

BODY OF EVIDENCE

A Study of Pollution in Maine People



Alliance for a Clean and Healthy Maine

May 2007

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The conclusions and recommendations in this report are those of the authors and sponsoring organizations and do not necessarily reflect the views and opinions of the project funders, University of Southern Maine, principal investigators, advisors, or participants. The authors and sponsors accept all responsibility for any errors or omissions in this work.

This study was sponsored by the Alliance for a Clean and Healthy Maine, a diverse coalition of Maine-based organizations committed to protecting human health from toxic chemical exposure. Forty-five organizations have endorsed the Alliance, representing health-affected children, workers, doctors, public health professionals, environmentalists, and impacted communities.

Visit the Alliance for a Clean and Healthy Maine at www.cleanandhealthyme.org or contact us at (207) 772-2181, One Pleasant Street, Fourth Floor, Portland, Maine 04101.

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Executive Summary

Maine people are polluted with dozens of hazardous industrial chemicals, according to a new study conducted by the Alliance for a Clean and Healthy Maine with help from the University of Southern Maine. In 2006, thirteen Maine men and women volunteered to have their bodies tested in the first-ever study of chemical pollution in Maine people. This study found a total of 46 different chemicals (of 71 tested) in samples of blood, urine, and hair. On average, each participant had measurable levels of 36 toxic chemicals in their bodies.

These findings show that Maine people are routinely exposed to hazardous industrial chemicals including phthalates from cosmetics and vinyl plastic, brominated flame retardants (PBDEs) from televisions and furniture, Teflon chemicals from stain-resistant and non-stick coatings, bisphenol A from reusable water bottles and baby bottles, and toxic metals such as lead, mercury and arsenic.

These chemicals are found in products we use every day: plastic containers, toys, furniture, fabric, automobiles, TVs and stereos, water bottles, medical supplies, and personal products like shampoo, hairspray, and perfume. They are in our homes and offices, food and water, and the air we breathe.

Scientific research shows that these chemicals are hazardous and that even tiny amounts may threaten human health. They are toxic or harmful to life and many are slow to degrade and also build up to high levels in the food chain. Babies in the womb and young children are especially vulnerable because they are still growing. Animal and human studies have linked these chemicals to learning and developmental disabilities, endocrine system damage, changes in sexual development, reproductive harm (including decreased sperm count in men), low birth weight and some cancers.

Despite proven and suspected dangers to our health, industry is not required to demonstrate the safety of chemicals before adding them to consumer products, nor are they required to use safer alternatives to chemicals known to be hazardous. Recognizing that the safety system for industrial chemicals is broken, the Alliance for a Clean and Healthy Maine recommends that a comprehensive safer chemicals policy should be developed and adopted by government to:

CLOSE THE SAFETY GAP by phasing out the most harmful chemicals in favor of safer alternatives, searching for safer substitutes for all chemicals shown to be hazardous, and requiring that all industrial chemicals are proven safe, especially for children;

CLOSE THE DATA GAP by honoring the public's right-to-know which hazardous chemicals are in what products, and by requiring manufacturers to provide health and safety data on all chemicals;

CLOSE THE TECHNOLOGY GAP by investing in green chemistry research and development (R&D) to make bio-based plastics from Maine potatoes and other crops to boost the state's economy through production of safer alternatives to toxic petroleum-based plastics.

The People

Regina Creeley, 54, lives in Hudson. She is married with two grown children. Regina began working as a classroom assistant when she was 14 years old. Now a special education instructor, Regina says she has noticed a dramatic increase in the number of students in her school with special needs.

Regina had the highest total arsenic level of all study participants, which was probably due to her recent meal of shellfish, which contain a non-toxic form of arsenic. She had the lowest mercury level.

Paulette Dingley, 48, has spent much of her life working to provide home care for adults with disabilities. She now works with the American Red Cross as a health and safety instructor. She lives in Auburn.

Paulette had the highest level of two types of phthalates. She was one of three participants who had bisphenol A chemicals in their bodies at levels several times higher than the national average.

Dana Dow, 56, is a Republican state senator who represents his hometown of Waldoboro and 20 other towns in midcoast Maine. He is serving his second legislative term and sits on the Marine Resources and Labor committees. Married with four children, Dana also owns a furniture store.

Dana had the highest levels, and most different types, of perfluorinated chemicals (PFCs), the Teflon chemicals. Senator Dow's levels were more than twice the national average level for PFOA and several other PFCs.

Amy Graham, 35, works out of her home in Farmington and splits her time between writing children's books and being a homemaker. She and her husband have two young daughters, Phoebe and Sylvie. Amy works to make the safest choices for her family. She breastfed both of her daughters, makes her own non-toxic cleaners, and includes many organic fruits and vegetables in her family's diet.

Amy had the second-highest level of one of the PBDEs which is a breakdown product of Deca, the toxic fire retardant, but the rest of her results were low to medium compared to the other participants.

Bettie Kettell, 60, is a nurse who lives in Durham. Bettie worked with her hospital's administration to implement pollution prevention goals to eliminate the use of unnecessary chemicals.

Bettie had the highest total level of PBDE flame retardants compared to the other Maine participants and was third highest in the PFCs. Of the 71 chemicals that were tested in this study, 41 were detected in Bettie, a tie for the most chemicals found.

Russell Libby, 50, is an organic farmer and executive director of MOFGA, the Maine Organic Farmers and Gardeners Association. He became involved with MOFGA after the first Common Ground Fair in 1977, where he saw a connection between local, organic food and a strong Maine economy. Russell lives on his farm in Mount Vernon.

Russell was tied for the most chemicals detected (41 of the 71 that were tested). He also had the greatest number of PBDEs detected (27 of 46) and higher levels of individual PBDEs.

Hannah Pingree, 30, is serving her third term in the Maine Legislature, and first as House Majority Leader, representing her hometown of North Haven and ten other islands and coastal towns. After learning that PBDE flame retardants were being found in breast milk, Hannah sponsored a bill that successfully phased out two hazardous flame retardants in 2004. She continues to be a strong voice for the phase-out of unnecessary dangerous chemicals in Maine.

Hannah had the second highest level of total phthalates and second highest level of mercury in the Maine study group. Her mercury levels were above the safety standard for protection of a developing fetus from subtle but permanent brain damage.

Lauralee Raymond, 28, grew up in Aroostook County and attended Bates College. She currently lives in Winthrop, but lived in Hallowell at the time of our study. She works as a lobbyist. Lauralee and her mother Vi both participated in the Body of Evidence study.

When she received her results, Lauralee was struck by the fact that many of her chemical levels were higher than her mother's. She expected the opposite, since her mother is older and has had more time to build up her levels of contaminants. Lauralee had higher levels of mercury, arsenic, and each of the PBDE flame retardants than her mother.

Vi Raymond, 51, moved to Winthrop after spending 40 years in Fort Kent. Married with five grown children, including fellow participant Lauralee, Vi has long advocated for safer workplaces in her jobs with PACE, the paperworker's union, and the AFL-CIO.

Vi had the highest phthalates total, and the highest level of BADGE-4OH, one of the bisphenol A chemicals tested. Her bisphenol A levels were several times the national average.

Elise Roux, 18, is a senior at Cheverus High School in Portland. She lives in Windham. A soccer goalie and cancer survivor, Elise is active in Kids Against Toxins, a Portland area group of students working to reduce pollution and eliminate unnecessary uses of toxic chemicals.

Elise had the highest level of bisphenol A, which was about ten times the national average, and the second-highest level of the related compound BADGE-4OH.

Charlie Schmidt, 42, is an award-winning freelance science writer from South Portland. Charlie has a master's degree in public health, and also has worked as a toxicologist.

Charlie brings a professional appreciation to the growing interest in the human chemical body burden, and the challenging implications for public health.

Eric Stirling, 32, owns and operates a sporting camp on First West Branch Pond, near the Appalachian Trail in the unorganized territory TA-R12. Except for his four years of college at Bates, Eric spent almost his entire life at the camp, which has been in his family for four generations and is thought to be Maine's oldest continuously operated sporting camp.

Eric had the highest level of mercury found among the study participants, and his total arsenic amount was above the normal exposure level.

Denyse Wilson, 39, is a writing instructor. She is married with two children, Cecil (six years old) and Francine (four years). While renovating their 85-year-old home in downtown Bangor, Denyse and her family were exposed to lead dust from old paint. Since then, her family's lead levels have decreased, and Denyse has spoken out to improve education about lead poisoning prevention.

Denyse had the highest inorganic arsenic and arsenic(III) levels of all study participants, and the total arsenic measured was higher than normal exposure.

Introduction

Pollution of our air, water, and land has been studied for years, prompting laws to protect public health and the environment. Now scientists are finding pollution in people. Much of it comes from the unnecessary use of toxic chemicals in common consumer products and plastics. Yet no effective policies are in place to keep hazardous chemicals out of our bodies. It's time that these new studies motivate government to act once again.

Until now, the level of dangerous chemical pollution in Maine people has not been known. Through a cooperative effort with the University of Southern Maine led by Dr. Rick Donahue, 13 men and women from Maine volunteered in 2006 to be tested for chemicals encountered in their everyday lives.

The results represent the first-ever report of nearly 50 toxic pollutants found in Maine people. By releasing these findings, the Alliance for a Clean and Healthy Maine seeks to elevate the public discussion about pollution in Maine people and promote action to fix our broken safety system that allows chemicals to build up in our bodies.

Chemicals are all around us—in the air we breathe, the water we drink, the food we eat, and the products that fill our homes, schools and workplaces. While some of these substances are harmless, other chemicals still in widespread use are known to be hazardous to our health and environment. The effects of most chemicals in commerce are largely unknown, since the chemical industry is not required to test their products for health and safety threats. Medical research is revealing that common chemicals can disrupt the normal functioning of our cells and organs and damage our health. Some chemicals also accumulate over time, building up and combining with each other inside our bodies.

Together, the chemicals inside of a living being add up to a total “body burden” of contamination. Each of us carries a chemical burden; for some, this burden can be more risky than for others, depending on our genetic makeup, health status, and socio-economic background. Some groups such as babies in the womb are especially vulnerable.

These 13 Maine residents join others across the United States, Canada and Europe who have volunteered for testing so that we may begin to understand our relationship with the chemicals in the world around us. By comparing the levels found in Maine people to other, similar populations in the U.S., we can track our exposure to toxic chemicals, while we work with others to change government policy and business practices to switch to safer alternatives.

This study focused on five groups of chemicals that have been linked to harmful effects:

- **Phthalates**, chemicals added to nail polish and many other beauty products, and to PVC plastic (vinyl) to make it more flexible for shower curtains and other soft plastics;
- **Polybrominated diphenyl ethers (PBDEs)**, the toxic flame retardants added to the plastic cases of televisions, and the fibers in draperies, furniture and other textiles;
- **Perfluorinated chemicals (PFCs)**, the extremely persistent Teflon chemicals used as protective coatings for fabrics, furniture, carpets, cookware and fast-food packaging;

- **Bisphenol A (BPA)**, a chemical used to make reusable plastic water bottles and baby bottles, the linings in metal food cans and dental sealants;
- **Metals**, including *lead*, found in old paints and plumbing, and in batteries, electronics, electrical wiring, ammunition, wheel weights, and other products; *mercury*, released from products and power plants into air and water, where it builds up in fish; and *arsenic*, a former pesticide that occurs naturally in soils and some Maine well water.

These particular chemicals were chosen because they are found in common products that are part of our modern lifestyle and have come under increasing scrutiny as potential threats to human health. Some are also known to be very long-lived in the environment (or persistent) and to build up in the food chain (or bioaccumulate). More and more, these chemicals are being shown to have adverse health effects at extremely low levels, in some cases levels below what was previously considered safe, especially at crucial moments in human growth. For example, we know that low-level exposures to lead and mercury harm the developing brain, causing lowered intelligence and learning and behavior problems.¹ How might exposure to other, less well-studied chemicals be harming our health?

Only in the last decade have scientists and doctors discovered that some chemicals, like brominated flame retardants and fluorinated stain-resistant coatings, move from the products in which they are used into the environment and into humans and wildlife. We know that these chemicals are harmful from animal studies. We don't understand very well the combined effects of these chemical exposures on human health, especially on fetuses and children who are more sensitive to toxic effects. We do know that human exposure levels for some of these chemicals approach or exceed toxic levels in animals.

Chemicals that interrupt the intricate processes of developing life can, at high levels, wreak havoc in the form of severe birth defects, or at lower levels cause subtle but important changes in development that surface later in childhood as learning or behavioral problems, or in adulthood in the form of certain cancers or deteriorating brain function. Researchers are only just beginning to understand these connections. Monitoring levels of toxic chemicals in people's bodies (or biomonitoring) can help set priorities for policy, substitution with safer alternatives, and further research.

Sources of potential exposure vary with our individual day-to-day routine activities. In this survey, information gathered from interviews with participants was used to develop possible routes of chemical exposure. Because of the multitude of chemicals we face every day, such exposure pathways are difficult to establish, but participants were provided with information about possible sources such as food consumption and product use.

Moving from the sources of chemicals inside us to what effects they might have on our health is a formidable, sometimes impossible task for environmental health professionals. It can be difficult to come up with easy answers to questions on the health impact of chemicals.² The results of this pilot study cannot be used to predict how a participant's health will be affected by his or her chemical body burden. Many factors influence whether or not exposure to toxic substances will result in a health problem, including:

- the type and nature of the chemical;
- when in his or her lifetime a person was exposed;
- how often a person was exposed, and for how long;
- the amount of the chemical exposure;
- the individual's genetic makeup and physical condition;
- the person's health and nutrition, and their access to quality health care; and
- the person's socio-economic status.

While we cannot make direct links to the health of Maine people, we can place the Maine results in the context of other national and regional biomonitoring studies and surveys, particularly the Centers for Disease Control and Prevention's Third National Report on Human Exposure to Environmental Chemicals,³ and similar small studies in Washington,⁴ California,⁵ Canada,⁶ and in six studies in the U.S. conducted by the Environmental Working Group, an early pioneer in the use of biomonitoring.⁷ However, it is important to note that this is not a statistical study (see Box) and comparisons should be made cautiously.

[BOX – This is not a controlled research study. Because of the small sample size, the study results can not be used to draw statistical conclusions about chemical exposures for various population sectors or the Maine public as a whole. The data from these tests provide a snapshot of the accumulation of and exposure to some chemicals in some long-time Maine residents. The only statistically-based compilation of nationwide measurements is the National Health And Nutrition Examination Survey (NHANES), conducted by the U.S. Centers for Disease Control and Prevention (CDC), which does not test for all the chemicals assessed in our project.]

Reviewing laboratory reports that describe the measurements of chemicals in humans can be overwhelming. The units are so small—parts per billion—they can be incomprehensible. As improved laboratory technology allows for the detection of smaller and smaller amounts of substances, we now can find all kinds of substances we never knew were inside of us. This creates a dilemma for both health professionals and the public, who are trying to understand whether they are at risk from the chemicals inside of them (see Sidebar). Yet absence of knowledge is not proof of safety.

The findings presented in this report beg us to err on the side of caution, for our health and the sake of our children's future. The history of widespread harm caused by toxic substances like lead, PCBs, and mercury demonstrates the need to act on early warnings. And when controlled laboratory experiments reveal a connection between exposure to these chemicals and brain damage or chronic diseases, our concern only increases. When there is plausible concern about serious environmental public health hazards, and scientific uncertainty about the cause-and-effect relationship, then precautionary action should be taken to prevent exposure and possible harm.

[SIDEBAR: Do Low Doses Pose a Danger?

A common argument against concerns about chemicals in people is that the presence of minute amounts of chemicals in our bodies is not necessarily a threat. Our technological ability to detect trace amounts of substances—parts per billion or trillion—is advancing faster than our scientific ability to determine the effects of such small amounts of chemicals. We can prove that a person was exposed, but we can not as easily pinpoint the source of the exposure, or say what the health effects

might be, or what the person should do about it.⁸ Just because we haven't found conclusive evidence in humans that a chemical causes some effect does not mean it is harmless. Many prescription drugs aimed at addressing a host of medical conditions cause the intended biological effects at effective doses similar to the low levels found for the chemical pollutants in this study. And emerging science reveals that many chemicals mimic natural hormones in the body that act at extremely low levels to regulate development, reproduction, immune function and many other biological systems.]

About this Report

All of the protocols for this project were approved by the University of Southern Maine, with oversight of methodology, data collection, laboratory testing, and data analyses provided by Dr. Rick Donahue and Dr. Vincent Markowski. Samples of blood, urine, and hair were analyzed by two accredited laboratories, AXYS Analytical Services in Victoria, British Columbia and Brooks Rand Labs in Seattle, Washington. See the Project Methodology section at the end of this report for further details.

The next section of this report discusses the overall findings. Following sections provide more detailed information on each group of chemicals found in the 13 Maine participants. The Conclusions and Recommendations place the report's findings in a larger context and identify the actions that government, businesses, and individuals can take to prevent pollution in people.

At the end of the report, the project and analytical methods are described, followed by a series of tables in the Appendix that report on the detailed results of every chemical tested for every participant. The report closes with authoritative references to the scientific journal articles, government reports, and other information sources that support the growing concern about pollution in people.

Discussion of Project Findings

Our study reveals that Maine people are polluted. We found a total of 46 different toxic chemicals of the 71 that we tested for in the bodies of 13 Maine people. The average body burden was 36 toxic chemicals detected in the blood, urine and hair of each participant. The chart below visually displays these results.

These findings show that Maine people are routinely exposed to many industrial chemicals.

These chemicals have hazardous properties such as toxicity (ability to harm life), and in some cases persistence (being slow to degrade) and bioaccumulation (building up in the food chain). Therefore, the routine exposure of Maine people to these chemicals poses a potentially serious health threat.

Many of the chemicals we found in Maine people are added to everyday consumer products, ranging from cosmetics and personal care products, televisions and electronics, furniture and carpeting, to cookware and clothing. They are found in common materials such as plastics, coatings, and adhesives. People are exposed during the use and disposal of these products, the ingestion of household dust, indoor air pollution, contaminants in the food supply, and drinking water.

The finding of dozens of mostly unregulated toxic chemicals in average Maine people shows that the safety system for industrial chemicals is broken and needs to be fixed. Current laws and practices do not prevent routine exposure to hazardous chemicals in our daily lives.

Chemical Tested	PHTHALATES							POLYBROMINATED BIPHENYL ETHERS																			
	mMeP	mEtP	mBuP	mBzP	mEHP	mEOHP	mEHHP	BDE-7	BDE-8/11	BDE-10	BDE-12/13	BDE-15	BDE-17	BDE-19	BDE-20	BDE-28	BDE-30	BDE-32	BDE-35	BDE-37	BDE-47	BDE-49	BDE-51	BDE-66	BDE-71	BDE-75	
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For detailed results for all chemicals measured in each participant, see the tables in the Appendix at the end of this report. Table 1 identifies the 71 chemicals tested, which fall into five chemical groups: phthalates, PBDEs (a group of brominated flame retardants), PFCs (the Teflon chemicals), bisphenol A chemicals (BPA), and metals (lead, mercury, and arsenic). Every one of the five groups of chemicals tested was detected in Maine people, although not every chemical was found in every participant.

Table 2 reports all of the chemical testing results for each individual participant. It shows which chemicals were found and at what level. It also indicates which chemicals were not detected and the lowest level measurable (i.e., the limit of detection). Table 3 summarizes the results for the whole group of Mainers and compares them to similar results from the national biomonitoring program or similar body burden studies conducted in certain states or nationally.

The findings for each of the five chemical groups are summarized on the following pages. The sections of the report that follow provide more details on each group of chemicals, along with citations to authoritative sources of information.

																					PERFLUORINATED CHEMICALS								BPA	METALS										
BDE-105	BDE-116	BDE-126	BDE-128	BDE-140	BDE-153	BDE-154	BDE-155	BDE-181	BDE-183	BDE-190	BDE-203	BDE-206	BDE-207	BDE-208	BDE-209	PFBA	PFPeA	PFHxA	PFHpA	PFOA	PFNA	PFDA	PFUnA	PFDoA	PFBS(80)	PFHxS(80)	PFOS(80)	PFOSA	BPA	BADGE-101	Lead	Methyl/Ine	Arsenic	Arsenic	Arsenic(III)					

Phthalates -- from Beauty Products to Beastly Vinyl. These chemicals are ubiquitous and unregulated in the United States. All seven of the phthalate compounds were detected in nearly every person tested. The levels of six of the seven phthalates detected in the participants were higher than the national average. For one phthalate, the median Maine value was higher than 95% of all Americans tested. Three phthalates were found in Maine people at levels higher than 75% of all Americans. Phthalates are added to thousands of personal care products and soft polyvinyl chloride (PVC) plastic used in everything from shower curtains and packaging to inflatable toys and IV bags in hospitals.

PBDEs -- the Toxic Fire Retardants. These chemicals are found everywhere we look. Maine and others have recently banned two commercial PBDE products known as Penta and Octa, and is considering legislation to replace the most widely used PBDE mix called Deca with safer alternatives. We found 28 of the PBDEs in Maine people out of the 46 that we tested for (out of the 209 PBDEs known to science). The PBDE levels found in Maine people were generally comparable to those found in other studies. Two of the PBDEs that are known breakdown products of Deca, BDE-153 and BDE-183, were higher than levels found in Washington and California residents. Eighty percent of Deca is added to the plastic casing of televisions to slow the spread of flames in a fire, with additional uses in textiles (commercial drapes and furniture) and electrical wires and cables.

PFCs -- the Stain-Resistant Teflon Chemicals. Of the 13 perfluorinated chemicals (PFCs) tested, we found six in Maine people. These persistent chemicals are found everywhere in the environment. They are largely unregulated, although voluntary actions are being taken to reduce the use of PFOS and PFOA, the two most studied of the PFCs. Three of the PFCs detected in Maine people were found at levels above the average or median levels reported in other studies. The PFCs are used as stain- and water-resistant coatings on furniture, clothing, and carpets; grease-resistant coatings in fast-food packaging; non-stick coatings for cookware; and other Teflon products including the breathable, water-resistant fabric known as Gore-Tex.

Bisphenol A -- the Hormone-Disrupting Plastic Building Block. Three Maine women had blood levels of bisphenol A that were six to ten times higher than the average reported for women in the scientific literature. In five women of the 13 Mainers tested, we found a metabolite of a related chemical known as bisphenol A diglycidyl ether (BADGE). Bisphenol A is widely used and totally unregulated. BPA mimics the actions of naturally occurring hormones in the body, so exposure to very low doses may adversely affect reproduction, sexual development, and other biological systems. Early fetal exposure in the womb may predispose adults to breast cancer, obesity, and other chronic diseases. BPA is a basic building block chemical used to make polycarbonate plastics used in baby bottles, reusable water bottles, and many other products. BPA and BADGE exposure also results from the epoxy resins used in the plastic linings of canned foods and in dental sealants.

Toxic Metals -- the Age-Old Poisons. Every one of the 13 Mainers tested had measurable levels of lead, mercury, and arsenic in their bodies. Blood lead levels were generally below the national average, although no safe level may exist. Lead exposure results from renovating lead-painted houses, old drinking water pipes, and handling products containing lead, such as ammunition and electrical wire. The methylmercury levels measured in hair almost certainly resulted from consumption of mercury-contaminated fish, such as canned tuna and tuna sushi. Total arsenic levels were relatively high in a few of the Mainers tested, although this may be due to relatively non-toxic

forms of organic arsenic found in shellfish. Detections of the highly toxic inorganic form of arsenic probably resulted from natural drinking water contamination. Arsenic was formerly widely used as pesticide in pressure-treated wood and for orchards and along roadways.

THE CHEMICALS

Phthalates – Beauty Products and Beastly Vinyl

Phthalates (pronounced THAL-ates) are widely used industrial chemicals that are found everywhere. They are added to PVC plastic products to make them softer or more flexible, such as toys, car interiors, medical devices like blood IV bags and tubing, vinyl flooring, vinyl wallpaper, and vinyl shower curtains. Phthalates are also added to many cosmetics and personal care products including scented lotion, shampoo, perfume, aftershave, nail polish, and hair spray. Phthalates can make up a major portion of a product by weight, but since they are not chemically bound, the chemicals leach out over time.⁹ For example, a new vinyl shower curtain can elevate indoor air toxics concentrations for over a month.¹⁰

According to the federal Centers for Disease Control, phthalates are found in Americans of all ages, sizes, and races.¹¹ Phthalates are present in breast milk and can cross the placenta to enter a growing fetus. Humans are exposed by ingesting contaminated food and water, and to a lesser extent through inhalation and skin contact.¹² In one study, babies in neonatal intensive care units using phthalate-containing vinyl medical products had levels of phthalates seven times higher than babies in a hospital not using phthalate-containing products.¹³ Infants and children are especially vulnerable to phthalate exposure because they put plastic objects in their mouths.

Phthalates in Maine People

Phthalates were detected in all 13 participants, and those who reported using certain products had higher levels than others. Phthalates do not build up in the body (or bioaccumulate), so internal levels may fluctuate throughout the day reflecting recent or continuous exposure. We tested for seven phthalate monoesters, which are metabolites (breakdown products) of five phthalate diesters added to consumer products. (See Table 2 in the Appendix for complete results).

All seven of the phthalate compounds were detected in nearly every Maine person tested. The median (or middle) levels of six of the seven phthalates measured in the participants were greater than the national median (the middle value of more than 2,500 Americans randomly tested). For one phthalate, a metabolite of dimethyl phthalate (DMP), the median Maine value was higher than 95% of all Americans tested. DMP is used in hair sprays, insect repellants, and soft plastics. (A metabolite is the chemical that forms from the biological breakdown of the original chemical).

Three phthalates were found in Maine people at levels greater than 75% of all Americans tested. Two of these are metabolites of DEHP or di-(2-ethyl hexyl) phthalate. The levels of another DEHP byproduct were higher than the national median. DEHP is widely used in PVC (vinyl) products such as medical IV bags and tubes, auto interiors, diaper covers, shower curtains, and other consumer items.

A byproduct of benzylbutyl phthalate (BBzP) was the other phthalate found in Maine people at levels higher than 75% of all Americans. BBzP is added to vinyl flooring, car care products, personal care products, adhesives, and sealants.

In the bodies of the Mainers tested, levels of a phthalate found in nail polish and other personal care products, dibutyl phthalate (DBP), was found higher than the national median and approaching the 75th percentile level for all Americans, as indicated by DBP metabolite measured.

Figure 1 shows the sum total of the seven phthalate compounds measured in each of the Maine study participants. Six people had total phthalate levels that exceeded the national median for the same seven phthalate compounds. The data in Figure 1 are creatinine-corrected. That means they are normalized to the levels of a protein normally found in urine so that the results are not biased by dilution from drinking lots of fluids before the test.

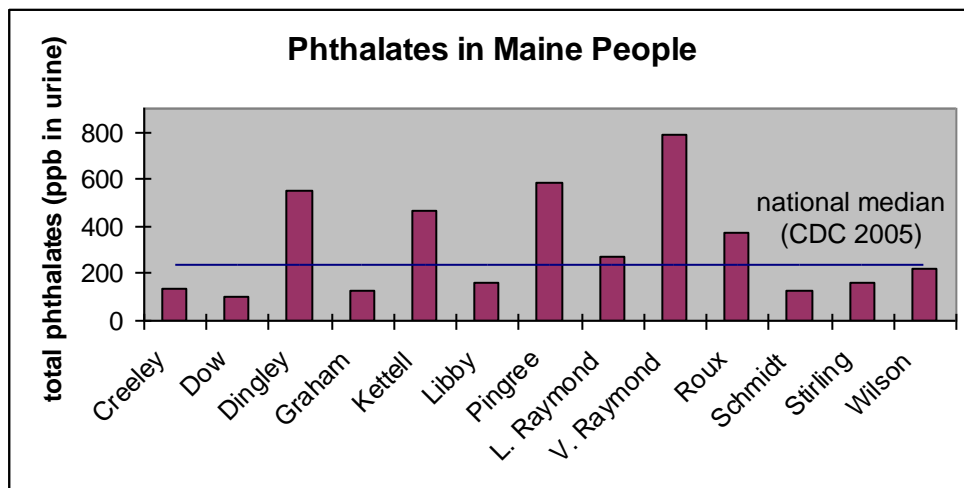


Figure 1: Phthalate monoester levels, measured in urine and creatinine-corrected

In our study, the seven women who reported using perfume at least once every three days had high levels of phthalates in their urine. Perfume and other scented products are known to contain phthalates. An independent testing of name-brand beauty products in 2002 found phthalates in 52 of 72 products, none of which listed phthalates as an ingredient, although all 17 products labeled with “fragrance” contained phthalates.¹⁴

Vi Raymond's total phthalate level was over twice the group median. She reported using perfume at least every three days. In contrast, Amy Graham, who had the lowest total phthalate level of the nine women tested, did not report using perfume or any products that are known to contain phthalates.

HEALTH EFFECTS: Male Reproductive Damage Tops Concerns

Phthalates are hormone-disrupting chemicals that threaten reproductive health, especially in males. In 2004, a scientist at University of Rochester found that baby boys whose mothers were exposed to high levels of phthalates during pregnancy were more likely to have altered genital development.¹⁵ Animal tests show that phthalate exposure leads to small or otherwise abnormal testes, hypospadias (abnormal urinary openings on the penis) and undescended testes in young males.¹⁶ Researchers believe that the phthalates that have these effects, such as DEHP and DBP, act by reducing levels of testosterone and important growth factors in young males. In adult males, phthalate exposure has been linked to lower sperm counts, reduced sperm motility, and damaged sperm.¹⁷

Other potential effects include reduced female fertility¹⁸ and premature breast development in young girls,¹⁹ liver and kidney damage and asthma.²⁰ EPA classifies the phthalate DEHP as a probable human carcinogen.²¹

Policy Changes Needed

Given widespread human exposure to phthalates and the threat of reproductive harm, government and industry action is needed to eliminate their use in PVC plastics and beauty products.

A large number of hospitals, consumer product companies, and government purchasers have taken first steps to replace PVC plastics containing phthalates with safer alternatives. Revlon, L’Oreal and other major companies are phasing out phthalates in nail polish, and 300 cosmetic companies have pledged to eliminate phthalates in their products in response to consumer demands from the Campaign for Safe Cosmetics.

In 2005, the European Union banned six phthalate softeners in PVC plastic toys that can be placed in children's mouths, following restrictions on three phthalates in toys imposed in 1999, and prohibited the use of some phthalates in cosmetics in 2003. Mexico, Japan, and Canada have also taken action on the chemicals.

In contrast, phthalates remain largely unregulated in the United States. The U.S. Food and Drug Administration (FDA) has failed to take action on cosmetic and medical uses of phthalates, citing a lack of compelling evidence that phthalates pose a safety risk.²² FDA has, however, encouraged medical providers to voluntarily switch to alternative products that do not contain phthalates. The City and County of San Francisco adopted an ordinance to restrict the use of phthalates in children’s products. Similar statewide legislation is under consideration in California, Maine, and other states.

Reducing Your Exposure to Phthalates

While market trends and personal actions by consumers are not likely to dramatically reduce phthalate exposure without coordinated policy action by state and federal governments, there are ways you can reduce your family’s exposure to phthalates.

Avoid PVC plastic. Unless made by a U.S. manufacturer who has indicated the product is phthalate-free, avoid soft plastic toys and soft vinyl products with a strong plastic smell such as plastic shower curtains. For information on PVC-free products for the home, office, and building materials, check out the resources available at: <http://www.preventharm.org/take.buyg.shtml#pvc>.

Purchase phthalate-free beauty products. Avoid nail polish, perfumes, colognes, and other scented products that are labeled as containing phthalates. Since many products simply list “fragrance” as an ingredient, avoid those products or do more research. For more information on phthalate-free cosmetics and personal care products, visit these Web sites: <http://www.safecosmetics.org>, by the national Campaign for Safe Cosmetics, and <http://www.ewg.org/issues/cosmetics/virtualdrugstore.php>, a database on cosmetic products and their ingredients by Environmental Working Group.

PBDEs – the Toxic Flame Retardants

Polybrominated diphenyl ethers (PBDEs), a major class of brominated flame retardants (BFRs), are added to plastics and synthetic fibers in TVs, computers and other plastic-encased electronics, mattresses, upholstered furniture, foam cushions, curtains, and hair dryers, to slow the spread of fire. Maine and others have recently banned two commercial PBDE products known as Penta and Octa, and is considering legislation to replace the most widely used PBDE mix called Deca with safer alternatives.

About 49 million pounds of Deca, or nearly half the world's production, was added to consumer products in North America in 2001. Deca can make up 10 to 15% of the plastic casing of a television and 18 to 27% of upholstery fabrics by weight.²³ Because PBDEs are not chemically bound to plastics, they leach out of the products over time. For example, older computers and automobiles can release PBDEs into the air.²⁴ When Deca leaches out of products, it is converted by sunlight into more toxic forms.²⁵

The alarm on PBDEs was sounded in 1998 when Swedish scientists first determined that these chemicals were increasing rapidly in human breast milk.²⁶ (Breastfeeding is still best – see Box.) Today, PBDEs are being found virtually everywhere scientists look—in indoor air and household dust,²⁷ in food,²⁸ breast milk²⁹ and umbilical cord blood.³⁰ Children and adults in the United States have 10 to 40 times more PBDEs in their bodies than people living in Europe³¹ or Japan,³² because the U.S. is the largest consumer of PBDE flame retardants in the world.³³

[BOX: Breastfeeding is Still the Best for Babies

While researchers have found PBDEs and other chemicals in breast milk, mothers should not be discouraged from breastfeeding. Breast milk is still the best food 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 and may increase exposure due to contaminants and leaching of chemicals from plastic baby bottles.

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>. *Adapted from Washington Toxics Coalition.*]

PBDEs in Maine People

Figure 2 shows that a wide range of PBDEs were detected in all 13 Maine participants, from 6.8 parts per billion (ppb) to 59 ppb. We found 28 different congeners of PBDEs out of the 46 that we tested for in blood. (Congeners are similar types of chemical compounds; there are 209 possible PBDE congeners). The PBDE levels found in Maine people were generally comparable or somewhat lower than those found in other studies. Two of the PBDEs that are breakdown products of Deca, known as BDE-153 and BDE-183, were higher in Maine participants than in similar small studies in Washington and California.

Bettie Kettell had the highest individual blood level of total PBDEs, at three times the group median. She works in Surgical Services in a recently constructed community hospital containing new rugs, drapes, and furniture. Her department has a large amount of equipment, including computers and monitors. This might explain her exposure levels since commercial buildings have higher fire safety standards—and thus more flame retardant-containing furniture and equipment—than private homes.

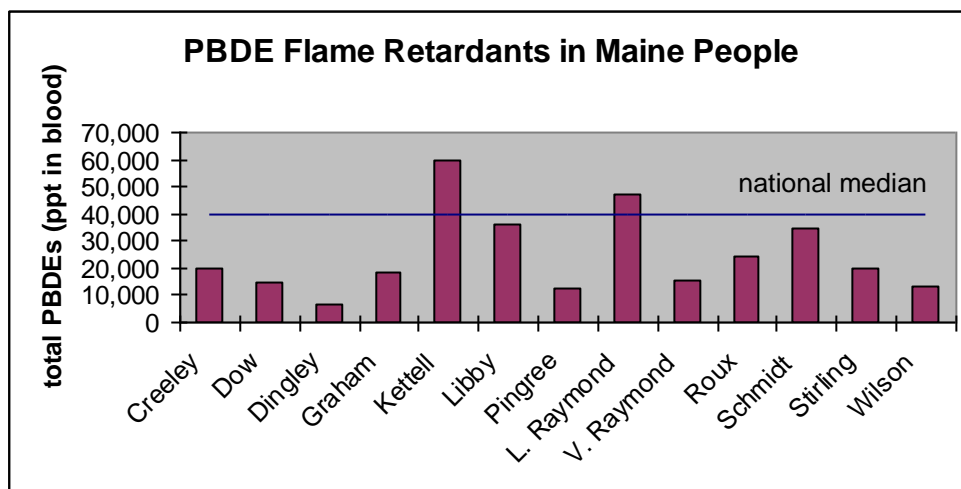


Figure 2: PBDE levels, measured in blood serum, expressed on a lipid weight basis. National median from McDonald (2005).

Two of the Maine participants, Bettie Kettell and Lauralee Raymond, had total PBDE levels higher than the median value reported in 62 women from California and Indiana. They also had the second and third greatest number of individual PBDE congeners detected, 26 and 25 respectively (out of 46 tested). Russell Libby, an organic farmer, had the most PBDEs detected (27) but his total levels were somewhat lower than levels in Bettie and Lauralee.

HEALTH EFFECTS: Learning Disabilities and Behavior Problems

Everyday exposure to PBDEs may be enough to cause children to struggle to keep up in school and reach their full potential. Most recently, for example, a study conducted at the University of Southern Maine found that newborn mice exposed to Deca had delays in brain development and reduced thyroid levels when young, and as an adult suffered from long-term learning and behavior problems.³⁴

PBDEs are chemically similar to PCBs, chemicals banned in the 1970s that damage brain function and cause a variety of other toxic effects. Research suggests that PBDE exposure affects thinking and learning abilities,³⁵ reproductive development,³⁶ liver tumors, and functioning of thyroid,³⁷ a hormone essential for brain development and healthy metabolism. Thyroid effects have been shown in wildlife.³⁸ EPA has also classified Deca as a possible human carcinogen, based on a valid two-year animal study.³⁹

Policy Changes Needed

The PBDE flame retardants can be completely replaced with safer alternatives. A number of leading electronics and furniture companies are making their products fire safe without the use of PBDEs. The Penta and Octa commercial mixtures of PBDEs have been banned in Maine and eight other U.S. states and throughout the European Union, and manufacturers have withdrawn them from production.

Government action is needed to phase out the use of Deca-BDE. In the European Union, the commercial Deca formulation can not be sold because it contains banned PBDEs and is therefore in violation of the Restriction on Hazardous Substances for electronics and electrical equipment, which went into effect on July 1, 2006. The temporary regulatory exemption granted to pure Deca has been challenged in the European Court of Justice by the European Parliament because the European Commission failed to consider the availability of safer alternatives.

Sweden has banned the use of Deca in textiles (furniture, mattresses, drapes, etc.) and for other uses not covered by European-wide directives on electronics and automobiles. Other European countries are considering a similar approach.

The use of Deca in electronic casing, mattresses, and home furniture will be prohibited in Washington state under a new law enacted in 2007. Legislation in Maine, LD 1658, will implement the goal adopted by law in 2004 to phase out similar uses of Deca in favor of safer alternatives. Deca phase-out legislation has also been introduced in California, Illinois, Michigan, Montana, Hawaii, Minnesota, and Massachusetts.

It is encouraging that PBDE levels in Swedish breast milk began to decrease after PBDE use was reduced. Following this lead, Maine law now prohibits the sale of products containing the highly toxic Penta and Octa PBDEs and requires the state to phase out sales of Deca if safer alternatives are available. Because flame retardants can reduce the risk of some household fires, finding safer flame retardants is an important step in eliminating PBDEs from the market.

A report by the University of Massachusetts concluded that non-halogenated alternatives to Deca-BDE (i.e., those not containing bromine or chlorine) are widely available, effective, and affordable for electronic enclosures (e.g., the plastic cases of TVs) and textiles.⁴⁰ The phosphate-based flame retardants such as RDP enable televisions to meet the highest fire safety standard without Deca, and are already used by leading TV and computer manufacturers. A recent independent analysis of three flame retardant chemicals concluded that RDP is safer than Deca, and is preferable as we search for even greener solutions that ensure fire safety and environmental public health protection.⁴¹

The Maine Department of Environmental Protection (DEP) recently noted in a report to the Legislature that safer alternatives are available for TV cabinets and textiles, the applications that consume most Deca, and that there are no significant costs or technological barriers preventing this change. The DEP recommends that the state ban the sale of televisions and other consumer electronics that have plastic casings containing Deca effective January 1, 2012, and should ban the sale of mattresses and upholstered furniture that contain Deca after January 1, 2008.⁴²

Reducing Your Exposure to PBDEs

You can take steps to reduce your family's exposure to PBDEs, including:

Buy PBDE-free furniture and electronics. Since products do not have to be labeled, it is difficult to know what individual items are free of PBDEs. Furniture without brominated flame retardants is available from IKEA and Herman Miller. Many of the leading electronics companies are using alternative flame retardant chemicals. For example, televisions made by Sony, Phillips, Panasonic/Matsushita, and Samsung are now all Deca-free. For further information, visit <http://www.safer-products.org>.

Consider housecleaning with a high efficiency vacuum. These are expensive but filter our dust much better than conventional vacuum cleaners. About 80% to 90% of PBDE exposure of Americans is thought to come from household dust contamination.⁴³

Reduce animal fats and avoid farmed salmon. Most PBDEs are fat-loving food chain contaminants. Choose lean cuts of meat and low-fat or non-fat dairy products. Choose wild salmon over farmed salmon since it is lower in PBDEs and other contaminants such as PCBs and dioxins.

PFCs -- The Stain-Resistant Teflon Chemicals

Perfluorinated chemicals (PFCs) are synthetic chemicals designed to repel grease and water. Used since the 1950s in a wide range of consumer products, PFCs have been used more recently as stain-resistant coatings such as Scotchgard and Stainmaster for carpets, couches, and other upholstered furniture and automobile seats; to make water-repellent fabrics like Gore-Tex, and now in popular clothing lines like Gap Kids, Dockers, and Levis.⁴⁴ They are also used to make Teflon coatings for non-stick cookware and grease-resistant food packaging and paper products (food wrap, microwave popcorn bags, French fry boxes, candy wrappers, etc.). Personal care products including makeup, moisturizers, and dental floss may also contain PFCs.

The most widely used and studied among the many different PFCs are the chemicals known as PFOS (perfluorooctane sulfonate) and PFOA (perfluorooctanoic acid). The use of these two chemicals has led to widespread contamination of people and the environment. Perfluorinated chemicals are extremely persistent. PFOA, which is used to make Teflon and is a breakdown product of stain- and grease-proof coatings, does not degrade at all. It has a half-life in the human body of more than four years. Other PFCs break down and turn into PFOA. PFOS, which was in the Scotchgard formulation until 2000, has a half-life of more than eight years. Exposure appears to be continually renewed through daily contact.

Humans are exposed through contaminated water and food, including fish,⁴⁵ and by breathing contaminated air.⁴⁶ When Teflon pans are heated, such as during cooking or when discarded products are burned in incinerators, toxic greenhouse gases are produced.⁴⁷ The PFCs build up in the bloodstream and liver,⁴⁸ umbilical cord blood,⁴⁹ and breast milk.⁵⁰ In a 2001 study by 3M company, PFOA were found in 96% of 598 children tested in 23 states and the District of Columbia.⁵¹ 3M discontinued production of PFOS in 2001, though other PFCs may degrade into PFOS over time.

PFCs in Maine People

Figure 3 shows that all of the Maine participants were found to have perfluorinated chemicals in their blood. We found six different perfluorinated chemicals of the 13 PFCs that we tested. PFOS and PFOA were the only PFCs that were detected in every Mainer. See Table 2 in the Appendix for complete results.

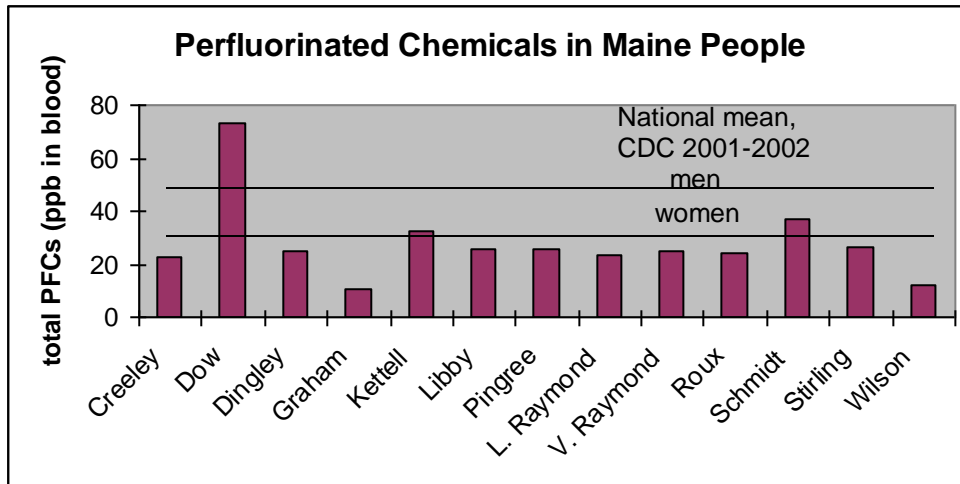


Figure 3: Total PFC levels, measured in blood serum.

PFOS was detected at the highest level among Maine participants, with a median value of 14.2 ppb. This is lower than the mean estimate for PFOS in a national study of more than 900 people tested in 2001 and 2002. This could reflect a decline in PFOS exposure since production and its use in Scotchgard ceased in 2001. See Table 3 for comparisons.

The median PFOA level among the tested Mainers falls within the national average range for men and women. Three of the other PFCs related to PFOA were found at levels above the average or median levels reported in other studies, while PFOA levels were about average.

State Senator Dana Dow had the highest total PFC level, which was over three times the group median, and the greatest number of PFCs detected—six compounds. Senator Dow’s levels were more than twice the national average levels for PFOA and related compounds, and for PFHxS (used in carpet treatment). Dana owns a furniture store, and he says that in the past he would spray some furniture in his store with PFC-containing stain-repellent products. He also says he often has new furniture in his home.

HEALTH EFFECTS: Persistent Chemicals Pose Multiple Health Risks

These stain-resistant, non-stick chemicals have been around for decades but have been under scrutiny only recently, and there are few studies of whether low doses of PFCs cause health effects in people. A study of men who had worked in jobs where they were exposed to PFOS found a high rate of deaths due to bladder cancer.⁵² Laboratory animal studies show that PFOA and PFOS damage the liver and other organs, cause immune disruption, endocrine effects, reproductive harm, and developmental defects.⁵³ However, unlike humans, laboratory rats rid their bodies of the chemical in days rather than years.

In response to evidence that PFOA causes testicular, pancreatic, mammary and liver tumors in rats, and increases worker risks of cancer, in January 2006 an expert panel of the Environmental Protection Agency (EPA) upgraded PFOA to a “likely human carcinogen.” They described the chemical as an indestructible toxic chemical group that pollutes nearly every American’s blood. The panel urged the EPA to adopt stricter guidelines to protect human health.⁵⁴

Policy Changes Needed

Under pressure from the EPA, the 3M Company halted production of PFOS-containing products in 2001, reformulating their Scotchgard product to minimize the release of PFCs into the environment. However, non-U.S. producers continue to manufacture PFOS. In 2006, the EPA signed a non-binding voluntary agreement with DuPont, 3M, and six other chemical companies to reduce PFOA from emissions and product content by 95% by 2010, with the ultimate goal of total elimination by 2015. PFOA and related chemicals are still used to manufacture Teflon and Gore-Tex.

While these steps represent tremendous progress, they will not by themselves fully protect public health and the environment from PFCs. Scientific evidence reveals that a wide variety of PFCs that remain in use break down over time into both PFOA and other persistent PFCs. For example, EPA cites a growing body of data indicating that PFCs known as fluorotelomer alcohols degrade or breakdown into PFOA.⁵⁵

Government should review all remaining PFCs and take action to replace any found to be persistent or that break down into persistent PFCs. Health and safety testing should be required for all PFCs and biomonitoring expanded to determine the extent of human exposure.

Reducing Your Exposure to PFCs

To reduce personal exposure, which has not been well studied, avoid purchasing or at least minimize use of products containing PFCs. Consider these tips:

Reduce greasy packaged foods and fast foods in your diet. The packaging for food like microwave popcorn, French fries, and pizza are often treated with grease-resistant coatings.

Avoid stain-resistant furniture and carpets. Decline optional treatments and ask for products that have not been pretreated.

Avoid Teflon or non-stick cookware. If you choose to use non-stick cookware, do not overheat or burn pans, as chemicals can be released when they reach 450°F,⁵⁶ and discard pans when they get scratched. The fumes from overheated Teflon are deadly to pet birds.

Choose alternatives to clothing with Teflon labels or treated for water or stain-resistance. Many of the treated outerwear and gear are coated with PFCs.

Look out for personal care products. PFCs are added to some cosmetics (nail polish, moisturizers, and eye makeup), shaving cream, and dental floss. Avoid those that have ingredients that include the words “fluoro” or “perfluoro.”

Bisphenol A -- the Hormone-Disrupting Plastic Building Block

Bisphenol A (or BPA) is a high-volume industrial chemical used as a monomer (or chemical backbone) to make polycarbonate plastic, which is widely used in reusable water bottles, baby bottles, pacifiers, plastic utensils, children's toys, compact discs, and certain microwaveable and reusable plastic containers. BPA is also used in some dyes, enamels, varnishes, flooring, adhesive, fungicides, antioxidants, dental sealants and artificial teeth. A chemical derivative of BPA called bisphenol A diglycidyl ether (BADGE) is used to make epoxy resins which are widely used in many applications. Human exposure to bisphenol A (and BADGE) results from the use of BADGE in the clear lining of metal food and drink cans, and from some dental sealants and composite dental fillings.

Over time, bisphenol A migrates from cans into food⁵⁷ and leaches from polycarbonate plastic bottles, especially when the plastic is heated or as it ages.⁵⁸ As evidence of the chemical's "leaky" nature, BPA has been found in 40% of stream water samples surveyed by the U.S. Geological Survey.⁵⁹ Humans are exposed through ingesting contaminated food, liquids and breast milk, and during some dental procedures.

Bisphenol A in Maine People

We tested the blood of our Maine participants for both bisphenol A and for a metabolite of BADGE known as BADGE-4OH which forms in the body. Since BPA and BADGE are not persistent in the body, detection reflects recent exposure.

Figure 4 shows that bisphenol A was found in three women of the Mainers tested, at levels ranging from 3.75 to 6.64 parts per billion. These results range from six to ten times greater than the average blood levels of BPA reported in one study of 14 women published in the scientific literature, and were also higher than the median and average in two other small studies (see Table 3 in the Appendix).

The related BADGE metabolite was found in five of the Maine participants, including the same three people with detectable levels of BPA in their blood. The two highest of the five reported Maine levels for BADGE-4OH (59.7 ppb and 119 ppb) were more than five to ten times greater than the geometric mean level of about 9.33 parts per billion resulting from one other small study (see Table 3).

Vi Raymond and Elise Roux had the highest levels of bisphenol A and BADGE exposure. Their results were significantly higher than the average levels reported elsewhere. We cannot explain their elevated levels of bisphenol A and its related compound based on their exposure surveys. The fact that these chemicals have been used in a multitude of products makes it difficult to determine the source of exposure.

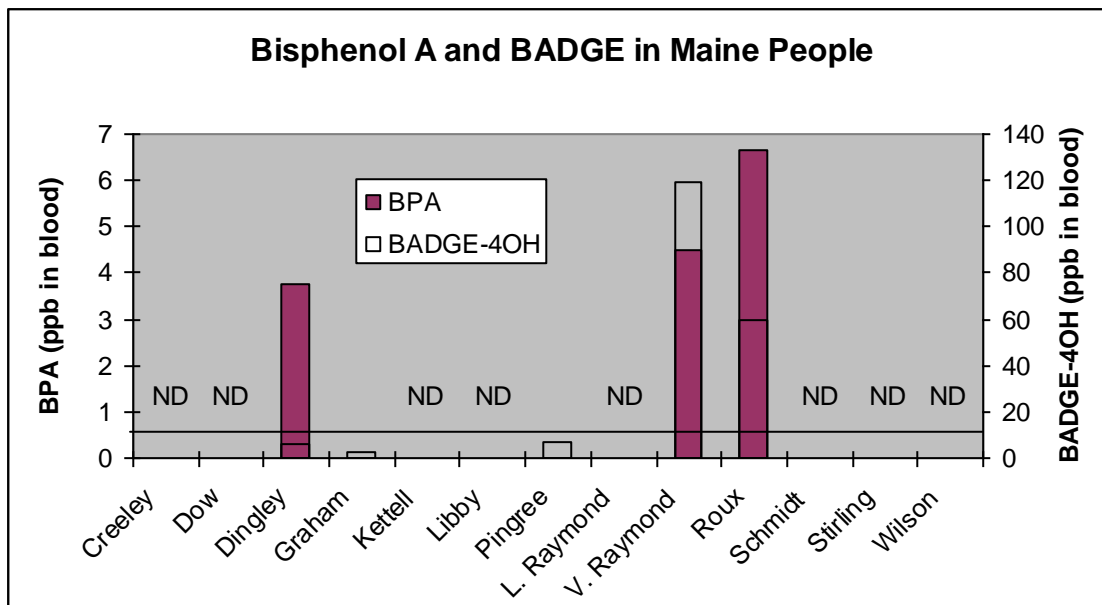


Figure 4: Bisphenol A and BADGE levels, measured in blood serum. The horizontal line is the mean BPA level for women (Takeuchi, 2002). ND = none detected.

HEALTH EFFECTS: Ultra Low-Dose Hormone Disruptor

Bisphenol A is a potent endocrine disrupting chemical in lab animals at very low doses⁶⁰ that is suspected of causing reproductive damage⁶¹ and birth defects⁶² that may lead to prostate⁶³ and breast cancer.⁶⁴ Studies have found that BPA can have adverse health effects at levels thousands of times lower than what the EPA considers safe. According to the low dose hypothesis, small and repeated exposures to bisphenol A can have an amplified effect on the human body by mimicking human sex hormones, or promoting cell proliferation.⁶⁵ Bisphenol A has been found to cause estrogenic changes in animal cells at the same concentrations that are found in pregnant women and their fetuses.

Controversy over toxicity exists between public health advocates and the plastics industry, which describes bisphenol A as a weak estrogen, and says there is little concern with human exposure levels. Between 1998 and 2005, 115 studies of BPA were published. None of the 11 studies funded by industry reported adverse effects at low level exposures, whereas 94 of 104 government-funded studies found statistically significant effects on animals. Adverse effects were found at levels to which many people in the U.S. are currently exposed, levels much lower than the EPA's current acceptable level.⁶⁶

Much less is known about the risks of exposure to BADGE. Environmental Working Group cites research suggesting strong evidence of hormone activity with limited evidence of other health concerns. They also cite a study showing that in the human body, BADGE can break down into BPA, which raises concerns about the compound's toxicity.⁶⁷

Policy Changes Needed

Growing scientific evidence on the health effects of very low doses of bisphenol A merits a much more protective Reference Dose (like a safety standard) than currently supported by U.S. EPA. It will be necessary to further reduce public exposure to bisphenol A.

The City and County of San Francisco banned the manufacture, sale, and distribution of child care articles and toys containing bisphenol A and some phthalates for children under three years old as of December 1, 2006.⁶⁸ Under the ordinance, San Francisco manufacturers of baby bottles, pacifiers, and toys for young children must replace BPA and phthalates with the least toxic alternatives. A similar measure was introduced in the California Legislature in 2006 but failed to pass. Similar legislation is pending in several states including Maine. All of these policy initiatives have been aggressively challenged by the chemical industry.

A similar concerted effort is needed by government and product manufacturers to switch to safer substitutes for uses of polycarbonate plastic and epoxy resins that may expose the fetus and young children to bisphenol A and BADGE.

Reducing Your Exposure to BPA

Bisphenol A has been used as an ingredient in consumer products for a long time, and is difficult to avoid. In some cases, alternatives are available.⁶⁹ Consider these tips, especially if you are or may become pregnant or are parents choosing for a child:

Avoid reusable plastic water and baby bottles. Most Nalgene reusable water bottles are made of polycarbonate plastic that leaches bisphenol A into the water. Use polyethylene or aluminum bottles instead. Use glass baby bottles instead of plastic. Discard old or damaged bottles.

Avoid polycarbonate plastic food containers and table ware. These may be labeled 'PC' underneath a plastic code #7 in the recycling triangle on the bottom of the container. (The #7 means 'other', so you need to see the 'PC' to confirm that the plastic is polycarbonate).

Minimize the use of canned foods and canned drinks. Until industry reformulates the laquer lining of metal cans (as is being done in Japan), choose fresh or frozen foods or glass containers or bottles. A recent study by Environmental Working Group found bisphenol A in more than half of 97 cans of brand-name fruit, vegetables, soda, and other common canned goods.⁷⁰

Ask your dentist for BPA-free sealants and composite fillings. Some dental resins are free from or low in BPA and BADGE. Ask your dentist if they know about BPA and request the MSDS sheet (Material Safety Data Sheet) for the sealants or composite fillings to look for BADGE in the list of ingredients. Make sure your family brushes and flosses regularly to prevent the need for dental work!

METALS -- The Age-Old Poisons

Lead

Lead is a natural metallic element that occurs in rocks and soils and has been put to industrial use for a few thousand years. Lead has been used in metal alloys, paint, batteries, solders, ceramic glazes, bullets, metal toys, and building materials. Currently, most lead exposure comes from old lead paint dust from Maine homes built before 1978. Sanding or burning old paint during renovations dramatically increases exposure, which also results from normal wear and tear around lead-painted window frames and doorways. Water pipes in some older homes may contain lead solder that can leach out into the water. Other sources of lead exposure include making and firing ammunition, handling lead-containing plastics, metal products, and lead acid batteries. Some cosmetics and folk remedies have been found to contain lead. Many PVC (vinyl plastic) products can contain lead, including electrical wires and cables, mini-blinds and children's lunch boxes. Handling these products can result in lead exposure. Lead can also be released to the environment from disposal of PVC products, television sets, and older computer monitors in landfills and incinerators.

Exposure to lead can occur from breathing contaminated air or dust, eating contaminated foods, or drinking contaminated water. Children can be exposed from eating lead-based paint dust or playing in contaminated soil. Children from low-income families or who live in older homes are especially at risk.

Lead in Maine People

Lead was measured in the blood of twelve of the Maine participants (one sample was lost). The Maine results were generally below or near the median (or middle value) of the blood lead results for nearly 9,000 randomly selected Americans tested by the federal Centers for Disease Control and Prevention (CDC).

The blood lead level tends to indicate an exposure that occurred during the previous three to five months. However, these results could reflect recent exposures to lead or residual exposure from cumulative exposure to lead over a lifetime.

Lead bioaccumulates in bone as a result of exposure to multiple sources over time. Lead in the blood is taken up by the bones or is very slowly excreted, so once exposure to lead stops, the blood lead level will decrease gradually over the following months. Adults who were exposed to high levels of lead as children when leaded gasoline and paint were in widespread use still have lead stored in their bones. Bone lead leaches out over a period of years or less during pregnancy and in conditions of high bone turnover such as osteoporosis. This lead leaching from bone may be a factor in the lead levels seen in this survey.

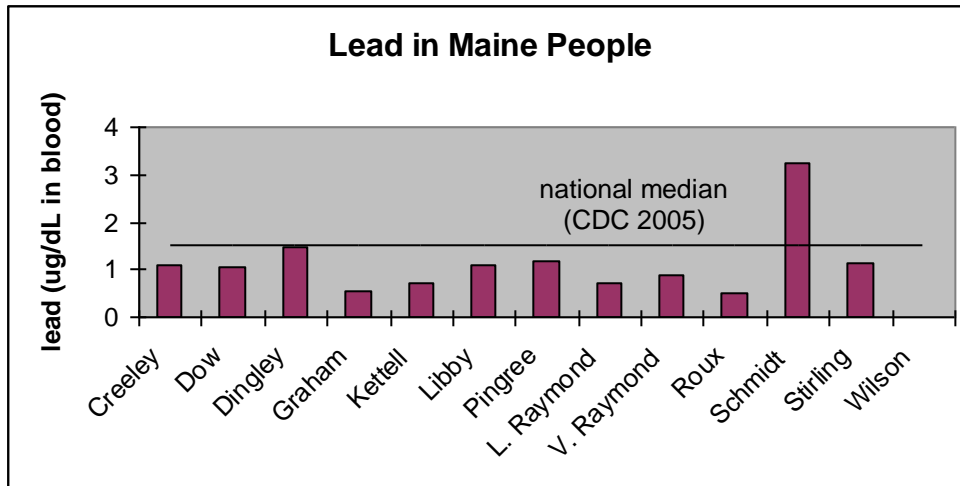


Figure 5: Lead levels, measured in whole blood.

HEALTH EFFECTS: Lowered Intelligence and Lifelong Health Threats

The toxic nature of lead has been well known for hundred of years, with childhood lead poisoning from lead paint first documented a century ago. Yet adults and especially children continue to be exposed to this dangerous metal. The toxic effects of lead are well documented in both children and adults.

Lead causes damaging health effects at extremely low doses, and the main target is the central nervous system. Recent research shows that accumulated bone lead also leaches out faster during pregnancy and breastfeeding, exposing the fetus and infant to higher lead levels. Proven harmful effects include impaired brain development, premature births, smaller babies, learning difficulties, and reduced growth in young children. Children exposed to lead at a young age are more likely to suffer from shorter attentions spans and are less able to read and learn than their peers.⁷¹ Studies in children show that reducing blood lead levels by 10 ug/dL significantly raises the IQ by an average of 2.6 points, which across a large population has a huge effect.⁷²

Lead in adult bones leaches out over years and is one of the risks for hypertension. Long-term exposure of adults can result in decreased mental performance, or cause weakness in fingers, wrists, or ankles. Besides affecting the brain, lead exposure also causes kidney damage, anemia, increased blood pressure in older adults, and ultimately death. High-level exposure in men can damage the organs responsible for sperm production, and in pregnant women may cause miscarriage.⁷³ Workers in construction, police protection, and other industries are at especially high risk of adult lead poisoning.⁷⁴

The federal government has concluded that lead and lead compounds are “reasonably anticipated to be human carcinogens,” due to increased lung and stomach cancer in humans and cancer of the kidney, brain and lung in lab animals.⁷⁵

Policy Changes Needed

Had the warnings of public health scientists been heeded in the 1920s, leaded gasoline would have never entered the marketplace and the use of leaded paint would have ended 50 years earlier in the

United States.⁷⁶ In a triumph of politics and profits over public health, an epidemic of lead poisoning ensued through the twentieth century, resulting in many deaths and disabilities in both children and adults. Although eliminating the use of lead in gasoline and paint by the late 1970s also marked one of the great public health successes of the last century, the toxic legacy of low-level lead poisoning continues to this day.

Average blood lead levels in American children have dropped by 85% since unleaded gasoline was first introduced in 1979, when the average U.S. child lead level was 16 ug/dL. Still, the U.S. Centers for Disease Control and Prevention estimates that more than 300,000 American children remain at risk of being exposed to harmful lead levels.⁷⁷ And the latest science shows toxic effects on childhood brain development at levels much lower than the current federal action level of 10 ug/dL of lead in blood, suggesting that there is no safe level of exposure to lead.⁷⁸

Therefore, policy action is needed to eliminate current exposures to lead and remaining uses of lead in commerce. Maine has begun to make recent progress to further reduce lead. Legislation enacted in 2005 creates a lead-poisoning prevention fund for education and outreach programs aimed at reducing lead exposure. And, as of January 1, 2006, cathode ray tubes from computer monitors and televisions, which can contain four to eight pounds of lead, can no longer be thrown in Maine landfills and incinerators. Tubes must be collected and recycled instead.

Much more is needed. Maine should follow the lead of Rhode Island's successful lawsuit to force lead pigment manufacturers to pay the enormous costs of lead cleanup at the 240,000 homes in the state that still have lead paint. In the interim, all renovations of lead-painted houses should be required to use lead-smart procedures to minimize lead exposures. Unnecessary uses of lead in wheel weights, PVC plastic, ammunition, and other sources should be phased out by law in favor of safer alternatives.

Reducing Your Exposure to Lead

Test for Lead. Children should be tested for lead at age one and two years, and at under six years of age if they have never been tested. Test soil within 20 feet of your house for lead before growing edible plants. If you have an older home (pre-1978), test your painted surfaces and drinking water for lead.

Make you home lead-safe. In a lead-painted home, consider replacing window frames and door frames which are a constant source of lead dust. All renovations should be done using lead-safe methods preferably by a certified contractor and at a time when young children or pregnant women are not living in the home. Do not allow children to chew or mouth painted surfaces. If you believe your home contains lead-based paint, clean the house regularly of dust and tracked in dirt and wash children's hands and faces often to remove lead dust. If you have a water lead problem, run water that has been standing overnight before drinking or cooking with it.

Avoid lead-containing products. Some types of paints and pigments used in makeup or hair coloring contain lead. Keep these away from children. Do not use imported glazed ceramic containers for eating or drinking. Avoid PVC plastic products. Watch out for cheap jewelry and kids toys that may contain lead. For more information on avoiding lead hazards, see resources at <http://www.preventharm.org/take.redu.shtml#aroundthehome>.

Mercury

Like lead, mercury is another natural metal that has significantly increased in our environment as a result of human activities. Most of the mercury in Maine comes from emissions from coal-fired power plants both near and far. Sources of mercury within the state include trash waste incinerators, wood and fuel oil boilers, the breakage and disposal of mercury-containing products such as fluorescent light bulbs (including compact fluorescents), thermostats and thermometers, and dental amalgam fillings. Mercury is released into the air, where it drifts with prevailing winds and falls out of the sky in dust, rain, and snow, and settles onto land or the ocean. Once on land, mercury is washed into streams and lakes, where it is converted to its more toxic and available form, methylmercury, and builds up in the food chain.

We are exposed to mercury mainly by eating contaminated fish, especially tuna but also shark, swordfish, tilefish, and king mackerel, which have the highest concentrations of mercury. Elemental mercury can also be directly inhaled from broken thermometers, dental fillings, and fluorescent bulbs.

Mercury in Maine People

Mercury was detected in the hair of all 13 Maine participants, indicating exposure to mercury over the past four to six months. Fish consumption is the largest source of individual mercury exposure, and this fact may shed some light on the study results.

Mercury levels in these Mainers (median 396 ppb) were generally higher than the national median (200 ppb) reported for 702 women of childbearing age (see Figure 6). Every one of the five Maine women of childbearing age had mercury hair levels greater than the national median level (see Tables 2 and 3).

The amount of methylmercury measured in the hair of House Majority Leader Hannah Pingree was above the U.S. EPA-established reference dose for mercury, which is the level above which the developing brain of the fetus may be harmed due to maternal mercury exposure. Hannah Pingree, Elise Roux, and Lauralee Raymond all reported eating tuna in sushi or swordfish at least twice a month, which may explain their elevated mercury levels.

Eric Stirling had the highest mercury level in the group, a level much greater than the national median. Eric reported that he commonly ate fresh-caught brook trout. While brook trout are not usually considered to be high in mercury, it is possible that Eric's lifestyle as a sporting camp owner means he eats more local, freshwater fish than the average Mainer. It's also possible that Eric fishes in streams or ponds that are particularly polluted with mercury, but such analysis was not part of this study.

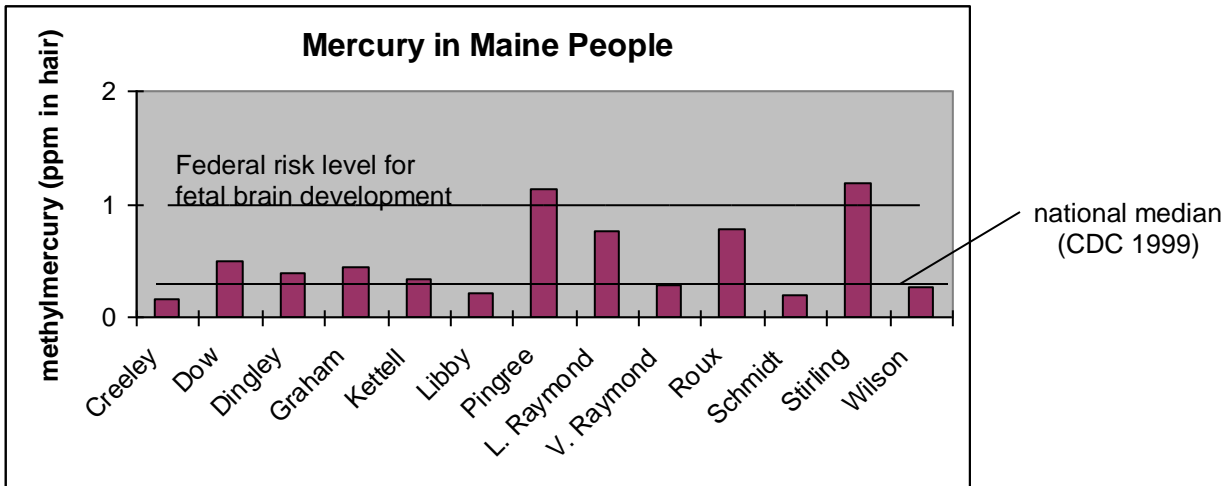


Figure 6: Methylmercury levels measured in hair

HEALTH EFFECTS

Mercury is a potent neurotoxin. Responsible for “mad hatter” syndrome, mercury can cause birth defects and brain damage, learning disabilities, loss of vision and blindness, kidney damage, numbness, and lack of coordination. The developing brain of babies in the womb, nursing infants and children are especially vulnerable to mercury. The adverse effects of prenatal mercury exposure, even at low levels, include deficits in memory, attention span, motor control, and the ability to learn.⁷⁹ According to the EPA, eight percent of women of childbearing age in the U.S. have elevated blood mercury levels of concern for fetal development.⁸⁰ The EPA estimates that 630,000 infants born each year in the United States are at risk for neurological damage from exposures to methylmercury.⁸¹

Policy Changes Needed

The policy goal to virtually eliminate mercury from manmade sources was adopted by the New England Governors and Eastern Canadian Premiers in 1998. Since then a number of medical waste incinerators and trash combustors have been closed and many of those remaining have upgraded air pollution controls. Maine has also banned the sale of many mercury-containing products including blood pressure monitors, thermostats, and electrical switches; and required that mercury products be labeled and recycled at the end of their useful life.

Aggressive actions are needed to reduce mercury emissions from coal-burning power plants and to reduce reliance on coal and fuel oil in favor of low- or no-mercury power and heat sources. More efforts are needed to warn people of hazardous levels of mercury in swordfish, canned and raw tuna, and other fish that are higher in mercury or consumed in large quantities. Restaurants and grocery stores should be required to advise women who may become pregnant and young children to avoid fish high in mercury.

Manufacturer responsibility to pay incentives for mercury product recycling need to be extended beyond old automobile switches and thermostats to include fluorescent light bulbs and other

mercury products. Efforts to replace mercury-containing fluorescent lighting with even more energy efficient digital lighting should be accelerated.

Reducing Your Exposure to Mercury

Eat fish that are low in mercury. Nearly all fish and shellfish contain traces of mercury, but some have more mercury than others. Women who are or may become pregnant should avoid eating swordfish and tuna (canned, steaks or in sushi). (See Maine’s fish consumption advisory and visit <http://www.preventharm.org/take.redu.shtml#eatingfish>).

Recycle old mercury products. Contact your town about the hazardous waste collection schedule for recycling fluorescent light bulbs, thermostats, and other mercury-containing products. Carefully handle and thermometers, fluorescent light bulbs and other mercury products to avoid breakage prior to recycling. Do not vacuum up spilled mercury, because it will vaporize and your exposure will increase drastically.

Support mercury-free dentistry. Ask for composite fillings instead of “silver” fillings or amalgam, which are about 50% mercury. If you have metal amalgam fillings, consider having them replaced with composite fillings.

Demand that utilities slash mercury from coal-burning. Support Maine legislative efforts in conjunction with other New England states to force the federal government to reduce mercury emitted by coal fired power plants, as required by the Clean Air Act.

[BOX : Fish Consumption Advisory

All of Maine's lakes, ponds, and rivers are under a fish consumption advisory due to mercury pollution. According to the Maine Center for Disease Control and Prevention, women who are pregnant, nursing, or may become pregnant, and children under age 8 should eat no freshwater fish from Maine's inland waters except for one meal per month of brook trout or landlocked salmon. All other adults and children older than 8 can eat one to two fish per month from Maine lakes and rivers.⁸²

The State also advises that pregnant and nursing women, women who may get pregnant, and children under age 8 limit their consumption of canned light tuna to 2 meals per week or white tuna to 1 meal per week; all other adults and children age 8 and older can eat 2 meals per week of canned tuna.⁸³

Other public health advocates suggest that pregnant and nursing women should avoid all canned tuna (and tuna in sushi) because the advice above will result in mercury exposures to the fetus that exceed the federal reference dose or safety level established to protect the children’s health from neurotoxicity.⁸⁴]

Arsenic

Arsenic is a common element in the earth's crust, occurring naturally in soil and bedrock. Many people in Maine are exposed to arsenic by drinking contaminated water from bedrock wells. In eastern New England, 20-30% of private wells exceed the arsenic drinking water standard of 10 micrograms per liter.⁸⁵ Arsenic has also been used in pressure-treated lumber, is still used in industrial applications, and can still be found in decks, playgrounds, and other structures. Arsenic was used as a pesticide in Maine between 1920 and the late 1960s, and high arsenic levels can still be found in areas of Maine where arsenic-containing pesticides were applied to apple orchards, potato and blueberry fields, and along roadways.

Arsenic in Maine People

The Maine participants exhibited a wide range of arsenic levels in urine. The median level of total arsenic in Mainers (30.7 ppb) was about three times higher than the median level reported in a study published in the scientific literature (see Table 3). This might be due to relatively greater reliance in Maine on drinking water wells contaminated with highly toxic inorganic arsenic, or could represent recent consumption of seafood, which contains an essentially nontoxic, organic form of arsenic. (See Box on Arsenic's Complex Chemistry).

Five of the Mainers exhibited above normal total arsenic exposure levels of greater than 50 ppb in urine (see Figure 7 and Table 2). One of the participants approached the total arsenic exposure level considered excessive (without consumption of seafood) at 100 ppb in urine.

Another participant, Regina Creeley, had the highest level of total arsenic at 839 ppb. Regina reported that she ate a large meal of mussels prior to her test. Certain seafood including shellfish contributes large amounts of a non-toxic organic form of arsenic to a total arsenic measurement in urine. (See discussion in Box).

[BOX – Arsenic's Complex Chemistry

The arsenic story is complicated. We measured total arsenic, which includes at least five different types of arsenic: the two most toxic forms of inorganic arsenic – arsenic(III) and arsenic(V); two somewhat less toxic, organic forms of arsenic formed in the body after exposure to inorganic arsenic, monomethylarsinic acid (MMA) and dimethylarsinic acid (DMA); and arsenobetaine – the nontoxic form of organic arsenic found in shellfish and some other seafood. We also measured total inorganic arsenic, arsenic(III) plus arsenic(V), and we separately tested for the most toxic form of inorganic arsenic, arsenic(III). We did not measure for MMA, DMA, or arsenobetaine.

When people are exposed to highly toxic inorganic arsenic (from well water or old pressure treated wood, for example), that arsenic is metabolized in the body. In just a few days, this biologically transformed arsenic is mostly excreted in urine as, on average, 10 to 30 percent inorganic arsenic As(III) and As(V), 10 to 20 percent MMA and 55 to 76 percent DMA.⁸⁶ [Note: As(III) is the abbreviation for arsenic(III) and As(V) is short for arsenic(V)].

This complex biochemistry and the limits of our testing mean that our results underestimate exposure to toxic inorganic arsenic by a factor of three-fold to ten-fold. And, we can't tell how

much of the exposure to organic arsenic, which is high for several Mainers, is due to MMA and DMA (reflecting exposure to toxic inorganic arsenic) and how much is due to the nontoxic organic arsenic from seafood, arsenobetaine, which is passed through the body without being absorbed.]

While three participants obtained more than 90% of their drinking water from a private well, this was too small a group to show a relationship between urinary arsenic levels and well water consumption by geographic area, and participant wells were not tested for this project.

Denyse Wilson had the highest inorganic arsenic level in the group, and her total arsenic level was also above the normal exposure level. Denyse does not use well water, but she does eat herbs and vegetables from a raised-bed garden built in the spring of 2006 using purchased soil and older pressure-treated wood. Follow-up testing of arsenic in the soil and municipal water supply is pending. Lauralee Raymond also had a relatively higher inorganic arsenic and total arsenic result.

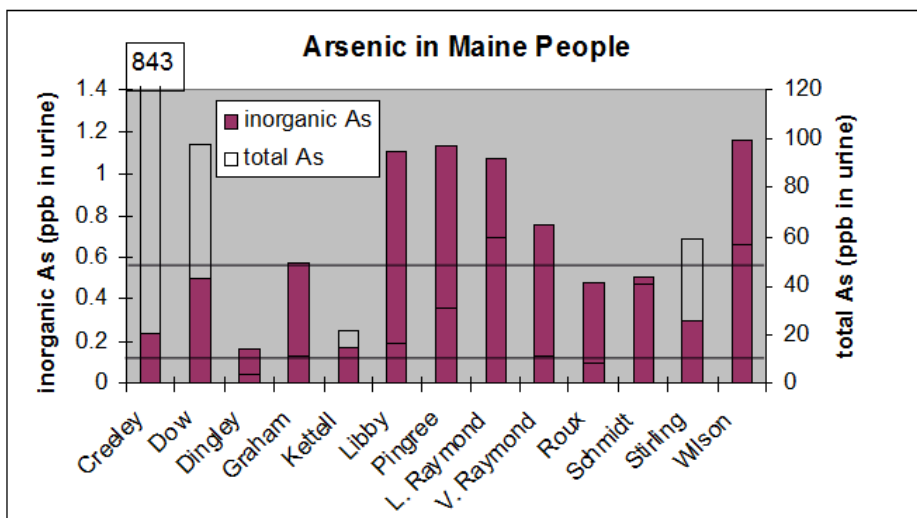


Figure 7: Total arsenic and inorganic arsenic levels, measured in urine. The top horizontal line is the above normal exposure of total As (50 ppb; Klaassen 2001); the bottom line is the national median for total As (10.23 ppb).

HEALTH EFFECTS: Potent Cancer-Causing Agent

Long-term exposure to low levels of arsenic in drinking water has been linked to bladder, lung, kidney, liver, prostate, and skin cancer. The higher the level of arsenic in the water, the greater the cancer risk, and cigarette smokers who drink arsenic contaminated water are the most at risk.⁸⁷ Maine has one of the highest rates of bladder cancer in the United States. Arsenic can also harm the nervous system, heart and blood vessels. EPA classifies arsenic as a human carcinogen.⁸⁸ Because their bodies are less efficient at processing arsenic, children may be more susceptible. Arsenic can cross the placenta and has been found in fetal tissues. Research suggests long-term exposure to arsenic in children may result in lower IQ scores.⁸⁹

Policy Changes Needed

Maine needs a law to require routine testing of private well water for arsenic, especially when a home is sold or rented. The prevalence of arsenic in Maine groundwater, the potent carcinogenicity of arsenic and the unacceptably low level of public awareness of the arsenic threat, make this an urgent public health issue.

Publicly accessible institutions with old pressure-treated wood structures, such as decking, railings and playground structures, should be required to replace them with arsenic-free construction. If replacement is not economically feasible, then the arsenic-treated wood should be coated with a sealant on an annual basis to reduce arsenic exposure on contact with the wood.

Reducing Your Exposure to Arsenic

Test your well water. Well water is a major source of arsenic exposure in Maine. Arsenic-contaminated drinking water does not smell, taste, or look different; the only way to find out if your well has arsenic is to have your water tested by a certified laboratory. The longer water sits in a well, the more time there is for arsenic to dissolve into drinking water, so wells used by seasonal homes and camps may have higher levels of arsenic. If your water comes from a public supply, contact your local water district for the most recent arsenic test results. Treatment (or even drilling a new well) is required to remove arsenic from drinking water. As of January 2006, the maximum amount of arsenic allowed in public drinking water systems is 10 parts per billion (0.010 mg/L), a level probably not sufficiently protective of public health.

Replace old pressure-treated wood structures. Maine was among the first states to ban the sale of arsenic-treated wood used in playgrounds and decks in 2003, around the time that a national voluntary ban was negotiated. If you can afford it, remove and replace pressure-treated wood structures installed before 2004. These include boards used for vegetable gardens, playgrounds, decks and railings. Avoid sawing pressure-treated wood. Paint or seal all arsenic-treated wood with a penetrating oil deck treatment. To learn more about what to do if you have pressure treated wood at home, visit <http://www.healthybuilding.net/arsenic>.

Conclusions and Recommendations

Maine people can now join others across the nation whose bodies are testament to a chemically-saturated world. While Maine seems remote and at times pristine, the Maine people who participated in this survey have provided evidence that the byproducts of modern life pervade our air, water, and food.

What is most unsettling for the project participants is that no one, not even the doctors leading the study, can explain with any certainty why particular chemicals were found in their bodies, why levels of some chemicals are higher than others, or how the chemicals are affecting their health now or in the future.

However, based on these findings of this survey of pollution in Maine people and similar studies, we draw a few conclusions:

- 1. People are routinely exposed to many hazardous chemicals.** All five of the chemical groups surveyed were detected in the bodies of Maine participants, although all chemicals were not found in all of the people. This represents a partial first snapshot of what chemicals might be found in Maine people.
- 2. These chemicals pose a potentially serious threat to human health.** While more needs to be learned about the health effects of chemicals in humans, review of the latest scientific and medical research reveals mounting evidence that these chemicals can harm the health of adults, children, and the unborn. Several of these chemicals are not only toxic but also possess other troubling hazardous properties such as being long-lived in the environment (persistent) and building up to high levels in the food web and our bodies (bioaccumulative).
- 3. Everyday products and materials are a major source of chemical exposure.** The chemicals that are present in Maine people are also common ingredients in the products, plastics, and synthetic materials that fill our homes and workplaces. From the morning's shower, through the work day, to the meals we eat and the beds we sleep in at night, we are surrounded by chemical-laden man-made products. Unfortunately, these chemicals do not stay inside the product. They get into the dust, the air and water, the food supply, and—as we now know—our bodies.
- 4. The safety system for industrial chemicals is broken.** Most of these chemicals that enter our environment are manufactured by the chemical industry and added to the thousands of items in daily commerce that support our modern lifestyle. Yet industry is not required to prove that a chemical is safe before it is manufactured, sold, or used in consumer products. Nor are product makers required to use the safest alternatives, even when non-toxic substitutes are effective, available and affordable. Under our current system, thousands of toxic chemicals have been “grandfathered” in without adequate health and safety testing. And government is handcuffed with undue burden to prove harm before any precautionary actions can be taken to prevent chemical exposure. If this system was working, we would not find hazardous chemicals in people’s bodies.

Recommendations

To prevent pollution in Maine people, government should enact comprehensive safer chemicals policy at the state and federal level. Three actions are needed to close the gaps in our broken chemical system to ensure chemical *safety*, provide useful *data*, and promote innovative *technology*. Together, these reforms will revitalize our toxic-dependent economy through green chemistry.

✓ CLOSE THE SAFETY GAP

- Phase out the most harmful chemicals in favor of safer alternatives, for example Deca-BDE in electronics and furniture, and phthalates and bisphenol A in baby products.
- Search for safer substitutes for all chemicals shown to be hazardous.
- Require that all industrial chemicals be proven safe, especially for children.

✓ CLOSE THE DATA GAP

- Honor the public's right-to-know which hazardous chemicals are in what products.
- Require manufacturers to provide health and safety data on all industrial chemicals.
- Require that chemical manufacturers test and prove the safety of all industrial chemicals in commerce.

✓ CLOSE THE TECHNOLOGY GAP

- Invest in research and development (R&D) of bio-based plastics from Maine potatoes and other "green chemistry" solutions that will boost the state's economy.
- Establish a Green Chemistry Center for Sustainable Production within the University of Maine System to assess hazards and alternatives for harmful chemicals.

Chemical policy reforms at the state and federal level should allow the continued sale of products and production of chemicals only if these safety, data, and technology gaps are effectively closed for industrial chemicals. A safer chemical policy will protect the health of the most vulnerable among us.

The Governor's Task Force on Safer Chemicals in Consumer Products is charged with developing recommendations for a more comprehensive chemical policy for the State of Maine. The Task Force will issue final recommendations to the Governor by October 1, 2007. In Maine, this Task Force should advance meaningful chemical policy reform to ensure that no product sold in the state contains a hazardous chemical for which safer alternatives are available, affordable, and effective.

BOX: What action can individuals take?

Besides supporting chemical policy reform, consumers can take immediate action to protect their family's health. Maine people can take personal action to reduce exposure to toxic chemicals by using safer products for homes and businesses, where available. Low-cost solutions can help reduce toxic exposure until our broken chemical safety system is fixed by policy makers; for example, eating fish low in mercury, testing well water for arsenic, and avoiding personal care products containing phthalates and other toxics. For specific resources to help you choose safer products and smarter practices that reduce chemical exposure, visit www.protectmainefamilies.org/saferproducts.shtml.

Institutional consumers, such as businesses, schools, state government, and hospitals, should take action too. They can purchase safer alternatives and adopt policies to avoid hazardous chemicals in products and materials. One positive example was set when Maine Governor John Baldacci issued an Executive Order in February 2006 calling on state government to reduce pesticide use on the grounds of state office buildings, replace lead wheel weights in the state fleet with lead-free alternatives, avoid dry-cleaning of state uniforms with perchloroethylene and avoid other state purchasing of products and services containing carcinogens or persistent, bioaccumulative and toxic chemicals.⁹⁰

Project Methodology

All project protocols were approved by the University of Southern Maine Office of Research Compliance and Institutional Review Board. Dr. Vincent Markowski and Dr. Richard Donahue, the project's Principal Investigators, provided oversight of the study methodology, data collection, laboratory testing, and data analyses.

The 13 participants in this pilot survey were selected for diversity in occupations, geography, age, and gender. Trained research assistants met with potential subjects to review project goals and methodologies, answer questions, and complete formal consent documents, including a biographical and demographic questionnaire to provide information about their residences, occupations, diet, and potential toxic exposures.

Samples were collected in June, July and August of 2006 using containers and procedures supplied by the analytical laboratories and NorDx clinical laboratories. Phlebotomists at professional collection centers drew blood samples into vacutainers. Approximately 125 ml of blood was collected from each participant following all necessary safety and sample collection protocols. 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. Staff were present to ensure proper hydration. Samples were processed as necessary, frozen, placed upright in appropriate containers with ice packs, and mailed via overnight courier to the analytical laboratories.

Participants provided first morning void urine samples for phthalate, arsenic and creatinine clearance testing. Urine samples were collected in appropriate containers. Samples were transferred to chemically clean 60 ml glass jars for analysis of phthalates and sterile plastic containers for arsenic testing. Urine samples were refrigerated and mailed overnight by the medical facility to the analyzing laboratory. The analytical laboratories provided collection materials and shipping instructions. Hair samples, used to measure long-term mercury exposure, were cut from the base of the scalp (or beyond, if necessary).

All samples were coded to preserve anonymity of the participants. All samples collected were used solely for this project and will be destroyed at its conclusion.

Chemicals Analysis

AXYS Analytical Services, LTD, a private laboratory in Victoria, British Columbia that specializes in trace and ultra-trace detection of environmental contaminants, analyzed urine samples for phthalates and blood serum samples for PBDEs, perfluorinated chemicals, and BPA.

Phthalates. Urine samples were analyzed for phthalate monoesters by AXYS Method MLA-059, *Analysis of Bisphenol A and Phthalate Metabolites in Urine by LC/MS/MS*. Because phthalate esters in humans are metabolized to their respective monoesters, which in turn may be glucuronidated, urine samples were enzymatically hydrolyzed prior to extraction to convert any monoester glucuronides to their respective free monoesters. Samples were extracted on SPE cartridges, eluted, and analyzed by liquid chromatography tandem mass spectrometry (LC/MS/MS). 1 mL samples were buffered with ammonium acetate, and spiked with ¹³C-labeled phthalate monoesters, ¹³C-labeled 4-methylumbelliferone, native 4-methylumbelliferone glucuronide, and β-glucuronidase enzyme. The

treated samples were then incubated to hydrolyze the glucuronides (the completeness of hydrolysis was monitored by the ratio of native to labeled 4-methylumbelliferone). The incubated urine was diluted with high purity water, pH adjusted, and loaded onto pre-conditioned Waters Oasis HLB SPE cartridges, which were washed and then eluted with methanol. Extracts were reduced in volume and spiked with a ¹³C-labeled recovery standard. Analysis was performed on a Micromass Quattro Ultima MS/MS coupled to a Waters 2795 HPLC equipped with a reverse-phase C18 column (7.5 cm, 2.1 mm i.d., 3.5µm particle size). The LC/MS/MS was operated in the MRM mode at unit resolution, using Negative Ion Electrospray ionization. Phthalate monoester concentrations were determined by the isotope dilution method.

PBDEs. Serum samples were analyzed for polybrominated diphenyl ethers (PBDE) via EPA Method 1614, an HRGC/HRMS method that uses isotope dilution internal standard quantification. 6 mL samples were spiked with ¹³C-labeled PBDE surrogates and extracted with formic acid. The extracts were loaded onto pre-conditioned Waters Oasis HLB SPE cartridges, which were washed and then eluted with DCM. Extracts were further cleaned-up on silica, reduced in volume, and spiked with ¹³C-PCB recovery standards. Analysis of the extracts was performed on a Micromass Ultima or VG70 mass spectrometer (MS) coupled to a Hewlett Packard 5890 or 6890 gas chromatograph equipped with a DB-5HT chromatography column (30 m, 0.25 mm i.d., 0.10 µm film thickness). The HRMS was operated at a static (5000) mass resolution in the electron ionization (EI) mode using voltage selected ion recording. PBDE concentrations were determined by isotope dilution or internal standard quantification against the labeled surrogates added at the beginning of analysis using Micromass OPUSQUAN software.

PFCs. Serum was analyzed for PFCs by AXYS Method MLA-042, *Analysis of Perfluorinated Organic Compounds (PFC) in Blood Serum by LC-MS/MS*. 0.5 mL samples were spiked with ¹³C-labeled PFCs and extracted with formic acid. Extracts were loaded onto pre-conditioned Waters Oasis WAX SPE cartridges, which were washed and then eluted with basic methanol. The cleaned-up extracts were spiked with ¹³C-labeled PFC recovery standards, diluted to final volume with methanol, and analyzed by LC/MS/MS. Analysis was performed on a Micromass Quattro Ultima MS/MS coupled to a Waters 2795 HPLC equipped with a reverse-phase C18 column (7.5cm, 2.1mm i.d., 3.5µm particle size). The LC/MS/MS was operated in the MRM mode at unit resolution, using Negative Ion Electrospray ionization. PFC concentrations were determined by isotope dilution or internal standard quantification against the labeled surrogates added at the beginning of the analysis.

BPA. Serum was analyzed for Bisphenol A (BPA) and Bisphenol A diglycidyl ether (BADGE), according to procedures documented in AXYS Method MLA-0056, *Analysis of Bisphenol A and Bisphenol A Diglycidyl Ether in Human Blood Serum by Liquid Chromatography–Mass Spectrometry*. BPA and BADGE may be present in serum as both the free phenol and glucuronated conjugate. Samples were therefore enzymatically hydrolyzed to convert any glucuronates to the free phenol. BADGE is unstable in aqueous solutions due to hydrolytic ring opening of the two epoxide rings and for this reason BADGE was analyzed as its hydrolyzed product, BADGE-4OH. 1mL samples were spiked with deuterated BPA and ¹³C-labeled 4-methylumbelliferone, buffered with ammonium acetate, and further spiked with native 4-methylumbelliferone glucuronide, and β-glucuronidase enzyme. The treated samples were then incubated to hydrolyze the BPA and BADGE glucuronides (the completeness of hydrolysis was monitored by the ratio of native to labeled 4-methylumbelliferone). The incubated samples were diluted with high-purity water and loaded onto preconditioned Waters Oasis HLB SPE cartridges. The cartridges were washed with a series of solutions, and then eluted with ethyl acetate. The cleaned extracts were reduced in volume, reconstituted with methanol,

filtered, spiked with recovery standard, and analyzed by LC/MS/MS. Analysis was performed on a Micromass Quattro Ultima MS/MS coupled to a Waters 2795 HPLC equipped with a reverse-phase C18 column (7.5cm, 2.1mm i.d., 3.5 μ m particle size). The LC/MS/MS was operated in the MRM mode at unit resolution, using Negative Ion Electrospray ionization. BPA concentrations were determined by isotope dilution, while BADGE-4OH was determined by internal standard quantification against the labeled BPA added at the beginning of the analysis.

For phthalates, PFCs, and PBDEs, 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.

Metals Analysis

Lead in blood samples, methyl mercury in hair samples, and both total arsenic and arsenic species in urine samples were performed by Brooks Rand Labs, a private laboratory located in Seattle, Washington, which specializes in trace level metals analysis.

Arsenic. For total arsenic (As), urine samples were closed-vessel oven digested with nitric acid. Digests were then analyzed by Inductively Coupled Plasma-Dynamic Reactive Cell-Mass Spectrometry (ICP-DRC-MS). For inorganic arsenic species, urine samples were extracted with HCl. Aliquots for inorganic arsenic were adjusted to pH 1.5. Sample aliquots for As(III) were adjusted to pH 6. Samples were then analyzed by hydride generation with NaBH₄ reduction, cryogenic trap pre-collection, H₂/Air flame quartz furnace decomposition and atomic absorption detection.

Lead: Whole blood samples were diluted 50x with a diluent comprised of EDTA, TMAH, ethanol, and Triton X-100 in DI water. Digests were then analyzed by Inductively Coupled Plasma-Mass Spectrometry (ICP-MS).

Methylmercury. Hair samples were cut into ~1 cm segments and then washed to remove contaminants deposited on the surface of the hair. Successive wash and filtering cycles were done with Triton-X, acetone and deionized water, followed by oven drying. Washed hair samples were then digested in a KOH/methanol solution. Digestates were then analyzed by ethylation, Tenax trap pre-concentration, gas chromatography separation, pyrolytic combustion and atomic fluorescence spectroscopy.

APPENDIX

Table 1 The Chemicals Tested in Thirteen Mainers

Table 2 Complete Results of Chemical Screening of Thirteen Mainers

Table 3 Summary of Results of Maine Body Burden Study

Table 1 -- The Chemicals Tested in Thirteen Mainers

Chemical Group Medium Tested Units of Measurement	Chemical Tested		Chemical Description
Phthalates Tested in Urine Results reported as nanograms per milliliter (ng/ml) or parts per billion (ppb)	MMP	Mono-methyl phthalate	A metabolite of DMP (dimethyl phthalate)
	MEP	Mono-ethyl phthalate	A metabolite of DEP (diethyl phthalate)
	MBP	Mono-butyl phthalate	A metabolite of DBP (dibutyl phthalate)
	MBzP	Mono-benzyl phthalate	A metabolite of BzBP (benzylbutyl phthalate)
	MEHP	Mono-2-ethylhexyl phthalate	All three are metabolites of DEHP, which is di-(2-ethylhexyl) phthalate
	MEOHP	Mono-(2-ethyl-5-oxohexyl) phthalate	
	MEHHP	Mono-(2-ethyl-5-hydroxyhexyl) phthalate	
PBDEs Tested in blood Results reported as picograms per gram (pg/g) on a lipid weight basis or parts per trillion (ppt)	Polybrominated diphenyl ethers 46 different PBDEs were measured of the 209 congeners that exist. See Table 2 for full list.		PBDE congeners are named from BDE-1 to BDE-209. They differ only by the location and number of the bromine atoms, which varies from 1 to 10. Congeners are chemical compounds that share the same basic structure.
PFCs or perfluorinated chemicals Tested in blood Results reported as nanograms per milliliter (ng/mL) or parts per billion	PFBA	Perfluorobutanoic acid	PFOA is the most prominent among this group of perfluorinated carboxylic acids. It has eight carbon atoms. The related compounds in this group range from having four to twelve carbon atoms. While PFOA is being phased out of some products, all of these compounds are possible breakdown products or manufacturing intermediates of other commercial PFCs.
	PFPeA	Perfluoro-n-pentanoic acid	
	PFHxA	Perfluorohexanoic acid	
	PFHpA	Perfluoroheptanoic acid	
	PFOA	Perfluorooctanoic acid	
	PFNA	Perfluorononanoic acid	
	PFDA	Perfluorodecanoic acid	
	PFUnA	Perfluoroundecanoic acid	
	PFDoA	Perfluorododecanoic acid	
	PFBS	Perfluorobutanesulfonate	Among these perfluorinated sulfonates, PFOS was phased out of Scotchgard in 2000 and replaced with PFBS. PFHxS is still used.
	PFHxS	Perfluorohexanesulfonate	
	PFOS	Perfluorooctanesulfonate	
	PFOSA	Perfluorooctanesulfonamide	A breakdown product of PFCs, which breaks down itself into PFOS
BPA Tested in blood Results in ng/mL or ppb	BPA	Bisphenol A	Monomer for polycarbonate plastic
	BADGE-4OH		A metabolite of BADGE (bisphenol A diglycidyl ether) used in epoxy resins
Metals Lead: tested in blood Results in ug/dL Methylmercury: tested in hair Results in ng/g or ppb Arsenic: tested in urine Results in ug/L or ppb	Lead		A soft metal that readily escapes from products with skin contact, as a dust that can be ingested or inhaled, or dissolved in drinking water.
	Methylmercury		A highly toxic form of mercury produced by bacteria in wetland environments from mercury pollution of the air and water, which builds up to high levels in fish and wildlife.
	Arsenic (total, inorganic and As(III))		Total arsenic includes organic arsenic which is relatively low in toxicity as well as highly toxic inorganic arsenic. Arsenic(III) is the most toxic form of inorganic arsenic.

Table 2 -- Complete Results of Chemical Screening of Thirteen Mainers

Chemical Class	Chemical Tested	Regina Creeley	Dana Dow	Paulette Dingley	Amy Graham	Bettie Kettell	Russell Libby	Hannah Pingree	Lauralee Raymond	Vi Raymond	Elise Roux	Charlie Schmidt	Eric Stirling	Denyse Wilson
Phthalates in URINE In each box: The 1 st result is in ng/mL or ~ parts per billion (ppb); The 2 nd result is in ug/gCr-L (creatinine-corrected) or ~ ppb	MMP	< 3.32 < 3.42	< 1.73 < 1.16	12.1 46.5	5.17 7.18	2.99 7.29	13.1 8.9	26.6 23.1	21.6 14.2	19.6 22.7	15.8 8.19	< 6.28 < 5.87	5.56 6.78	< 26.3 < 17.2
	MEP	10.3 10.6	81.5 54.7	45.9 177	26.7 37.1	24.4 59.5	29 19.6	172 150	105 69.1	121 140	395 205	20.7 19.3	38.1 46.5	73.9 48.3
	MBP	26.2 27.0	32 21.5	22.6 86.9	15.7 21.8	28 68.3	38.7 26.1	75.7 65.8	107 70.4	66.2 76.5	97.4 50.5	48.5 45.3	35.4 43.2	141 92.2
	MBzP	28.2 29.1	20.8 13.6	17.9 68.8	9.12 12.7	25.1 61.2	46.8 31.6	54 47	12.7 8.36	26.5 30.6	127 65.8	6.73 6.29	22.5 27.4	30.5 19.9
	MEHP	10.2 10.5	2.42 1.62	8.18 31.5	5.23 7.26	13 31.7	24.7 16.7	45.3 39.4	23.3 15.3	57.9 66.9	7.89 4.09	4.52 4.22	2.82 3.44	10.8 7.06
	MEOHP	15.4 15.9	6.15 4.13	8.05 31.0	7.47 10.4	30.9 75.4	27.6 18.6	106 92.2	52.1 34.3	114 132	29.1 15.1	8.56 8.00	8.81 10.7	22.6 14.8
	MEHHP	41.3 42.6	12.5 8.39	29.5 113	23.9 33.2	66.4 162	59.5 40.2	197 171	95.8 63.0	280 324	54.5 28.2	43.6 40.7	20.6 25.1	49.6 32.4
	Total Phthalates	133 137	156 105	144 555	93.3 130	191 465	239 162	677 588	418 275	685 793	727 377	136 127	134 163	342 223
	Chemical Class	Chemical Tested	Regina Creeley	Dana Dow	Paulette Dingley	Amy Graham	Bettie Kettell	Russell Libby	Hannah Pingree	Lauralee Raymond	Vi Raymond	Elise Roux	Charlie Schmidt	Eric Stirling
PFCs in blood SERUM results shown in ng/mL or ~ parts per billion (ppb)	PFBA	< 0.576	< 0.576	< 0.576	< 0.576	< 0.576	< 0.576	< 0.576	< 0.576	< 0.576	< 0.576	< 0.576	< 0.576	< 0.576
	PFPeA	< 0.544	< 0.544	< 0.721	< 0.544	< 0.544	< 0.544	< 0.544	< 0.544	< 0.576	< 0.544	< 0.544	< 0.544	< 0.544
	PFHxA	< 0.476	< 0.476	< 0.478	< 0.476	< 9.86	< 0.476	< 0.476	< 0.487	< 0.476	< 0.719	< 0.755	< 0.539	< 0.811
	PFHpA	< 0.556	< 0.556	< 0.556	< 0.556	< 0.556	< 0.556	< 0.556	< 0.556	< 0.556	< 0.556	< 0.556	< 0.556	< 0.556
	PFOA	1.52	18.4	3.05	1.05	4.55	4.43	5.03	3.7	7.69	3.55	4.41	5.61	1.23
	PFNA	0.923	3.07	1.21	< 0.468	1.57	1.86	1.96	0.93	1.61	1.16	1.56	1.37	0.697
	PFDA	< 0.504	1.21	< 0.504	< 0.504	0.615	0.551	0.683	< 0.504	1.23	0.628	< 0.504	0.826	< 0.504
	PFUnA	< 0.512	1.39	< 0.512	< 0.512	< 0.512	< 0.512	1.19	0.595	0.744	0.69	0.633	0.932	< 0.512
	PFDoA	< 0.576	< 0.576	< 0.576	< 0.576	< 0.576	< 0.576	< 0.576	< 0.576	< 0.576	< 0.576	< 0.576	< 0.576	< 0.576
	PFBS	< 1.41	< 1.41	< 1.41	< 1.41	< 1.41	< 1.41	< 1.41	< 1.41	< 1.41	< 1.41	< 1.41	< 1.41	< 1.41
	PFHxS	3.44	9.01	1.55	< 1.29	2.19	< 1.29	< 1.29	2.46	< 1.29	< 1.29	2.74	1.57	< 1.29
	PFOS	13.7	38	14.4	6.11	21.4	15.4	14.2	13.4	10.9	14.9	25	14.1	6.69
	PFOSA	< 0.48	< 0.48	< 0.48	< 0.48	< 0.48	< 0.48	< 0.48	< 0.48	< 0.48	< 0.48	< 0.48	< 0.48	< 0.48
	Total PFCs	22.3	73.3	25.0	10.8	32.8	25.4	26.0	23.6	25.0	23.8	36.9	26.7	12.0
Chemical Class	Chemical Tested	Regina Creeley	Dana Dow	Paulette Dingley	Amy Graham	Bettie Kettell	Russell Libby	Hannah Pingree	Lauralee Raymond	Vi Raymond	Elise Roux	Charlie Schmidt	Eric Stirling	Denyse Wilson
BPA in blood SERUM	BPA	< 0.752	< 0.52	3.75	< 0.52	< 0.52	< 0.52	< 1.64	< 0.571	4.49	6.64	< 3.24	< 0.52	< 0.52
	BADGE-4OH	< 2.6	< 2.6	6.35	2.81	< 4.06	< 2.6	6.69	< 2.6	119	59.7	< 2.6	< 2.6	< 2.6
results shown in ng/mL or ~ parts per billion (ppb)														

Chemical Class	Chemical Tested	Regina Creeley	Dana Dow	Paulette Dingley	Amy Graham	Bettie Kettell	Russell Libby	Hannah Pingree	Lauralee Raymond	Vi Raymond	Elise Roux	Charlie Schmidt	Eric Stirling	Denyse Wilson
PBDEs in blood SERUM results shown in pg/g on a lipid weight basis, which is approximately the same as parts per trillion (ppt)	BDE-7	< 27.4	< 22.1	< 27.2	< 43.6	< 25.7	< 25.7	< 24.6	< 31.3	< 23.4	< 34.1	< 27.7	< 19.1	< 31.9
	BDE-8/11	< 27.4	< 22.1	< 27.2	< 43.6	< 25.7	< 25.7	< 24.6	< 31.3	< 23.4	< 34.1	< 27.7	< 19.1	< 31.9
	BDE-10	< 27.4	< 22.1	< 27.2	< 43.6	< 25.7	< 25.7	< 24.6	< 31.3	< 23.4	< 34.1	< 27.7	< 19.1	< 31.9
	BDE-12/13	< 27.4	< 22.1	< 27.2	< 43.6	< 25.7	< 25.7	< 24.6	< 31.3	< 23.4	< 34.1	< 27.7	< 19.1	< 31.9
	BDE-15	439	86.7	* 141	80.2	200	506	603	225	119	86.1	144	60.6	480
	BDE-17/25	154	72.1	36.7	58.1	506	212	58.7	217	69.4	138	202	84.4	* 58.2
	BDE-28/33	900	599	350	518	2200	1850	554	1350	608	694	1340	483	801
	BDE-30	< 27.4	< 22.1	< 27.2	< 52.1	< 25.7	< 25.7	< 24.6	< 31.3	< 23.4	< 34.1	< 27.7	< 19.1	< 31.9
	BDE-32	< 27.4	< 22.1	< 27.2	< 43.6	< 25.7	< 25.7	< 24.6	< 31.3	< 23.4	< 34.1	< 27.7	< 19.1	< 31.9
	BDE-35	* 102	* 101	* 67.1	46.3	* 69.4	* 101	* 119	* 47.9	* 100	* 41.4	< 27.7	* 68.5	* 66.1
	BDE-37	< 27.4	< 22.1	< 27.2	< 43.6	< 25.7	37.1	* 55.3	< 31.3	< 23.4	< 34.1	< 27.7	< 19.1	< 31.9
	BDE-47	8380	6490	2900	8390	33500	17200	5460	17500	7450	13000	18200	6550	6570
	BDE-49	136	57.5	48	83.9	275	140	97.6	117	98.3	112	156	70.5	82.6
	BDE-51	< 27.4	* 22.6	< 27.2	< 43.6	83.8	47.5	< 24.6	* 76.4	< 23.4	< 34.1	* 38	< 19.1	< 31.9
	BDE-66	122	* 73.4	* 55.2	128	506	240	87.4	215	* 101	169	207	93.3	93.5
	BDE-71	* 31.3	< 22.1	< 27.2	< 43.6	70.8	30.5	< 24.6	< 31.3	< 23.4	< 34.1	< 27.7	< 19.1	< 31.9
	BDE-75	< 27.4	< 22.1	< 27.2	< 43.6	85.1	32.9	< 24.6	* 38.5	< 23.4	< 34.1	< 27.7	< 19.1	< 31.9
	BDE-77	< 27.4	< 22.1	< 27.2	< 43.6	< 25.7	< 25.7	< 24.6	< 31.3	< 23.4	< 34.1	< 27.7	< 19.1	< 31.9
	BDE-79	39	23.6	< 27.2	* 55.5	140	103	26.6	78.1	32.8	* 38.8	48	25.5	36.3
	BDE-85	177	103	53.2	148	745	254	95.3	306	137	229	365	99.9	75.9
	BDE-99	2210	1210	987	1870	9280	3290	1400	3170	1490	3310	3680	1440	1130
	BDE-100	1550	1440	454	1260	6350	4780	866	7230	1650	2550	3380	1150	922
	BDE-105	< 32	< 22.1	< 27.2	< 57.8	< 34.3	< 26	< 24.6	< 31.3	< 23.4	< 34.1	< 36	< 19.1	< 31.9
	BDE-116	< 45.4	< 22.1	< 28	< 85.4	< 48.8	< 36.9	< 25.3	< 42.3	* 51.7	< 34.3	< 50.5	23.9	< 33
	BDE-119/120	< 29	< 22.1	< 27.2	< 53.6	* 54.8	* 48	< 24.6	* 56.2	< 23.4	< 34.1	< 32.8	< 19.1	< 31.9
	BDE-126	< 27.4	< 22.1	< 27.2	< 43.6	< 25.7	< 25.7	< 24.6	< 31.3	< 23.4	< 34.1	< 27.7	< 19.1	< 31.9
	BDE-128	< 27.9	< 134	< 31.4	< 52.6	< 43.2	< 34.6	< 31.5	< 40.8	< 25.2	< 39.4	< 37.3	< 20.8	< 36.5
	BDE-138/166	47.9	* 40.4	< 27.2	* 56.8	121	101	< 24.6	84.2	32.4	59	93.8	21.2	< 31.9
	BDE-140	56.7	28	< 27.2	49.4	77.8	68.3	< 24.6	113	31.1	49.6	72.6	50.5	< 31.9
	BDE-153	4840	3660	1390	4520	4060	5570	2780	15300	3030	2840	4300	9120	2150
	BDE-154	200	145	96.8	225	746	505	131	396	166	280	375	150	119
	BDE-155	* 45.9	* 31	< 27.2	* 70.5	* 78.6	* 59.5	* 38.4	* 57.8	* 34.6	* 57.6	* 62.3	* 42.2	* 37.3
	BDE-181	< 28.2	< 26.8	< 27.2	< 43.6	< 25.7	< 25.7	< 24.6	< 31.3	< 23.4	< 34.1	< 27.7	< 19.1	< 31.9
BDE-183	297	352	* 147	623	531	445	159	417	210	271	1400	262	328	
BDE-190	< 40.8	< 39.7	< 27.2	< 45.5	< 30	< 32.5	< 24.6	< 31.3	< 23.4	< 34.1	< 36.3	< 19.1	< 31.9	
BDE-203	190	* 248	90.1	* 177	91.1	* 153	134	* 111	* 94.8	* 129	303	104	* 191	
BDE-206	< 328	< 799	< 328	< 526	< 308	< 308	< 441	< 378	< 282	< 408	< 666	< 345	< 768	
BDE-207	< 328	< 799	< 328	< 526	< 308	< 308	< 441	< 378	< 282	< 408	< 666	< 345	< 768	
BDE-208	< 328	< 799	< 328	< 526	308	< 308	< 441	< 378	< 282	< 408	< 666	< 345	< 768	
BDE-209	< 4920	< 10700	< 4910	< 3940	< 4620	< 3080	< 8820	< 5660	< 4220	< 6120	< 9990	< 3450	< 11500	
Total PBDEs	20,088	14,751	6,918	18,579	59,869	35,955	12,782	47,343	15,518	24,229	34,594	19,971	13,410	

Chemical Class	Chemical Tested	Regina Creeley	Dana Dow	Paulette Dingley	Amy Graham	Bettie Kettell	Russell Libby	Hannah Pingree	Lauralee Raymond	Vi Raymond	Elise Roux	Charlie Schmidt	Eric Stirling	Denyse Wilson
Metals														
Pb in BLOOD in ug/dL	Lead	1.10	1.06	1.46	0.549	0.716	1.07	1.20	0.719	0.884	0.507	3.26	1.14	no data
MeHg in HAIR in ng/g or ppb	Methylmercury	* 156	497	396	437	333	* 215	1140	759	291	778	** 186	1180	257
As in URINE 1 st # is in ug/L	Arsenic (Total)	843 869	98.1 65.8	3.51 13.5	11.2 15.5	21.2 51.7	16.1 10.9	30.7 26.7	59.6 39.2	11.1 12.8	8.18 4.24	40.2 37.6	58.6 71.5	56.7 37.1
2 nd is ug/gCr-L (creatinine corrected) – both are ~ ppb	Arsenic (Inorganic)	0.238 0.245	0.496 0.333	0.162 0.623	0.575 0.799	0.173 0.422	1.11 0.75	1.13 0.98	1.07 0.70	0.753 0.871	0.48 0.25	0.508 0.476	0.299 0.365	1.16 0.76
	Arsenic(III)	0.210 0.216	0.420 0.282	0.160 0.623	0.450 0.625	0.150 0.366	0.740 0.500	0.730 0.635	0.83 0.55	0.620 0.717	0.44 0.52	0.140 0.131	0.200 0.244	0.83 0.54
Chemical Class	Chemical Tested	Regina Creeley	Dana Dow	Paulette Dingley	Amy Graham	Bettie Kettell	Russell Libby	Hannah Pingree	Lauralee Raymond	Vi Raymond	Elise Roux	Charlie Schmidt	Eric Stirling	Denyse Wilson
Protein in URINE	Creatinine (mg/dL)	97	149	26	72	41	148	115	152	86.5	193	107	82	153
These normal protein levels are used to adjust the measured chemicals in urine to account for dilution due to varying amounts of fluid intake per person														

NOTES:

Boldface type in a colored box indicates the chemical was detected

< the chemical was not found above the limit of detection indicated; the chemical might be present below this limit

* the chemical was detected but the quantification criteria were not met, therefore the result represents the estimated maximum possible concentration

** estimate

To calculate the sum total for Phthalates, PFCs and PBDEs, any value reported as non-detected (< #) was assigned a value of ½ the detection limit; For the same purpose, any PBDE value that was flagged (*) as not meeting quantification criteria was assigned a value of ½ the reported value.

Table 3 -- Summary of Results of Maine Body Burden Study

RESULTS FROM 13 MAINE PARTICIPANTS				RESULTS FROM OTHER STUDIES			
<i>Phthalates</i>	units = ug/gCr-L (creatinine corrected)			from federal CDC 3 rd National Exposure Report ⁹¹ n = 2,536 for MEP; n = 2,772 for all other phthalates ¹			
	Minimum	Maximum	Median – or 50 th %tile	Median – or 50 th %tile	75 th %tile	90 th %tile	95 th %tile
MMP	< 1.16	46.5	8.19	1.33	2.62	5.00	7.97
MEP	10.6	205	54.7	147	388	975	1860
MBP	21.8	92.2	50.5	26.0	51.6	98.6	149
MBzP	6.29	68.8	29.1	13.5	26.6	55.1	90.4
MEHP	1.62	66.9	10.6	3.89	7.94	18.2	32.8
MEOHP	4.13	132	15.9	11.2	21.3	45.1	87.5
MEHHP	8.39	324	40.7	16.6	32.3	70.8	147
Sum TOTAL	105	793	223	219	530	1,268	2,375
<i>PBDEs</i>	units = pg/g on a lipid weight basis			from McDonald 2005 ⁹² n = 62 women from CA & IN		n = 10	n = 11
	Minimum	Maximum	Median – or 50 th %tile	Median – or 50 th % tile	95 th %tile	Washington Median ⁹³	California Median ⁹⁴
BDE-15	60.6	603	144			275	-
BDE-17/25	36.7	506	84.4			61.7	-
BDE-28/33	350	2200	694			1128	-
BDE-35	< 27.7	119	* 68.5			< 5.64	-
BDE-37	< 19.1	* 55.3	< 27.7			10.0	-
BDE-47	2900	33500	8380	included below	incl. below	19950	14100
BDE-49	48	275	98.3			178	-
BDE-51	< 19.1	83.8	< 31.9			~ 12	-
BDE-66	* 55.2	506	122			170	-
BDE-71	< 19.1	70.8	< 31.3			< 17.4	-
BDE-75	< 19.1	85.1	< 27.7			25.0	-
BDE-79	< 27.2	140	* 38.8			* 61.1	-
BDE-85	53.2	745	148			346	-
BDE-99	987	9280	1870	included below	incl. below	4255	3100
BDE-100	454	7230	1550	included below	incl. below	3115	2100
BDE-116	< 22.1	* 51.7	< 36.9			< 22.4	-
BDE-119/120	< 19.1	* 56.2	< 31.9			22.8	-
BDE-138/166	21.2	121	47.9			73.8	-
BDE-140	< 24.6	77.8	49.6			~ 44	-
BDE-153	1390	15300	4060	included below	incl. below	2725	3400
BDE-154	96.8	746	200	included below	incl. below	368	280
BDE-155	< 27.2	* 78.6	* 45.9			43.4	-

¹ Based on total results for the U.S. population aged 6 and older from the 2001-2002 National Health and Nutrition Examination Survey (NHANES), except that results for MBP are from the 1999-2000 survey because it was reported then as the sum of the two isomers of mono-butyl phthalate.

BDE-183	* 147	1400	328			218	-
BDE-203	90.1	303	134			152	-
Sum TOTAL	6,918	59,869	19,971	40,700	305,000	47,500	22,980
PFCs	units = ng/mL in blood serum (wet weight)			n = 476 women & 442 men	n = 10	n = 12	
	Minimum	Maximum	Median	National Mean (estimated)⁹⁵	Washington Median⁹⁶	California Median⁹⁷	
PFOA	1.05	18.4	4.41	3.97 to 6.98	3.6	5.3	
PFNA	< 0.468	3.07	1.56	0.51 to 1.10	-	1.67	
PFDA	< 0.504	1.23	0.551	-	-	0.43	
PFUnA	< 0.512	1.39	0.595	-	-	0.40	
PFHxS	< 1.29	9.01	1.57	4.33	-	2.44	
PFOS	6.11	38	14.2	23.4 to 40.2	21.3	25.6	
Sum TOTAL	10.8	73.3	25.0	32.2 – 52.6	24.9	35.8	
BPA	units = ng/mL in blood serum (wet weight)			n = 7 for BPA n = 30 for BADGE	n = 11	n = 14 women ♀ n = 11 men ♂	
	Minimum	Maximum	Range Detected	Geometric Mean - EWG⁹⁸	California Median⁹⁹	Mean - Takeuchi and Tsutsumi¹⁰⁰	
BPA	< 0.52	6.64	3.75 – 6.64	1.08	0.46	0.64 ♀ to 1.49 ♂	
BADGE-4OH	< 2.6	119	2.81 - 119	9.33	12.8	-	
Metals							
Lead	Minimum	Maximum	Median - 50%	Median – or 50th%tile	75th%tile	90th%tile	95th%tile
	0.51	3.26	1.08	1.40	2.20	3.40	4.40
Units = micrograms of lead per deciliter of blood (ug/dL)				n = 8945; from federal CDC 3 rd National Exposure Report ¹⁰¹			
Mercury	Minimum	Maximum	Median - 50%	U.S. Women of Childbearing Age		U.S. Women of Childbearing Age	
Methylmercury				Mean	95th %tile	Median	90th %tile
Top row: n = 13 Mainers	156	1180	396	360	2400	200	1400
	257	1140	759				
Bottom row: n = 5 Maine women of childbearing age				Smith (1997) <i>cited in</i> National Research Council ¹⁰²		n = 702 (CDC, 2001) ¹⁰³	
Units = nanograms methylmercury per gram of hair (ng/g) or ppb							
Reference Dose, level above which fetal brain development is at risk = 1000 ppb methylmercury in hair							
Arsenic	Minimum	Maximum	Median - 50%	Pellizari & Clayton (2006)¹⁰⁴	Washington Median¹⁰⁵		
				Median – 50%			
Total As	3.51	843	30.7	10.23	11		
Inorganic As	0.16	1.16	0.51	-	-		
Arsenic(III)	0.14	0.83	0.44	-	-		
Units = micrograms of arsenic per liter of urine (ug/L) or ppb				n = 102		n = 10	

NOTES: Minimum and Maximum are the lowest and highest values reported among the 13 Maine participants. The median is the reported value that falls in the middle of the range of all reported values. The median is the same as the 50th percentile (50th %tile), which means that half or 50% of the reported values are less than this number and half are greater than it; The 75th percentile (75th %tile) is the number that is greater than three-quarters or 75% of all the reported values (and is less than one-quarter or 25% of all the reported values); The 90th percentile (90th %tile) is the number that's greater than nine-tenths or 90% of all the reported values (and

less than 10% of all reported values); The 95th percentile (95th %tile) is the number that is greater than 95% of all the reported values (and less than 5 % of all reported values); < means that the chemical was not found above the limit of detection indicated (but the chemical might be present below this level); * means that the chemical was detected but the quantification criteria were not met, therefore the result represents the estimated maximum possible concentration for that sample; n = the number of individuals sampled. For PFCs, the sum total is the median value of the minimum, maximum, and total among all Maine participants. For the comparative results, the sum total is the sum of the reported values. The values reported for the PFC national mean are the least squares means estimates of serum concentrations for non-Hispanic white females (lower value) and non-Hispanic white males (higher value) from pooled samples obtained through the 2001-2002 NHANES. The lead national mean is based on total results for the U.S. population aged one and older from 2001-2002 NHANES. See note 1, Table 16, p.38.

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