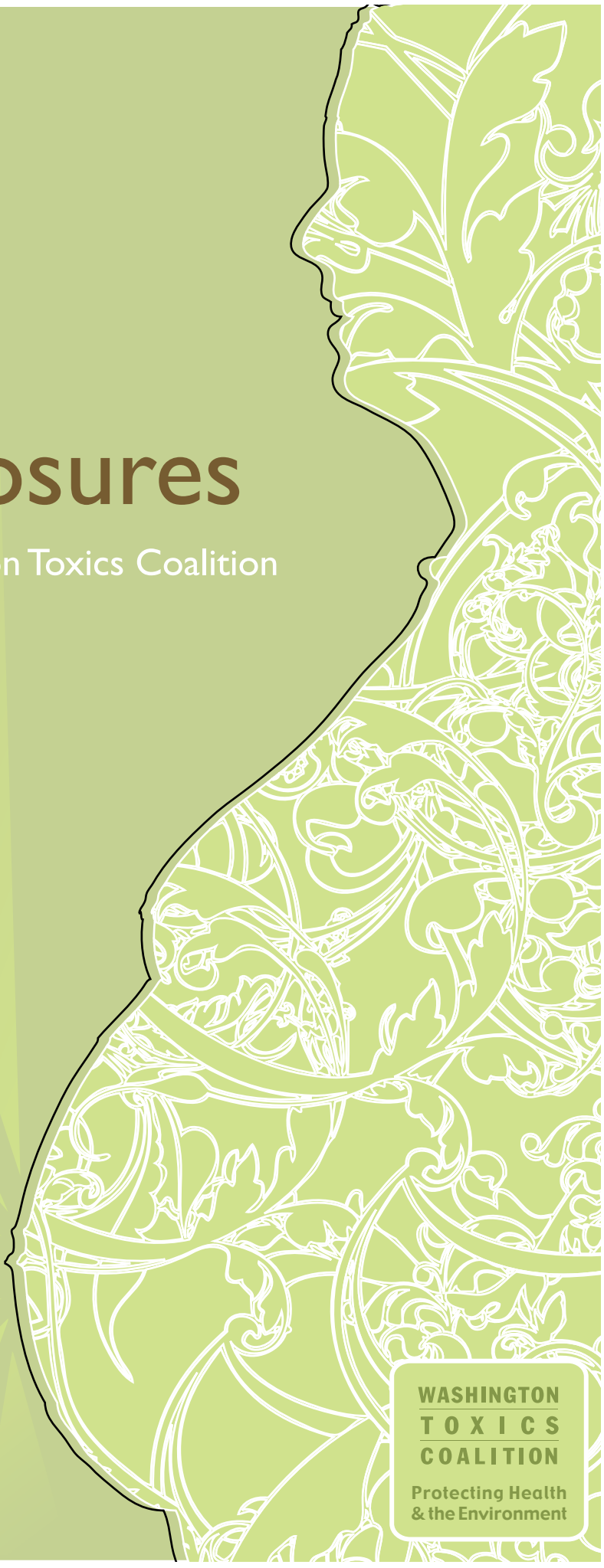
Earliest Exposures

A Research Project by Washington Toxics Coalition



WASHINGTON
TOXICS
COALITION

Protecting Health
& the Environment

Earliest Exposures

A Research Project by
Washington Toxics Coalition

Study completed in collaboration with
the Commonweal Biomonitoring Resource Center
and the Toxic-Free Legacy Coalition

Written by Erika Schreder, Staff Scientist, Washington Toxics Coalition

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Executive Summary

The fetus is uniquely vulnerable to the effects of toxic chemicals. In this study, we investigated the environment experienced by nine fetuses—their mothers. We tested nine pregnant women, from Washington, Oregon, and California, during the second trimester of their pregnancies.

Our tests measured levels of five chemical groups, including phthalates, mercury, perfluorinated compounds or "Teflon chemicals," bisphenol A, and the flame retardant tetrabromobisphenol A, in the blood and urine of pregnant women. Tests also measured levels of thyroid hormones, critical for a healthy pregnancy.

Results from this study reveal that children spend their first nine months in an environment that exposes them to known toxic chemicals.

Key Findings

1. Chemicals from everyday products contaminate mothers' bodies, and babies enter the world already exposed to known toxics. This study detected 13 foreign chemicals in pregnant women, including phthalates, bisphenol A, mercury, and "Teflon chemicals." These chemicals can cause reproductive problems and cancer, disrupt hormonal systems such as the thyroid, and can impair brain development.

Specific findings include:

- Every woman we tested was exposed to bisphenol A, the hormone disrupting chemical used to make polycarbonate plastic and the lining for food cans.
- Each woman had at least two and as many as four "Teflon chemicals," or perfluorinated compounds, in her blood. These are chemicals used to create stain-protection products and non-stick cookware.
- Mercury, known to harm brain development, was in the blood of every woman in our study.
- Every woman was exposed to at least four phthalates, the plasticizers and fragrance carriers found in consumer products from shower curtains to shampoo.

2. The developing fetus is exquisitely vulnerable to the effects of toxic chemicals. The fetus develops at a breakneck pace in the womb, and that development is easily derailed by toxic chemicals. The fetus also has a very limited ability to detoxify foreign chemicals. With chemicals like bisphenol A and the others in our tests passing easily through the placenta, there is cause for grave concern about their impacts on fetal development.

3. Policymakers can protect mothers and children by ensuring that only the safest chemicals are used in products sold in the United States. States have taken the lead by passing policies that begin to take action on the most hazardous chemicals, requiring manufacturers to report their use and replace them with safer chemicals. An updated federal law would protect mothers and children in all states from harmful chemicals.

Recommendations

The United States operates under a toxics law that allows manufacturers to continue using chemicals with known hazards. The Toxic Substances Control Act (TSCA), meant to keep chemicals that can harm our health out of the products we buy, has failed in its mission. Since its 1976 passage, it has resulted in testing of only 200 chemicals out of 80,000 believed in current production. This tremendous dearth of protection has inspired action at the state level and Congressional proposals to reform TSCA.

To adequately protect all people, we recommend the following actions for states and the federal government:

1. Pass policies that protect the most vulnerable. We need policies that keep toxic chemicals away from pregnant women and the developing fetus by doing the following:

- Immediately initiate action to eliminate the use of persistent toxic chemicals, which are those that build up in our bodies or are passed on to the next generation.
- Reduce the use of chemicals that can cause serious health problems such as cancer and reproductive harm, can disrupt the normal function of hormones, or can lead to learning disabilities.
- Allow manufacturers to create consumer products using only chemicals they have tested fully for safety and that do not cause cancer, reproductive harm, disrupt hormones, or cause learning disabilities.

2. Hold industry responsible for testing chemicals and providing full information on their hazards. Chemical manufacturers should test chemicals and provide full information on their hazardous properties and potential impact on health and the environment. The public, workers, and businesses have a right to know what possible harms might result from these chemicals, and health and environmental agencies need this information to make the right decisions to protect health.

3. Maintain the ability of states to set the highest standards to protect health. States are proving that they respond to the need to protect public health with strong, sensible policies. That ability to respond must be maintained, with enhanced coordination between state and federal governments and between federal agencies. Specifically, new federal laws must preserve the rights of the states to enact legislation that is more protective than federal law.

Introduction

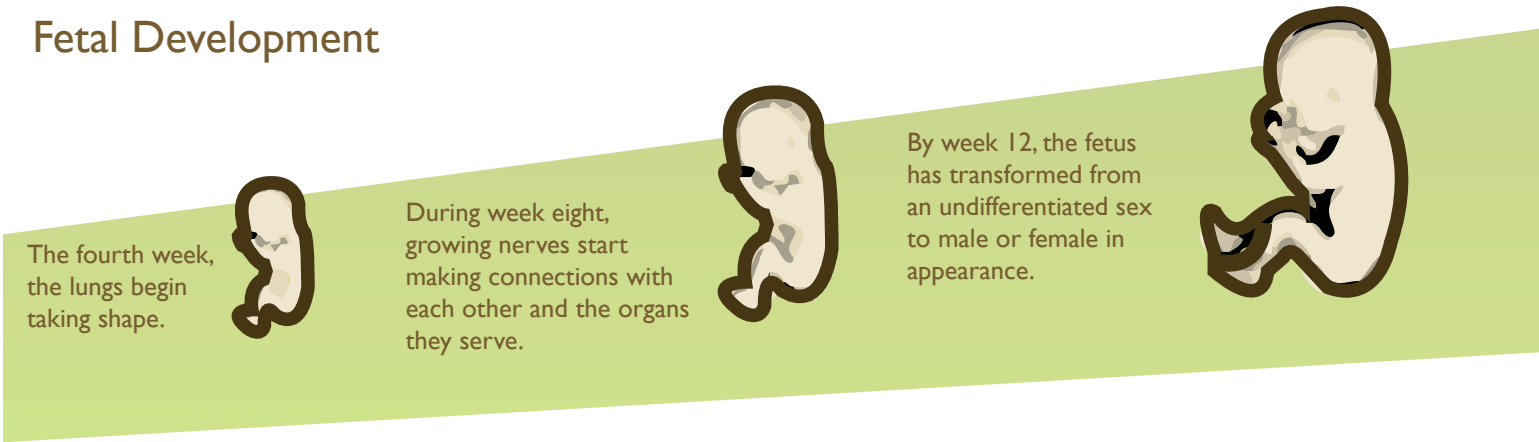
For every woman fortunate enough to experience pregnancy and childbirth, the nine months of experiencing a child growing within are nine months of magic. She has not yet given birth, but has already taken on the role of protector and nurturer. She has yet to see her baby's face, but has begun to assume responsibility for her future child's well-being, making sure he has adequate nutrition and is kept from the hazards of the world.

Once, those hazards were primarily infectious diseases that could harm the developing fetus. Today, babies also do battle with the foot soldiers of the chemical revolution: before a child meets his mother, he has made the acquaintance of harmful chemicals, ranging from the pesticides used in growing his mother's food to the plasticizers in her perfume. Toxic chemicals enter the body of the mother-to-be through her breath, food, drink, and skin. Then, to a large extent, they make their way to the developing fetus through the placenta and umbilical cord: 287 different chemicals have been found in the umbilical cord at the time of birth (*1*).

Unfortunately, the fetus is ill-equipped to face this chemical onslaught. Many of the key mechanisms for detoxifying and expelling hazardous chemicals are completely missing or underdeveloped in the fetus. At the same time, organ systems that will last a lifetime are developing at a breathtaking pace (*2, 3*):

- The fourth week, the lungs begin taking shape.
- During week eight, growing nerves start making connections with each other and the organs they serve.
- By week 12, the fetus has transformed from an undifferentiated sex to male or female in appearance.
- During the 18th week, ovarian follicles begin to form in female fetuses.
- Between weeks 28 and 36, the testes migrate from the abdomen into the scrotum in male fetuses.

Fetal Development



It should come as no surprise, then, that exposures to toxic chemicals before birth are consistently found to have the most serious and irreversible consequences when compared to exposures at other times of life. How this precisely occurs is in many cases still being clarified, but we are certain that exposures are occurring. Toxic chemicals found in umbilical cord blood include mercury, pesticides, PCBs, the perfluorinated compounds known as "Teflon chemicals," and toxic flame retardants (*1*). Amniotic fluid has been found to contain the DDT breakdown chemical DDE, bisphenol A, PCBs, and other chemicals (*4*).

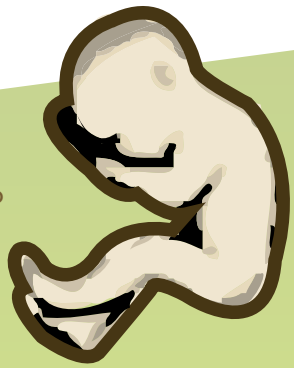
The powerful influence of toxic chemicals on the developing fetus has been dramatically demonstrated in several cases, as in the limb deformities that resulted from the use of thalidomide in the late 1950s to treat morning sickness. Also in the 1950s, mercury-contaminated fish eaten by pregnant women emerged as the culprit in the epidemic of children born with cerebral palsy-like symptoms in Minamata, Japan (*5*). More recently, growing evidence suggests that the exposure of pregnant women to chemicals in their everyday lives, including some produced in massive quantities such as bisphenol A and phthalates, may also affect the health of their children.

Phthalates are a class of chemicals used in countless products, from the vinyl floors in our kitchens to the creams we use on our skin. The Centers for Disease Control and Prevention (CDC) (*6*) has documented their essentially ubiquitous presence in Americans. And dozens of laboratory studies have illuminated a troubling pattern of birth defects in males, resulting from exposure to some phthalates during fetal development. The cause: phthalates appear to dampen testosterone production before and shortly after birth, setting off a cascade of abnormalities from undescended testes to irregular urinary openings on the penis (known as hypospadias).

Could this be happening to our children today? Could toxic chemicals in the fetal environment be at least partially responsible for rising rates of cancer, learning disabilities, and infertility?

Certainly, we are uniquely vulnerable to toxic chemicals before we are born. Given that, we have shockingly little information on toxic contamination before birth. Our study cannot answer these very important questions, but it opens a window to view the serious threats faced before entering the world—threats that could affect health and well-being for a lifetime.

During the 18th week, ovarian follicles begin to form in female fetuses.



Between weeks 28 and 36, the testes migrate from the abdomen into the scrotum in male fetuses.

Invaders in the Womb

Moms-to-be are known for their neuroses, and for good reason. From the time of the first doctor visit until the baby's birth, they are peppered with advice for keeping the baby safe. Take a prenatal vitamin. Stay out of the hot tub. Exercise, but not too much. Do everything you can to make the baby's environment as perfect as possible.

"..fetuses are uniquely defenseless when it comes to keeping toxic chemicals from harming them. Rapidly dividing cells and complex, fast-moving development put them at risk of that development going awry. "

Why does the new mom go to all this trouble to give her baby the best start? Perhaps she knows intuitively what scientists have been piecing together over many decades: the development of the fetus is a delicate dance. An incredibly complex array of factors must all line up properly, hormones at the right levels and genes turning on and off when and how they should. Timing and balance are everything.

As we have learned that toxic chemicals can actually mimic hormones and otherwise disrupt this delicate dance, the modern pregnant woman has a new list of worries. When she is expecting, she can expect to think about making sure her water bottle is safe, choosing the right fish to eat, and staying off stain-resistant couches.

And she should. Because even more than young children, fetuses are uniquely defenseless when it comes to keeping toxic chemicals from harming them. Rapidly dividing cells and complex, fast-moving development put them at risk of that development going awry. Changes during critical periods can result in permanent alterations with lifelong impacts (*7*). At the same time, the detoxification weapons wielded by adults are in some cases missing or just developing in the fetus, and differences in metabolism, fat content, kidney function, and other factors can mean that toxic chemicals have vastly different effects on the very young (*8*).

Some adult detoxification systems may also be turned down during pregnancy, resulting in slower breakdown of foreign chemicals and greater toxic effects (*9*). Scientists are still exploring these mechanisms, but it is possible that some chemicals not only have greater toxic effects during pregnancy, but may in fact be present at higher levels.

About This Study

This study explores the question of to what extent mothers-to-be and their developing fetuses are exposed to toxic chemicals commonly found in consumer products. We tested for five chemicals (or chemical groups): bisphenol A, phthalates, perfluorinated compounds, tetrabromobisphenol A, and mercury. We also tested levels of thyroid hormones, which can be impacted by these chemicals and which are important for proper brain development.

Participants reside in Washington, Oregon, and California, and include an environmental scientist, a naturopath/midwife, a realtor, a dietician, a nurse, a breastfeeding expert, an engineering manager, an environmental justice leader, and a reproductive health advocate. Our participants come from a diverse geographic area, including Olympia, Seattle, Richland, Issaquah, Milton-Freewater, Oregon (near Walla Walla, Washington), and Oakland and Livermore, California.

Participants qualified for the study if they were pregnant with their first child, had no major health problems, and could schedule testing during their second trimester. The second trimester was chosen to standardize the period of pregnancy among participants, and because of the rapid development occurring at that time. With each participant, we scheduled a one-time blood draw and urine collection. We sent urine to AXYS Laboratory in British Columbia, Canada, to test for bisphenol A and phthalates. We sent blood serum to AXYS for testing for perfluorinated compounds (PFCs) and tetrabromobisphenol A (TBBPA). Brooks Rand Laboratories of Seattle analyzed whole blood samples for mercury, and LabCorp analyzed serum samples for thyroid hormones.

To place the results in context, we compared them with levels found in studies conducted regularly by the CDC through their National Health and Nutrition Examination Survey. We also compared the results with levels of chemicals found in independent studies of pregnant women, where available, and with levels found to cause effects in laboratory studies or associated with harm in epidemiological studies.

Detailed Findings

We tested for five sets of chemicals, for a total of 23 chemicals. Thirteen of those were present in at least one woman, and ten in every woman. These chemicals cross the placenta to the developing fetus, and laboratory studies have connected exposure before birth with increased risk of reproductive problems, learning disabilities, cancer, and smaller birth weight. Controlled studies of the effects of these chemicals on people are not conducted for ethical reasons, so we don't know for sure how they affect people. In many cases, however, a combination of laboratory evidence as well as associations from research on people indicate that the concern likely extends to people.

For most of the chemicals we tested for, we saw a high degree of variation between participants. With this variation, some of the women in our study had particularly high levels of these chemicals in their bodies.

Here, we present detailed information on what we found for the five classes of chemicals in our tests.

Phthalates

Tested for: seven breakdown products of five phthalate chemicals
Found: all seven breakdown chemicals in eight women; six breakdown chemicals in one woman

Phthalates are plasticizers and fragrance carriers found in an impressive array of products: many PVC/vinyl items such as flooring, toys, and shower curtains; personal care products and cosmetics like nail polish and perfume; and products like adhesives and sealants (*6*). They are not chemically bound in these products, so people are exposed to them through breathing in phthalates that have escaped into the air, through house dust, and applying products to the skin (*10, 11*). A large amount of our exposure to some phthalates is also through food (*12, 13*).

Once phthalates enter our bodies, the first step in breakdown is to change them to their monoester forms, which are the forms we measured. We found that nearly all of the women in our study had been exposed to all of the phthalates in our test (results presented in Table 1). For four of the five chemicals we measured, average concentrations were higher in our participants than in larger studies of U.S. adults or women. For example, the geometric mean of MEHHP, a breakdown product of the very widely

"We tested for five sets of chemicals, for a total of 23 chemicals. Thirteen of those were present in at least one woman, and ten in every woman."

Table 1: Phthalate Levels in Nine Pregnant Women

Chemical	Parent Compound	Study Mean	CDC Mean
MMP	DMP (dimethyl phthalate)	4.75	1.06
MEP	DEP (diethyl phthalate)	128.34	191.00
MBP	DBP (dibutyl phthalate)	48.29	17.00
MBzP	BBzP (benzylbutyl phthalate)	12.75	12.70
MEHP	DEHP (di(2-ethylhexyl) phthalate)	5.06	4.20
MEOHP	DEHP (di(2-ethylhexyl) phthalate)	20.95	12.00
MEHHP	DEHP (di(2-ethylhexyl) phthalate)	29.69	18.10

This table presents the geometric means of the levels of phthalates (in ppb) found in nine pregnant women. For comparison, it also presents the geometric means found in approximately 1600 U.S. adults in a 2001-2002 CDC survey (6).

Study Participant

Amy Ellings
Public Health Nutritionist
Olympia, WA
Mother since July 31, 2009
12 chemicals detected

"The government should study how these chemicals affect our health, work with manufacturers to help consumers understand the levels of chemicals in products, and not allow companies to use chemicals that are clearly harmful to our health."

used DEHP, was 29.69 parts per billion (ppb), as compared to a geometric mean of 18.10 ppb found in adults from across the United States in a 2001-2001 CDC survey intended to represent the U.S. population (*6*).

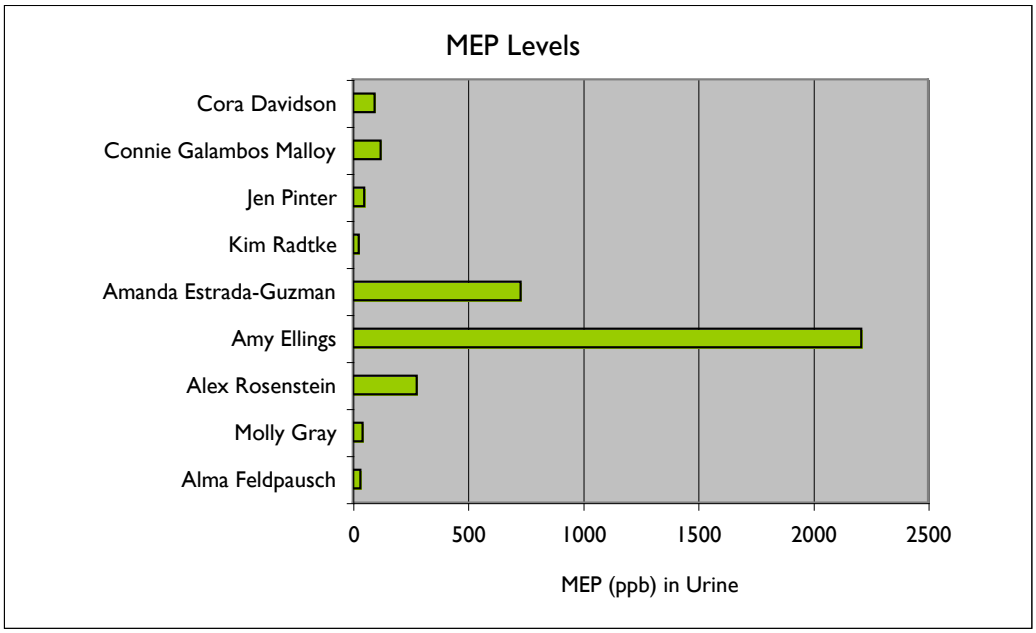
Amy Ellings didn't think she was dosing herself with phthalates when she soaped up in the shower—but her results showed a whopping 2210 ppb MEP in her urine, indicating exposure to the phthalate DEP. This level puts her above 90% of U.S. adults for levels of this phthalate, which is used primarily in fragranced personal care products. She was surpised and frustrated to find that her levels were relatively high despite her efforts to choose products carefully.

Of course, Amy can't be sure how she was exposed to DEP or any other phthalate—generally, they're not listed on product labels. "As far as I know, there is no way to easily tell if there are phthalates in shampoo, lotion, deodorant or other items, she said, and she's right. Manufacturers include

Phthalates are found in an impressive array of products including PVC/vinyl items such as shower curtains (6).

DEP in personal care products like shampoo. But pharmaceutical companies have also found it useful as an ingredient for coating some medications (*12*). For Amy and other U.S. residents, avoiding phthalates today is an impossible task.

Figure 1: Exposure of Nine Pregnant Women to the Phthalate DEP



Baby Boy Blues

The first and second trimesters are busy, busy times for the developing reproductive system. During the fifth week of gestation, male fetuses develop a penis and scrotum. During the ninth week, the uterus begins to form in female fetuses. The next week, girls take the first steps toward breast development, forming the ducts that might later carry milk to their children.

The pace doesn't let up much in the second trimester: week 14 brings formation of the urethra in male fetuses; week 18 starts the development and building up of eggs in girls' ovaries.

We collected samples from the women in this study during the second trimester, between 12 and 28 weeks of gestation. With the fetus in a frenzy of reproductive organ development at this time, exposure to hormone disrupting chemicals like phthalates is a real cause for worry, particularly for boys and men.

For example: evidence from laboratory studies links exposure to the phthalate BzBP during fetal development to decreased sperm production (*14*). Animals exposed in utero to DEHP or BzBP had smaller testes (*15*). DBP and DEHP exposure also resulted in reduced testosterone production before and shortly after birth, possibly the root cause of most if not all the reproductive problems seen with phthalate exposure (*16-18*).

Scientists don't know for sure whether phthalates have the same effect on people, but some evidence points in that direction. In her landmark study with pregnant women, Shanna Swan of the University of Rochester tested for exposure to phthalates during pregnancy. Among the 106 women that gave birth to boys, Swan found that the women with greater exposure to several phthalates were more likely to have baby boys with smaller penises, undescended testes, and other signs of feminization (*19*).

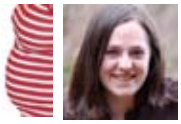
While these effects were seen in the more-exposed boys, the women whose sons had signs of feminization did not have exceptionally high phthalate levels during pregnancy. In fact, some of the women in our study had greater exposure to the phthalate DEHP than the women in Dr. Swan's study: considering the DEHP breakdown products we measured, two had higher levels of MEHP, five of MEOHP, and four of MEHHP. Looking at exposure to two other phthalates, all nine participants had MBP levels, and five had MBzP levels, higher than those of the mothers of affected boys in the Swan study. We can't draw any conclusions about the impacts of these levels, but they are certainly cause for concern.

We also tested the women in this study for levels of thyroid hormones because chemicals like phthalates have been shown to affect thyroid function in laboratory studies (*20*). Appropriate levels of thyroid hormones are key to brain and nervous system development, and levels that are too low can lead to learning disabilities as well as preterm birth and low birth weight (*21, 22*).

Thyroid hormone levels vary during pregnancy, and all of the women in our study had levels within normal ranges for pregnancy (*23*). However, we did see some indication that levels of phthalates may have impacted thyroid levels among our participants. In particular, the women in our study with higher levels of DEHP breakdown products had lower levels of the thyroid hormone T3 (Spearman correlation coefficient R = -0.71, p = 0.11). In a much larger study conducted in Taiwan, pregnant women with higher levels of the phthalate DBP had lower levels of the thyroid hormone T4 (*24*). Research has also found a relationship between thyroid hormone levels and DEHP exposure in American men (*25*). Clearly, further research is needed to investigate the impact of phthalates on thyroid hormone function.

Phthalates used as fragrance carriers can also be found in personal care products and cosmetics like nail polish and perfume (6).

Study Participant



Alex Rosenstein
Realtor
Issaquah, WA
Mother since
June 25, 2009
12 chemicals detected

"I figured that I'm a pretty healthy person, so how bad could it be? When I finally got the results, three months after the birth of my daughter, I was surprised. The levels were much higher than I expected them to be. And this is just from living what I consider to be a normal life."

Study Participant

Molly Gray
Midwife/
naturopathic
physician
Seattle, WA
Mother since
June 22, 2009
13 chemicals detected

"I do my best to live organic and chemical-free. Apparently, local/organic food only, toxin-free cleaners, off-gassed mattress, low/no VOC paint, and filtered water isn't enough. The answer I received from this study is that the fight is too big for just one person!"

Mercury

Tested for: total mercury in whole blood
Found: mercury in every participant

Though known to be harmful for centuries, mercury still finds a place in consumer products today, including fluorescent light bulbs, thermostats, medical equipment, and dental fillings. Coal-fired power plants are the primary source of emissions into the atmosphere, along with manufacturers, oil refineries, medical waste disposal facilities, and combustion of diesel, jet fuel, and heating oil (*26*).

Mercury from these sources and from mining makes its way into the food chain in waterways when bacteria convert elemental mercury into methylmercury, which then builds up in organisms including fish. U.S. fish are widely contaminated, with recent U.S. Geological Survey tests showing mercury in every freshwater fish sampled, and dangerous levels in 25% of fish tested (*27*). For most people, the primary source of mercury is eating contaminated fish, followed by dental fillings (*28*).

We tested our participants for total mercury in blood, which results primarily from methylmercury exposure in most people and is the measure used to date by the CDC (*6*). Figure 2 shows the results. The women in our study averaged a level of 1.04 ppb, slightly higher than the mean level of 0.92 ppb found in U.S. women tested by the CDC in 2005 and 2006 (*29*).

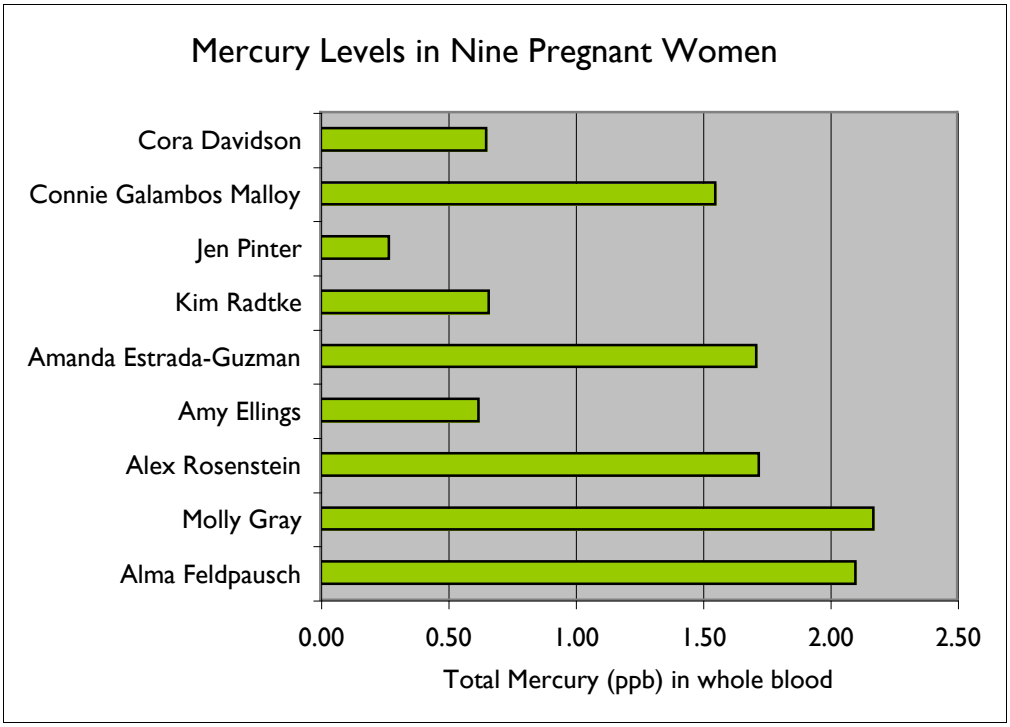
Mercury can harm adults, children, and fetuses. It demonstrated the magnitude of its power to harm the fetus during poisoning episodes like the one in Minamata, Japan, in the 1950s when apparently healthy women gave birth to severely disabled children (*30*). Mercury's primary target is the

nervous system, and exposure in the womb can cause defects in cognitive ability, memory, language, and motor skills. More severe effects can include vision problems and eventual blindness, seizures, lack of coordination, and hearing loss (*28*).

Mercury is considered a persistent toxic chemical because of its ability to build up in the body. When a woman is pregnant, some of the mercury passes through the placenta to the fetus, and methylmercury may build up to even higher concentrations in the blood of the fetus (*28*). Because mercury also contaminates breast milk, exposure can continue after birth.[1]

1 Despite contamination with toxic chemicals, physicians and experts continue to recommend breastfeeding because of the many risks of not breastfeeding, which include more ear and lung infections and greater chance of autoimmune illness. Breastfeeding provides optimal nutrition, important hormones, protective immune factors, and promoters of brain development.

Figure 2: Mercury Levels in Nine Pregnant Women



In the U.S. population, the severe effects listed above are extremely uncommon. What we are more likely to be seeing from everyday exposures are subtle effects, manifesting as a reduction in IQ caused by exposure before birth (*31*).

None of our study participants had mercury exposure above 5.8 ppb, which is the level the Environmental Protection Agency (EPA) has determined confers increased risk of health effects. The EPA has stated, however, that research has found no safe level in the range of study, which is as low as 1 ppb (*32*). Therefore, any exposure to mercury during fetal development is a concern, given the extensive evidence that this metal can cause learning disabilities.

At the same time, nutrients in fish—the primary source of mercury exposure—are believed to result in better brain development for fetuses. Health agencies now recommend that pregnant women continue to eat fish during pregnancy but choose low-mercury species based on research finding that babies born to mothers with higher fish consumption but lower mercury levels scored best in tests of cognition (*31, 33, 34*).

Study Participant

Alma Feldpausch
Environmental scientist
Seattle, WA
Mother since May 3, 2009
12 chemicals detected

"Being pregnant made me especially concerned about what I was being exposed to, from air pollution to chemicals in my food and water. If the levels of these test chemicals are considered unsafe–are known to result in adverse health impacts–then I would indeed expect our government agencies to work to reduce these chemicals."

Study Participant

Connie Galambos Malloy
Advocate for low-income communities
Oakland, CA
13 chemicals detected

"I was a bit anxious waiting for the results, as the waiting period coincided with the continued growth of my unborn child, through the delivery, and the child's first days of life. I consider myself very well informed on environmental issues and more conscious than most of what toxins I expose myself to on a regular basis; however, this study shows that my body has been invaded by toxins from all angles despite my efforts to the contrary."

PFCs – "Teflon Chemicals"

Tested for: 13 perfluorinated compounds, or PFCs
Found: both PFOS and PFOA in nine women, PFHxS in six women, and PFNA in three women

More than twenty years ago, two West Virginia babies were born with birth defects related to their eyes. But that wasn't the only thing they had in common—both of them had moms who worked in a DuPont™ chemical plant where they were exposed to PFOA, one of a complex class of chemicals known as perfluorinated compounds, or PFCs (*35*).

In 2009, nine West Coast babies were born after months-long exposures to PFOA and its chemical cousins in the womb. They may not have spent their formative months going to work with mom in a Teflon® factory, but somehow that didn't keep these highly persistent chemicals out of their mothers' bodies.

The mothers of the nine West Coast babies, the women in our study, tested positive for four different perfluorinated compounds in pregnant women, including PFOA (see Table 2 for summary information). None of these women had known sources of exposure to PFCs, so the chemicals likely entered their bodies through food, the dust in their homes, and direct contact with PFC-containing products (*36*).

PFCs are most famously used to make Teflon® and Scotchgard™, but manufacturers have developed a host of chemicals in this family to repel both oil and water from clothing, carpeting, furniture, and food packaging such as pizza boxes and fast-food containers (*37, 38*). Fire-fighting foams have also been a major use for decades, and other applications include cleaners, paints, roof treatments, and hardwood floor protectant (*38, 39*).

PFCs form a large family of chemicals, many of them breaking down to form PFOA and PFOS, two indestructible chemicals found in each of the women in our study. For the likely cancer-causing PFOA, levels were fairly consistent among participants, ranging from 1.49 ppb to 3.36 ppb in this study. Levels of PFOS, once the key ingredient in making Scotchgard™ (*40*), ranged more widely, with a low of 1.12 ppb and a high level of 11 ppb (see Figure 3). Interestingly, the trend in our group matched that of a much larger study of the U.S. population, in which older individuals had higher levels than younger people (*41*).

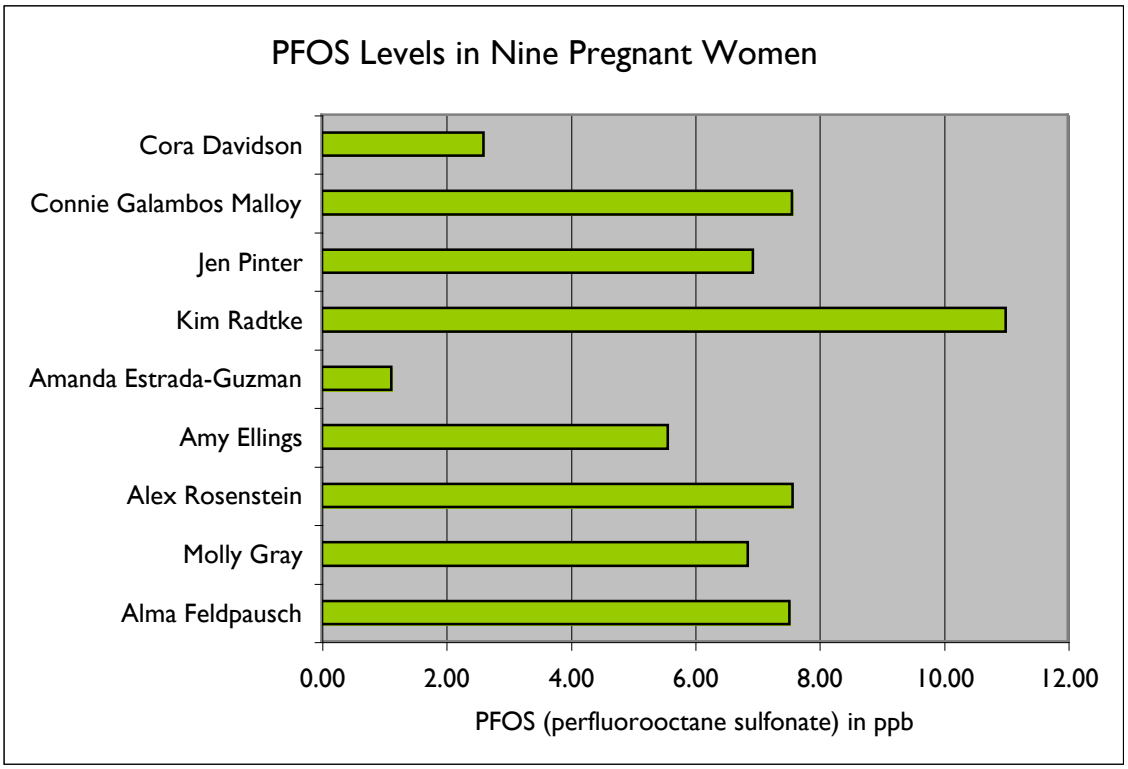
3M, the major manufacturer of PFOS, ceased producing the chemical in 2002 (*42*).
Though 3M no longer uses PFOS chemicals to make Scotchgard™ and other products, this highly persistent chemical achieved global distribution during its heyday (*43, 44*). In fact, in May 2009 PFOS was designated by the Stockholm Convention as a Persistent Organic Pollutant, where it keeps company with the likes of DDT and PCBs.

Table 2: PFC Levels in Nine Pregnant Women

Chemical (abbreviation)	Full Chemical Name	Study Mean	CDC Mean
PFOA	Perfluorooctanoic acid	2.24	3.9
PFNA	Perfluorononanoic acid	0.48	1
PFHxS	Perfluorohexanoic sulfonate	1.52	1.8
PFOS	Perfluorooctane sulfonate	5.36	18.7

Table 2 shows the geometric mean of four PFCs detected in our study, along with the geometric mean detected in 2,094 U.S. adults 20-39 years of age in a 2003-2004 CDC survey.

Figure 3: PFOS Levels in Nine Pregnant Women



Study Participant


Kim Radtke
Breastfeeding expert and advocate
Seattle, WA
Mother since October 31, 2009
11 chemicals detected

"Babies deserve to grow and develop in a healthy environment, in utero and out."

Non-stick toxic inheritance

Trained as a midwife, Kim Radtke has made a career out of helping babies get a good start in life. As the program manager for the Breastfeeding Coalition of Washington, she promotes breastfeeding-friendly policies in hospitals and workplaces. She has educated herself and many others on how children and pregnant women can avoid harmful chemicals. Despite her efforts, she had the highest PFOS level in the study group, perhaps because she is older than the other participants. Kim's reaction: "I'm surprised by my high PFOS result, but everyone should be angry about their body burden and especially angry that the most vulnerable, our unborn children, are exposed no matter how cautious we are before and during pregnancy."

Though PFOS is no longer manufactured, PFOA is still used to create Teflon® products and both chemicals are formed when other PFCs break down, contributing to ongoing contamination of people, wildlife, and the environment (*39*). Because these chemicals cross the placenta to reach the fetus, researchers also found them in more than 99% of 293 Maryland newborns tested in 2004 and 2005 (*45*). These researchers also found that children born with higher levels of PFOA and PFOS had lower birth weight, potentially increasing their risk of diabetes and obesity in adulthood. A larger study published at the same time found that babies whose mothers had higher levels of PFOA during pregnancy were born smaller (*46*).

More research is needed to confirm these findings, but they are consistent with other research using these chemicals. Laboratory studies find that pregnant animals exposed to PFOA or PFOS give birth to smaller young that grow and develop more slowly than non-exposed animals (*47*).

New research is also linking PFCs to obesity. In a study presented at the European Congress on Obesity in 2008, pregnant mice were exposed to PFOA (*48*). Though the exposed mice were smaller at birth, they grew to overweight adults.

One of the potential causes for this link may be effects on thyroid. Laboratory animals had lower levels of thryoid hormones when they were exposed to PFOS (*47*). This potential effect on thryoid creates a particular concern for exposure during pregnancy, though studies in humans have yet to find an association between PFCs and thyroid hormones (*47*).

Bisphenol A (BPA)

Study Participant


Amanda Estrada-Guzman
Nurse
Richland, WA
Mother since
July 16, 2009
11 chemicals detected

"The results were shocking and eye opening. There are too many toxins in the products I use on a daily basis. I was scared and worried how this will affect my baby."

Tested for: bisphenol A
Found: bisphenol A in every participant

It's produced in quantities of six billion pounds per year, making it one of the most popular chemicals of all time (*49*). As the building block for polycarbonate plastic, it's used in countless products in our homes, from the CDs in our stereo cabinets to our sports water bottles and Cuisinarts®. Yet most of us had never heard of bisphenol A until recently.

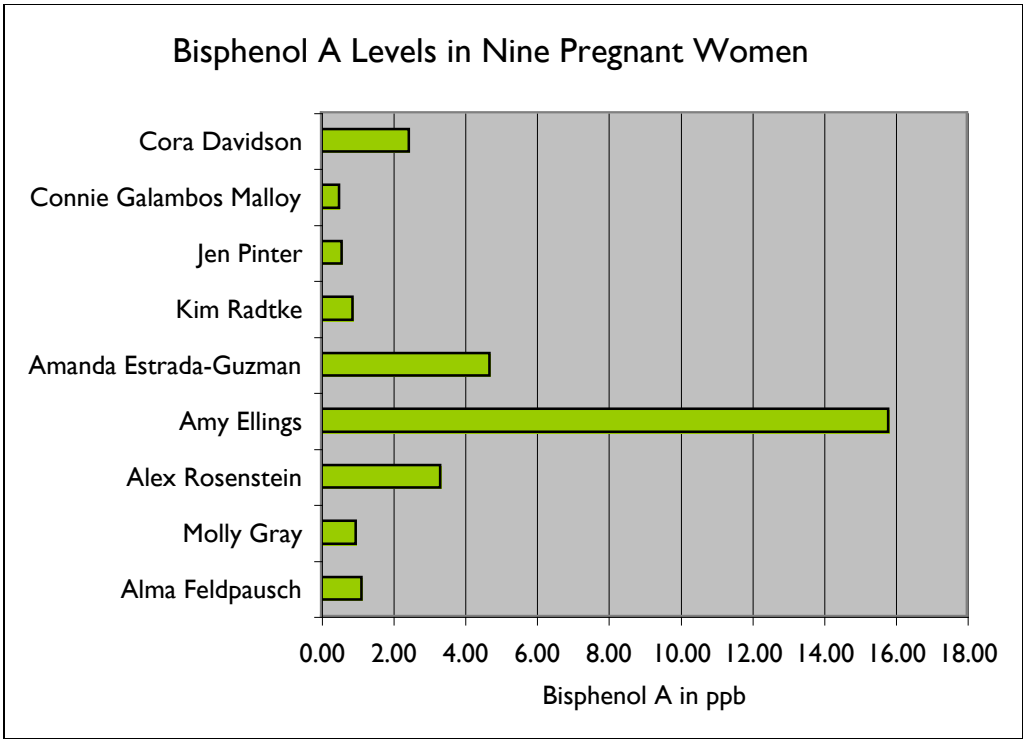
Bisphenol A started to make headlines in 2008 when the Canadian government took a hard look at the dozens of studies showing toxic effects at low doses and declared bisphenol A a hazardous substance. While Canadian scientists did not conclude definitively that the chemical will cause harm at current exposure levels, the government took action to protect infants and children from potential health effects (*50*). Canada also moved to ban baby bottles made of polycarbonate, prompting a number of major manufacturers to switch to alternate materials.

We tested pregnant women for bisphenol A because laboratory research shows it can have the most dramatic effects when exposure occurs during fetal development. All nine pregnant women in our study showed evidence of exposure to bisphenol A (see Figure 4 for levels).

Levels of chemicals like bisphenol A that are still present in consumer products can vary widely among individuals. Our data bear this out, with a more than 30-fold difference between the lowest level, in Connie Galambos Malloy, and the highest level, in Amy Ellings. Amy's level, in fact, is greater than that of 95% of Americans.

Each of us has reason for concern, with laboratory research indicating that health effects may be seen from bisphenol A at common exposure levels. Bisphenol A has been found in blood, urine, amniotic fluid, fetal blood, and breastmilk (*51-53*). The exposure of the women in our study likely came mostly from food—can linings commonly contain

Figure 4: Bisphenol A Levels in Nine Pregnant Women



bisphenol A—and may have come from drinking containers such as water bottles and pop cans, water pipes, dental work, carbonless papers, and dust in their homes contaminated with bisphenol A from household products (*49, 54, 55*).

Growing evidence suggests that the presence of bisphenol A in women's bodies during pregnancy could lead to diabetes or obesity, affect their children's brain development or ability to reproduce, or even cause cancer.

Developing brains: Laboratory studies have found that prenatal exposure to bisphenol A can change brain development, affecting a wide range of behaviors. In some cases, this means that animals exposed to bisphenol A don't show the normal differences expected between sexes (*56*). They also show more anxiety, hyperactivity, and aggression, and females lack normal maternal behavior, spending less time nursing their young (*56-58*).

Fertility: Bisphenol A is an estrogen mimic, so it is not surprising that it affects reproductive development in both males and females. In laboratory studies, female animals exposed *in utero* showed signs of early puberty (*59*). Male animals exposed in the womb produced less testosterone, had larger prostate glands, and made fewer sperm than unexposed animals (*56*).

Diabetes and Obesity: Two human studies have found a link between exposure to bisphenol A and obesity. In one investigation of 26 normal and obese women, the obese women had significantly higher levels of bisphenol A (*60*). In 2008, the Journal of the American Medical Association published a study of 1,455 individuals, finding that adults with greater exposure to bisphenol A were more likely to have diabetes and cardiovascular problems (*61*).

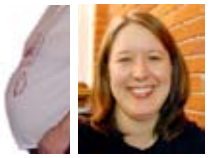
Cancer: Exposure to estrogen and related chemicals is known to increase the chance of developing breast cancer. The jury isn't yet in on whether bisphenol A belongs in this group, but a body of research has begun to paint a disturbing picture: in laboratory animals, exposure to bisphenol A before birth leads to changes in prostate and mammary development that raise the risk for cancer. Animals exposed to bisphenol A while in the womb had altered mammary gland development, and greater sensitivity to estrogen—and at puberty, they had higher levels of structures considered to be precursors to cancer (*62, 63*).
Some of the laboratory studies researching the effects of bisphenol A have been controversial because researchers have exposed animals through injections rather than orally, which would more closely parallel human exposure. While government agencies have struggled with the question of whether the exposure route makes a significant difference, independent research indicates that for very young animals, exposure route has no impact on blood levels of free bisphenol A (*64*).

We did not see any correlation between thyroid hormone levels and bisphenol A levels in this small study, but some studies have implicated bisphenol A as having potential to interfere with thyroid function. Two *in vitro* studies have found that bisphenol A interfered with the function of the thyroid hormone T3 (*65, 66*). As a result of this interference, the cells in the study produced less of the hormone prolactin, critical for lactation. A laboratory study also found thyroid disruption in the offspring of animals exposed to bisphenol A during pregnancy and lactation (*67*).

Dozens of studies are completed on bisphenol A each year, but we still have much to learn about how it could be affecting us. We are also still learning about how we are exposed to the chemical: a 2009 study of premature infants found that those with more medical treatment also had higher bisphenol A levels (*68*). These were the same infants who had higher exposure to phthalates, suggesting that the phthalate-containing medical equipment also contained bisphenol A.

In our study, women with greater exposure to phthalates also had higher levels of bisphenol A (see Figure 5 *next page*). Specifically, we found a moderately strong correlation between levels of bisphenol A and the phthalate MEP (Spearman correlation coefficient = 0.63, p=0.07). It is possible that the women in our study were exposed to both of these chemicals from the same types of products. To our knowledge, correlations between exposures to bisphenol A and various phthalates have never been described in the general population, but researchers and policymakers should consider this potential in the future as studies attempt to identify impacts of commonly-encountered exposures to these chemicals.

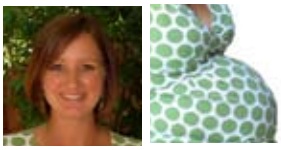
Study Participant



Cora Davidson
Reproductive rights advocate
Milton-Freewater, OR
(Walla Walla, WA area)
Mother since October 10, 2009
11 chemicals detected

"I'm a pretty healthy person and have had a very healthy pregnancy. However, knowing what I know now, it will give me pause when I go to purchase a new product – can I find one with fewer toxins or in safer packaging?"

Study Participant



Jen Pinter
Engineering project manager
Livermore, CA
Mother since September 27, 2009
12 chemicals detected

"Hopefully, when our baby boy is an adult, these consumer products that are currently filled with toxins that are affecting people's lives will have changed and our son's family will not have to worry about such problems."

Figure 5: Comparison of MEP and BPA Levels in Nine Pregnant Women

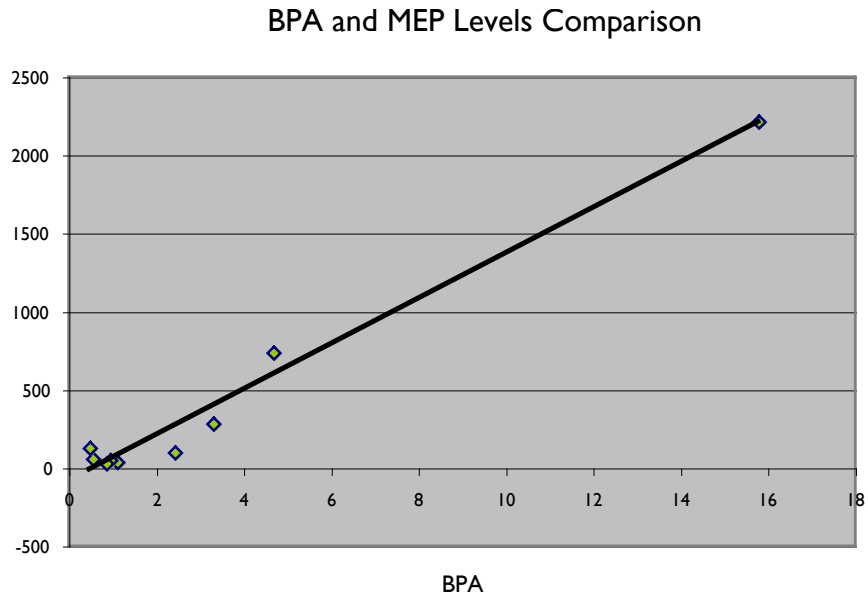


Figure 5 shows the correlation between levels of bisphenol A and the phthalate breakdown product in nine pregnant women. Each point represents one individual's levels of the two chemicals.

Tetrabromobisphenol A (TBBPA)

Tested for: TBBPA in blood serum
Found: no detections in nine women

Tetrabromobisphenol A, or TBBPA, is an example of a group of chemicals known as brominated flame retardants. These are compounds used to slow fire in a broad range of products, from building materials to furniture and electronics. TBBPA is the brominated flame retardant produced in the highest volume, and is used primarily in electronics and electrical equipment, with about 20% added to plastics (*69, 70*).

Though not particularly well studied, TBBPA may have even greater usage in the future as PBDEs, or polybrominated diphenyl ethers, are phased out. We decided to include TBBPA in our study because initial studies indicate it may affect thyroid hormone balance and depress immune response (*71*). Laboratory studies have also developed some evidence that exposure may affect learning and behavior, although results have been mixed (*72*).

We did not detect TBBPA in the pregnant women in our study at the level of detection achieved by the laboratory we used. Previous studies in Europe have detected TBBPA in human blood, but with apparently more sensitive laboratory techniques (*73, 74*). A recently published French study tested 93 mother-newborn pairs for TBBPA, and found the chemical in about 30% of the samples, with a high degree of variation in levels (*75*). With the differences in analytical methods, we cannot draw a conclusion from this study on whether this chemical is present or absent in the blood of U.S. pregnant women, and more study is needed to investigate its potential presence.

Detox Paradox

While we certainly have much to learn about precisely what the fetus experiences, we now know that far from serving as a protective barrier, the placenta allows many foreign substances to cross, mostly by simple diffusion (*76*).

But research indicates that the fetus possesses only a small proportion of the adult's ability to detoxify foreign chemicals. A subset of enzymes known as the cytochrome P450 enzymes, for example, form a key part of the adult's treatment of drugs, toxic chemicals, and self-made substances like hormones. In the fetus, however, a different subset of these enzymes is responsible, and even these are absent in the very early days of fetal development (*77*). One enzyme, for instance, appears at 16 to 24 weeks of gestation and can transform ethanol, but only at 12% to 27% of the adult rate.

Another of the body's main tools for detoxification—and the one responsible for the conversion of phthalates and bisphenol A, chemicals of major concern—is known as glucuronidation. As with the cytochrome P450 system, the enzymes responsible for glucuronidation appear at different times during development. Researchers testing for the activity of these enzymes in the human fetus found it totally missing at 20 weeks gestation (*78*). Even in young children, this detoxification pathway is completely underpowered. Children between one and a half years and two years old had one third to one fortieth the ability to detoxify chemicals using this pathway, depending on the chemical.

Of course, toxic chemicals must first pass through the mother to reach the fetus. But everything changes during pregnancy: blood volume increases 50%; it takes longer for the stomach to empty; kidney function changes; and the level of plasma proteins declines (*79*). These changes serve to maximize delivery of nutrients to the fetus and quickly remove its wastes. At the same time, they affect how the mother takes in and breaks down toxic chemicals.

Some evidence indicates that toxic chemicals can in fact quickly pass from the mother to fetus and actually accumulate at higher levels in the fetus. In one laboratory experiment, researchers found bisphenol A in fetal tissue just 20 minutes after pregnant animals were exposed (*80*). Fetal levels of bisphenol A in laboratory animals were higher than levels in maternal blood less than an hour after exposure. A second study using a low dose also found rapid transfer of bisphenol A to the fetus (*81*).

Besides the underdevelopment of detoxification mechanisms in the fetus, changes in the metabolism during pregnancy may be responsible for the build up of chemicals in the fetus. For some chemicals, hormonal changes during pregnancy actually hasten their elmination (*82*). However, a key laboratory study found that for some toxic chemicals, detoxification via glucuronidation works at about half the normal rate, allowing those chemicals to build up in the fetus. This appears to be the case for the glucuronidation enzyme responsible for bisphenol A and other estrogen mimics including nonylphenol and the infamous DES (*83*). Clearly, this is only one in a complex array of factors that determine what happens when a pregnant woman is exposed to a toxic chemical. Further research is needed to determine the extent to which changes during pregnancy in fact result in greater fetal exposure to toxic chemicals.

"..toxic chemicals can in fact quickly pass from the mother to fetus and actually accumulate at higher levels in the fetus."

We Can Do Better to Protect Children

"..in this country, we operate under a toxics law that has required testing of only 200 chemicals out of 80,000 produced since the law was passed in 1976."

Babies are born after nine months of protection and nurturing from their mothers, and nine months of toxic chemicals from the rest of the world. Babies born in the United States can blame the government: in this country, we operate under a toxics law that has required testing of only 200 chemicals out of 80,000 produced since the law was passed in 1976 (*84*). The Toxic Substances Control Act (TSCA), meant to keep chemicals that can harm our health out of the products we buy, has turned out to be a toothless framework that has failed to protect our health.

One of TSCA's most problematic elements is its grandfathering of the 62,000 chemicals already in production at the time of its passage: Congress protected these chemicals from regulation, and today they make up 92% of the chemicals used in the highest quantities in the U.S. (*85*). For any chemical introduced after that time, only notification, not approval, is needed before production can start. Once chemicals are in production, the federal government can only stop their use, even if they are found to be toxic, if it can jump over incredible hurdles—hurdles so high it couldn't even ban asbestos when it tried in 1989.

Where the federal government has held out a hollow shell, states have moved in to fill the gap. In 1998, Washington state made a bold move with its creation of a program to eliminate persistent toxic chemicals like mercury, the toxic flame retardants PBDEs, and lead. In the years following, the Washington legislature has followed suit with groundbreaking legislation to get mercury and PBDEs out of consumer products.

In 2007, parents and legislators throughout the country learned just how little the federal government was doing to keep consumer products safe when toy manufacturers recalled millions of toys for lead contamination. On the heels of this discovery, in 2008 legislatures in four states passed major laws designed to get toxic chemicals out of toys and other products.

Washington: passed the Children's Safe Products Act, banning lead, cadmium, and phthalates in toys and other children's products. Similar to Maine's law, the Act also requires the state to develop a list of high priority chemicals based on harm to the child or developing fetus. Manufacturers of children's products will then be required to disclose the levels of these chemicals in their products.

California: passed its Green Chemistry Initiative, which takes broad action to address toxics in consumer products. Under this law, the state must create a process to identify and prioritize chemicals of concern. Once it does so, it has authority to require additional information, take action to reduce exposure, restrict or ban the chemical, or fund development of safer alternatives.

Maine: passed the Kid Safe Products Act to replace chemicals that can harm children or developing fetuses with safer alternatives. In July 2009, Maine made public its list of toxic substances that can disrupt hormones, cause learning problems, cancer or reproductive harm, or that persist in people and the environment. The list includes bisphenol A, some phthalates, PFCs, and toxic flame retardants. Once the state designates priority chemicals, manufacturers of consumer products will be required to disclose which of these chemicals are in their products. The Act also gives the state the authority to require manufacturers to replace toxic chemicals with safer substitutes when they are available.

Connecticut: passed the Act Concerning Child Product Safety, banning lead and asbestos in children's products. The Act also requires the state to create a list of toxic chemicals that may create a hazard in children's products, and a list of safer alternatives to those chemicals.

Several common threads emerge from this legislation passed in four states. One, states are taking action on some hazardous chemicals already known to be present in products. Two, states are identifying and prioritizing chemicals that can cause cancer, learning or reproductive problems, disrupt hormones, or persist in people and the environment. Three, states are requiring disclosure of the presence of these chemicals and finding ways to replace them with safer substitutes.

Finding a vacuum at the federal level, these states are essentially inventing at the state level a common-sense, workable approach to getting toxic chemicals out of consumer products. State governments are working together, having created an interstate clearinghouse on chemicals and safer alternatives. In doing so, they are actively making changes to protect the health of children and families in their states.

This state action is beginning to inspire members of Congress to create change that will benefit people in all states. Members are developing legislation which will reform the Toxic Substances Control Act and give the federal government the ability to police the safety of consumer products.

Several states have made a strong start in creating policies that will ensure only the safest chemicals are used in consumer products. At the same time, the results of our study make clear that further action is urgently needed.

We recommend the following actions for state and federal governments:

1. Pass policies that protect the most vulnerable. We need policies that keep toxic chemicals away from pregnant women and the developing fetus by doing the following:

- Immediately initiate action to eliminate the use of persistent toxic chemicals, which are those that build up in our bodies or are passed on to the next generation.
- Reduce the use of chemicals that can cause serious health problems such as cancer and reproductive harm, can disrupt the normal function of hormones, or can lead to learning disabilities.
- Allow manufacturers to create consumer products using only chemicals they have tested fully for safety and that do not cause cancer, reproductive harm, disrupt hormones, or cause learning disabilities.

2. Hold industry responsible for testing chemicals and providing full information on their hazards. Chemical manufacturers should test chemicals and provide full information on their hazardous properties and potential impact on health and the environment. The public, workers, and businesses have a right to know what possible harms might result from these chemicals, and health and environmental agencies need this information to make the right decisions to protect health.

3. Maintain the ability of states to set the highest standards to protect health. States are proving that they respond to the need to protect public health with strong, sensible policies. That ability to respond must be maintained, with enhanced coordination between state and federal governments and between federal agencies. Specifically, new federal laws must preserve the rights of the states to enact legislation that is more protective than federal law.

Safer Chemicals Healthy Families

A nationwide effort to pass smart federal policies that protect us from toxic chemicals.

Organizations from around the country have banded together as the Safer Chemicals, Healthy Families campaign to win reform at the federal level. The campaign has established this platform to guide its efforts.

A Platform for Reform of the Toxic Substances Control Act

A reformed Toxic Substances Control Act (TSCA) would serve as the backbone of a sound and comprehensive chemicals policy that protects public health and the environment while restoring the luster of safety to U.S. goods in the world market. Any effective reform of TSCA should:

Immediately Initiate Action on the Worst Chemicals: Persistent, bioaccumulative toxicants (PBTs) are uniquely hazardous. Any such chemical to which people could be exposed should be phased out of commerce. Exposure to other toxic chemicals, like formaldehyde, that have already been extensively studied, should be reduced to the maximum extent feasible.

Require Basic Information for All Chemicals: Manufacturers should be required to provide basic information on the health hazards associated with their chemicals, how they are used, and the ways that the public or workers could be exposed.

Protect the Most Vulnerable: Chemicals should be assessed against a health standard that explicitly requires protection of the most vulnerable subpopulations. That population is likely to usually be children, but it could also be workers, pregnant women, or another vulnerable population.

Use the Best Science and Methods: The National Academy of Sciences' recommendations for reforming risk assessment at the Environmental Protection Agency (EPA) should be adopted. Regulators should expand development and use of information gleaned from "biomonitoring," the science of detecting human chemical contamination, to inform and impel efforts to reduce such exposures.

Hold Industry Responsible for Demonstrating Chemical Safety: Unlike pharmaceuticals, chemicals are currently presumed safe until proven harmful. The burden of proving harm falls entirely on EPA. Instead, chemical manufacturers should be responsible for demonstrating the safety of their products.

Ensure Environmental Justice: Effective reform should contribute substantially to reducing the disproportionate burden of toxic chemical exposure placed on people of color, low-income people and indigenous communities.

Enhance Government Coordination: The EPA should work effectively with other agencies, like FDA, that have jurisdiction over some chemical exposures. The ability of the states to enact tougher chemical policies should be maintained and state/federal cooperation on chemical safety encouraged.

Promote Safer Alternatives: There should be national support for basic and applied research into green chemistry and engineering, and policy should favor chemicals and products that are shown to be benign over those with potential health hazards.

Ensure the Right to Know: The public, workers, and the marketplace should have full access to information about the health and environmental hazards of chemicals and the way in which government safety decisions are made.

www.saferchemicals.org

Appendices and References

Appendix 1: Detailed Results

Compound	Medium tested	Full Name/Explanation	Participants: Alma Feldpausch	Molly Gray	Alex Rosenstein	Amy Ellings	Amanda Estrada-Guzman	Kim Radtke	Jen Pinter	Connie Galambos Malloy	Cora Davidson	Geometric Mean
Mercury (µg/L, or ppb)	**whole blood**		2.10	2.17	1.72	0.62	1.71	0.66	0.27	1.55	0.65	1.05
Perfluorinated Compounds (ng/mL, or ppb)	**serum**											
PFBA		Heptafluorobutyric acid	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	n/a
PFPeA		Perfluoropentanoic acid	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	n/a
PFHxA		Perfluorohexanoic acid	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	n/a
PFHpA		Perfluoroheptanoic Acid	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	n/a
PFOA		Perfluorooctanoic Acid	2.27	2.14	1.94	2.05	3.03	1.57	3.36	3.10	1.49	2.24
PFNA		Perfluorononanoic Acid	< 0.500	0.54	< 0.500	<0.771	< 0.500	< 0.500	1.14	0.75	< 0.500	0.48
PFDA		Perfluorodecanoic acid	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	n/a
PFUnA		Perfluoroundecanoic acid	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	n/a
PFDoA		Perfluorododecanoic acid	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	n/a
PFBS		Heptafluorobutyric sulfonate	< 1.00	< 1.00	< 1.00	< 1.00	< 1.00	< 1.00	< 1.00	< 1.00	< 1.00	n/a
PFHxS		Perfluorohexanoic sulfonate	2.42	3.36	2.11	1.81	< 1.00	1.92	< 1.00	2.02	< 1.00	1.52
PFOS		Perfluorooctane sulfonate	7.52	6.85	7.57	5.57	1.12	11.00	6.94	7.56	2.60	5.36
PFOSA		Perfluorooctanesulfonamide	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	< 0.500	n/a
Phthalates (ng/mL, or ppb)	**urine**											
m-MeP (MMP)		Metabolite of DMP (dimethyl phthalate) - used in hair-care products, solid rocket propellant, insect repellents, and plastics.	2.83	8.07	3.95	8.33	6.11	<3.57	3.48	2.94	10.40	4.75
m-EtP (MEP)		Metabolite of DEP (diethyl phthalate) - found in personal care products such as perfume, cologne, aftershaves, deodorants, shampoo, and hand lotion.	32.10	43.80	277.00	2210.00	730.00	25.80	50.80	121.00	94.80	128.34
m-BuP (MBP)		Metabolite of DBP (dibutyl phthalate) - found in personal care products such as nail polish and in pharmaceuticals.	33.90	76.70	32.80	40.10	345.00	35.40	32.30	34.70	30.50	48.29
m-BzP (MBzP)		Metabolite of BzBP (benzylbutyl phthalate) - found in vinyl flooring, car-care products, personal-care products, adhesives, and sealants.	5.62	22.90	14.90	13.50	71.40	5.36	8.29	16.70	6.48	12.75
m-EHP (MEHP)		Metabolites of DEHP (di-(2-ethylhexyl) phthalate) - found in PVC products including medical products such as tubing; auto interiors; consumer products such as clothing, diaper covers, shower curtains, and furniture.	6.63	9.85	5.57	5.50	5.52	7.66	4.45	3.58	1.62	5.06
mEOHP (MEOHP)			22.50	38.30	17.40	22.90	22.00	48.80	19.30	15.10	7.23	20.95
mEHHP (MEHHP)			35.70	49.10	28.10	30.00	25.10	68.90	28.40	19.60	12.60	29.69
Phthalates, Creatinine Adjusted Levels (µg/g)												
Creatinine (mg/dL)			74.00	164.00	102.00	77.00	89.00	84.00	55.00	55.00	62.00	
m-MeP (MMP)			3.82	4.92	3.87	10.82	6.87	<4.25	6.33	5.35	16.77	6.47
m-EtP (MEP)			43.38	26.71	271.57	2870.13	820.22	30.71	92.36	220.00	152.90	160.50
m-BuP (MBP)			45.80	46.77	32.16	52.08	387.64	42.14	58.73	63.09	49.19	60.39
m-BzP (MBzP)			7.59	13.96	14.61	17.53	80.22	6.38	15.07	30.36	10.45	15.94
m-EHP (MEHP)			8.96	6.01	5.46	7.14	6.20	9.12	8.09	6.51	2.61	6.33
mEOHP (MEOHP)			30.41	23.35	17.06	29.74	24.72	58.10	35.09	27.45	11.66	26.20
mEHHP (MEHHP)			48.24	29.94	27.55	38.96	28.20	82.02	51.64	35.64	20.32	37.13
Bisphenol A (ng/mL, or ppb)	**urine**											
Bisphenol A			1.13	0.96	3.32	15.80	4.70	0.88	0.57	0.50	2.44	1.76
Bisphenol A, Creatinine Adjusted Levels (mg/g)			1.53	0.59	3.25	20.52	5.28	1.05	1.04	0.91	3.94	2.20
TBBPA (ng/mL, or ppb)	**serum**		< 10.0	< 10.0	< 10.0	< 10.0	< 10.0	< 10.0	< 10.0	< 10.0	< 10.0	n/a

Appendix 2: Detailed Methods

Participants were recruited via health organizations and personal contacts. Each participant was asked to complete an exposure assessment questionnaire, to provide information about residences, occupations, diet, and potential toxic exposures. Samples were taken between March and May 2009. A nurse collected blood samples in vacuum tubes; after clotting, serum was obtained by centrifuging tubes and pouring off into storage vials. One vacuum tube of whole blood was reserved for each participant for mercury testing. Participants provided first morning void urine samples for phthalate and bisphenol A testing. Whole blood was cooled until delivery to the laboratory. Serum and urine samples for chemical analysis were frozen immediately and shipped after at least 24 hours. Analysis for bisphenol A, PFCs, TBBPA, and phthalates was performed by AXYS Analytical Services, Victoria, BC. Analysis for total mercury was performed by Brooks Rand Labs, Seattle, WA. Analysis for thyroid hormones was performed by LabCorp in Seattle, WA, and San Francisco, CA.

For calculation of geometric means and other data analysis, the limit of detection divided by the square root of two was used where the analyte was not detected.

Laboratory analytic methods are described below.

Perfluorinated Compounds (PFCs)

The sample was spiked with surrogate standards. Three ml of 50% formic acid were added and the mixture sonicated for 20 minutes. Cleanup was performed by solid phase extraction using a disposable cartridge containing a weak anion exchange sorbent. The eluate is spiked with recovery standards and analyzed by LC-MS/MS. Calibration solutions were prepared in bovine serum and processed through the same cleanup procedure. Analysis of sample extracts by HPLC-MS/MS was performed on a high performance liquid chromatograph coupled to a triple quadrupole mass spectrometer. The MS was run at unit mass resolution in the Multiple Reaction Monitoring mode. Target compounds were quantified using the internal standard method, comparing the area of the quantification ion to that of the ^{13}C-labeled standard and correcting for response factors.

Tetrabromobisphenol A (TBBPA)

The sample was spiked with surrogate standards. Three ml of formic acid were added and the mixture sonicated for 20 minutes. Cleanup was performed by solid phase extraction using a disposable cartridge containing a weak anion exchange sorbent. The eluate was spiked with recovery standards and analyzed by LC-MS/MS. Samples were co-extracted with PFCs, but two separate vials were submitted to instruments. Analysis by HPLC-MS/MS was performed on a high performance liquid chromatograph coupled to a triple quadrupole mass spectrometer. The MS was run at unit mass resolution in the Multiple Reaction Monitoring (MRM) mode. Initial calibration of the LC-MS/MS instrument was performed by the analysis of six calibration solutions. A mid-level calibration standard was analyzed to verify the initial calibration after every 20th sample (including QC samples) injected at a minimum.

Phthalate Monoesters and Bisphenol A

One ml urine samples were spiked with a suite of isotopically labeled surrogate standards and with 4-methylumbelliferyl glucuronide solution as an indicator for monitoring the deconjugation of glucuronidated forms of the analytes. The deconjugation is performed with glucuronidase enzyme at 37° C. BPA and phthalate ester metabolites were co-extracted from a single sub-sample of urine. Extraction and cleanup were performed by solid phase extraction on a hydrophilic-lipophilic balance sorbent cartridge. The analytes were eluted with methanol. The extract was spiked with recovery standards before proceeding to HPLC-MS/MS. Instrumental analysis of the sample extract was performed using a high performance liquid chromatograph coupled to a triple quadrupole mass spectrometer. Separate instrumental analysis runs using different instrument operating conditions were required for determination of BPA and for phthalate ester metabolites. Final concentrations were determined by isotope dilution quantification procedure.

Mercury

Mercury in whole blood was analyzed using a modification of *Method 1631, Appendix: Total mercury in tissue, sludge, sediment, and soil by acid digestion, BrCl oxidation, and cold vapor atomic fluorescence spectrophotometry*. Prior to analysis, the samples were digested in a combination of nitric acid and sulfuric acid and then heated to break down the sample matrix. They were then oxidized with bromine monochloride (BrCl) to convert all mercury species to mercuric ions. The method is a cold vapor atomic fluorescence technique, based upon the fluorescence of 253.7 nm radiation by excited elemental mercury atoms in an inert gas stream. Mercuric ions in the oxidized sample were reduced to HgO using stannous chloride ($Sn\ Cl_2$), and then preconcentrated onto gold amalgamation traps using nitrogen gas as a means of preconcentration. Analysis proceeded with cold vapor atomic fluorescence spectroscopy detection, as follows. Mercury vapor was thermally desorbed into the fluorescence cell via heat and an inert gas stream. The mercury atoms were excited by a fluorescence wavelength of 253.7 nm. Fluorescence intensity was measured as a function of total mercury collected, which was converted to concentration by the size of the aliquot purged.

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