia Dawson	Pam Tazioli	Denis Hayes	Lisa Brown	Bill Finkbeiner	Laurie Valeriano	Deb Abrahamson	Allyson Schrier	Karen Bowman
Metals	whole blood	Pb (µg/dl)	<3.0	<3.0	<3.0	<3.0	<3.0	<3.0	<3.0	<3.0	<3.0	3.4
	urine	As (ppb)	<10	12	13	16	15	<10	<10	<10	<10	14
		As (creatinine corrected, ppb)	N/A	10	54	10	9	N/A	N/A	N/A	N/A	34
	hair	Hg (ppb)	787	E987	587	2020	1080	1840	397	59.5	634	1860
Chemical Class	**Testing Medium**	**Chemical Tested**	**Ann Holmes Redding**	**Patricia Dawson**	**Pam Tazioli**	**Denis Hayes**	**Lisa Brown**	**Bill Finkbeiner**	**Laurie Valeriano**	**Deb Abrahamson**	**Allyson Schrier**	**Karen Bowman**
Organochlorine pesticides (shown as ppb)	blood serum	p,p'-DDT	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
		p,p'-DDE	8.67	12.56	0.8	0.42	1.8	<0.20	<0.20	2.21	0.26	1.94
		p,p'-DDD	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
		alpha-chlordane	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
		gamma-chlordane	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
		trans-nonachlor	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
		heptachlor	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
		heptachlor epoxide	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
		oxychlordane	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
		lindane (gamma-BHC)	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
		beta-BHC	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
		dieldrin	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
		hexachlorobenzene	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Chemical Class	**Testing Medium**	**Chemical Tested**	**Ann Holmes Redding**	**Patricia Dawson**	**Pam Tazioli**	**Denis Hayes**	**Lisa Brown**	**Bill Finkbeiner**	**Laurie Valeriano**	**Deb Abrahamson**	**Allyson Schrier**	**Karen Bowman**
PCBs (shown as ppb)	blood serum	PCBs	1.5	2.3	1	0.9	1.2	0.6	0.2	0.8	0.4	1.1
Chemical Class	**Testing Medium**	**Chemical Tested**	**Ann Holmes Redding**	**Patricia Dawson**	**Pam Tazioli**	**Denis Hayes**	**Lisa Brown**	**Bill Finkbeiner**	**Laurie Valeriano**	**Deb Abrahamson**	**Allyson Schrier**	**Karen Bowman**
Carbaryl (shown as ppb)	urine	1-napthol	4.4	<1.0	<1.0	<20	6.8	9.9	<1	1.3	6.8	<10
		1-napthol (creatinine corrected)	11.3	N/A	N/A	N/A	4.1	25.4	N/A	1.7	N/A	N/A
Chemical Class	**Testing Medium**	**Chemical Tested**	**Ann Holmes Redding**	**Patricia Dawson**	**Pam Tazioli**	**Denis Hayes**	**Lisa Brown**	**Bill Finkbeiner**	**Laurie Valeriano**	**Deb Abrahamson**	**Allyson Schrier**	**Karen Bowman**
Organophosphate pesticides (shown as ppb)	urine	DMP	5.5	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	16.1	<5.0	<5.0
		DMP (creatinine corrected)	14	N/A	N/A	N/A	N/A	N/A	N/A	21	N/A	N/A
		DMTP	12.8	7.4	<5.0	<5.0	13.5	<5.0	<5.0	13.9	<5.0	<5.0
		DMTP (creatinine corrected)	33	6	N/A	N/A	8	N/A	N/A	19	N/A	N/A
		DMDTP	<10.0	<10	<10	<10	<10.0	<10.0	<10.0	<10	<10.0	<10.0
		DEP	<5.0	<5	<5	<5	<5.0	<5.0	<5.0	5.6	<5.0	<5.0
		DEP (creatinine corrected)	N/A	N/A	N/A	N/A	N/A	N/A	N/A	7	N/A	N/A
		DETP	<5.0	<5	<5	<5	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0
		DEDTP	<10.0	<10	<10	<10	<10.0	<10.0	<10.0	<10.0	<10.0	<10.0
Chemical Class	**Testing Medium**	**Chemical Tested**	**Ann Holmes Redding**	**Patricia Dawson**	**Pam Tazioli**	**Denis Hayes**	**Lisa Brown**	**Bill Finkbeiner**	**Laurie Valeriano**	**Deb Abrahamson**	**Allyson Schrier**	**Karen Bowman**
Chlorpyrifos (shown as ppb)	urine	3,5,6-TCP	<2	<2	<2	<2	<2	<2	<2	<2	<2	<50
Chemical Class	**Testing Medium**	**Chemical Tested**	**Ann Holmes Redding**	**Patricia Dawson**	**Pam Tazioli**	**Denis Hayes**	**Lisa Brown**	**Bill Finkbeiner**	**Laurie Valeriano**	**Deb Abrahamson**	**Allyson Schrier**	**Karen Bowman**
2,4-D (shown as ppb)	urine	2,4-D	<2	<2	<2	<20	<2	<2	<2	<2	<2	<20

K-flagged values indicate a peak was detected but did not meet quantification criteria; the result represents the estimated maximum possible concentration. These values were not included in calculations of sums.

E-flagged value: the laboratory has qualified this value as an estimate due to sample inhomogeneity.

Table 4: Results Summary

Chemical	Anne Holmes Redding	Patricia Dawson	Pam Tazioli	Denis Hayes	Lisa Brown	Bill Finkbeiner	Laurie Valeriano	Deb Abrahamson	Allyson Schrier	Karen Bowman	Study Median	National Median/ Median Range
MBP	<LOD	24.8	16.4	14.9	158	78.1	17.1	9.13	68.8	134	30	19.1
MEHP	<LOD	3.8	7.52	3.3	10.3	43.7	<LOD	3.7	7.8	51.9	5.7	4.1
PBDEs	52.6	147.5	28.5	38.8	46.7	62.6	32.5	63.8	48.3	40	47.5	47.9
PFOS	6.36	19.4	20.2	26.3	25.2	49.4	3.27	29.8	8.15	22.3	21.3	10.4-40.2
PFOA	3.45	3.64	3.6	5.24	4.1	7.35	0.65	2.88	2.07	4.6	3.6	2.1-7
Mercury	787	987	587	2020	1080	1840	397	59.5	634	1860	887	430-620
Carbaryl (measured as 1-napthol)	4.4	<1.0	<1.0	<20	6.8	9.9	<1	1.3	6.8	<10	<LOD	<LOD
DMP	5.5	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	16.1	<5.0	<5.0	<LOD	<LOD
DMTP	12.8	7.4	<5.0	<5.0	13.5	<5.0	<5.0	13.9	<5.0	<5.0	<LOD	<LOD
DDT (measured as DDE)	8.67	12.56	0.8	0.42	1.8	<0.20	<0.20	2.21	0.26	1.94	1.3	1.8
PCBs	1.5	2.3	1	0.9	1.2	0.6	0.2	0.8	0.4	1.1	0.95	0.9-1.5

Notes:
LOD = limit of detection
Results are presented in ppb (parts per billion)

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