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CURRENT SCIENCE ON PUBLIC EXPOSURES TO TOXIC CHEMICALS

UNITED STATES SENATE, COMMITTEE ON ENVIRONMENT AND PUBLIC WORKS,

SUBCOMMITTEE ON SUPERFUND, TOXICS AND ENVIRONMENTAL HEALTH

ONE HUNDRED ELEVENTH CONGRESS, SECOND SESSION

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Henry Falk *[view pdf]*
Acting Director, National Center for Environmental Health
Agency for Toxic Substances and Disease Registry
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Statement of Frank R. Lautenberg
Hearing: Subcommittee on Superfund, Toxics and Environmental Health hearing entitled, "Current Science on Public Exposures to Toxic Chemicals."
Thursday, February 4, 2010

(Remarks as Prepared for Delivery)

WASHINGTON, D.C. – Senator Frank R. Lautenberg (D – NJ) delivered the following opening statement at a morning hearing of the Environment and Public Works subcommittee he chairs to examine the current science on public exposure to toxic chemicals.

"Let me thank everyone for being here as we focus on better protecting the health of our families updating our chemical safety laws.

There's no question that chemicals are essential to our modern lives.

They are used in household cleaners to kill germs. They are used in medical equipment that sav lives. They even help fight global warming by creating insulation for homes, better components f wind turbines and additives to make fuels cleaner.

But when we use these products, the chemicals in them can end up in our bodies. So in essenc American public has become a living, breathing repository for chemical substances.

And when the chemicals used in flame retardants, plastics or rocket fuel show up in our children bodies, we have a potentially dangerous situation.

We can trace this problem back to the current law that governs the safety of chemicals. That law Toxic Substances Control Act, or TSCA – fails to give EPA the tools it needs to protect against ur chemicals.

In fact, the Government Accountability Office has identified our current law as a "high risk" area o law.

In nearly 35 years, TSCA has allowed EPA to test only 200 of the more than 80,000 chemicals in products we use every day.

What's more, the EPA has been able to ban only five substances on EPA's inventory of chemica the market.

With EPA unable to require adequate testing, our children have become test subjects.

And we are seeing the results in a dramatic increase in childhood cancers, birth defects and hor problems across the population.

Studies have found that as much as five percent of cancers, ten percent of neurobehavioral diso and 30 percent of asthma cases in children are associated with hazardous chemicals.

Our children should not be used as guinea pigs. So it's time to update the law and protect them

Led by our friend from New Jersey Lisa Jackson and Assistant Administrator Steve Owens, who here with us today, the Environmental Protection Agency has taken steps to try to reduce the ris from chemicals.

But they cannot protect our children with one hand tied behind their back.

That's why I will soon introduce a bill that will overhaul our nation's chemical laws.

My safer chemicals bill will have a simple goal: force chemical makers to prove that their product safe before they end up in a store, in our homes, or in our bodies.

We already regulate pesticides and pharmaceuticals this way – it's just common sense that we c same for chemicals that are used in everyday consumer products.

Everyone from chemical manufacturers, to businesses that use chemicals in their products, to environmental, labor and health groups have called for reforming our chemical laws.

We cannot waste this opportunity.

I will be reaching out in the coming weeks to our colleagues – Republicans and Democrats alike support my safer chemicals bill.

This is a problem that affects us all, and we should all be committed to working on the solution.

# # #

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Statement of Barbara Boxer
Hearing: Subcommittee on Superfund, Toxics and Environmental Health hearing entitled, "Current Science on Public Exposures to Toxic Chemicals."
Thursday, February 4, 2010

(Remarks as prepared for delivery)

I would like to thank Senator Lautenberg for holding this hearing on the public health threats po by exposure to toxic chemicals.

The fact is that every one of us – and every man, woman and child in every state in our nation -- exposed to toxic chemicals.

The Centers for Disease Control and Prevention's fourth national biomonitoring report, which car out in December 2009, paints a chilling picture. This report measured 212 chemicals in people's bodies -- 75 of which had never before been measured in the U.S. population. The new chemica included bisphenol A and perchlorate.

The CDC found that 90% of those sampled were exposed to bisphenol A (BPA), a chemical usec make plastics and epoxies -- the National Toxicology Program has stated that BPA has "some cc for effects on the brain, behavior, and prostate gland in…infants and children at current [levels c human exposures to bisphenol A."

And the CDC found that 100% of the people they sampled -- every single one -- had detectable of perchlorate, a component of rocket fuel that can harm the development of the nervous system that poses special dangers for pregnant women and children.

When EPA Administrator Jackson appeared before the Environment and Public Works Committe December 2009 to discuss the Administration's principles for reforming the nation's toxic chemica regulation laws, she delivered a serious message. I want to quote from her testimony:

"The public is turning to government for assurance that chemicals that are ubiquitous in our eco our environment and our bodies have been assessed using the best available science, and that unacceptable risks have been eliminated. But, under existing law, we cannot give that assurance Restoring confidence in our chemical management system is a top priority for me and a top environmental priority for the Obama Administration."

The Government Accountability Office, an independent, nonpartisan agency, has put EPA's che regulation program on its "High Risk" programs because "EPA has failed to develop sufficient chemical assessment information to limit public exposure to many chemicals that may pose substantial health risks."

I agree with Administrator Jackson – fixing the nation's flawed system for regulating toxic chemic must be a top priority and is in this Committee.

We have a responsibility to America's families to ensure that the chemicals in the environment ar the products they use every day have been scientifically tested and that they and their children a not put at risk.

This Committee has the opportunity to strengthen our nation's toxics laws to ensure that evaluati on the safety of chemicals are made based on science and public health and that all people - especially the most vulnerable - are protected.

I look forward to hearing from all of our witnesses today, to get their views on the importance of t best ways to address the potential health risks from exposure to dangerous chemicals.

These hearings are part of our larger effort to reform our toxics laws and I am working closely wit Senator Lautenberg to craft legislation to address these issues.
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Statement of James M. Inhofe
Hearing: Subcommittee on Superfund, Toxics and Environmental Health hearing entitled, "Current Science on Public Exposures to Toxic Chemicals."

Thursday, February 4, 2010

Opening Statement of Senator James M. Inhofe, Ranking Member

Thank you, Chairman Lautenberg, for holding this hearing on the state of the science of human exposures t chemicals. My understanding is that this is the first in a series of hearings leading up to a legislative debate c revision of the Toxic Substances Control Act (TSCA). I welcome the opportunity to discuss the strengths ar weaknesses of the law and the science surrounding it.

Today we will hear perspectives on scientific approaches for evaluating human exposures to chemicals. In particular, I am interested in the discussion related to biomonitoring – one of the scientific techniques used assessing human exposures to natural and synthetic compounds in the environment.

I believe that biomonitoring can be a useful tool in assessing human chemical exposures. But, biomonitoring its limits, as it provides only information on exposure; it does not provide dose information. Simply put, the presence of a substance in the body, at any level, cannot be interpreted to mean that adverse effects will oc

I hope the witnesses here today remain objective in their discussions of biomonitoring, and avoid the tempta to rely on detection as a surrogate for risk. Misapplying biomonitoring data only serves to scare the public a some cases, advance political agendas. By invoking notions of "body burden" and "chemical trespass," peo who do not understand the limitations of biomonitoring are encouraged to reduce exposures to some substa that may increase, rather than decrease, their overall health risks. A perfect example is mothers refraining f breast feeding in order to avoid feeding their babies chemicals found, or that may be found, in breast milk. almost all circumstances, the benefits of breast feeding exponentially outweigh any possible risks from the presence of a chemical in the milk. This same advice is given to nursing mothers by public health authoritie

For over 30 years, TSCA has provided a scientifically sound framework for reporting, testing, tracking and restricting chemical substances and mixtures. As I have stated before, I am open to the idea of modernizing statute. But, to the proponents of radical reform and supporters of the precautionary principle, let me be ve clear: my principles for any regulatory or statutory changes to TSCA must be based on the best available sc including risk assessment; must include cost-benefit considerations; must protect proprietary information; a must prioritize reviews for existing chemicals. Further, I will not support changes that encourage litigation, for activist enforcement, or that compel product substitution.

I look forward to hearing from the witnesses here today and to the upcoming debate on how best to modern TSCA.

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Testimony of Steve Owens
Assistant Administrator
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U.S. Environmental Protection Agency
before the
Subcommittee on Superfund, Toxics, and Environmental Health
Committee on Environment and Public Works
United States Senate
February 4, 2010

Chairman Lautenberg, Ranking Member Inhofe, and other members of the subcommittee, thank you for the opportunity to discuss exposure to toxic chemicals and the need for reform of this nation's chemicals management program.

As this Committee knows, EPA's mission is to protect human health and the environment. Ensuring that our citizens, and especially our children, are protected from exposure to unsafe levels of toxic chemicals and pollution or other environmental threats in their homes, schools, or communities is central to EPA's work. Simply put, protecting people from the adverse health effects that result from exposure to harmful chemicals is our job. As EPA Administrator Lisa Jackson recently testified before this committee, the public expects the government to provide assurance that chemicals which are ubiquitous in our economy, our environment, and our bodies have been assessed, using the best available science, and that unacceptable risks have been eliminated. Restoring confidence in our chemical management system is a top priority for EPA and a top environmental priority for the Obama Administration.

Chairman Boxer and Chairman Lautenberg, we stand ready to work with you and other Members of this committee to improve the safety of chemicals and restore the public's confidence in effective chemicals regulation. The public is rightly concerned that we are all being exposed to numerous chemicals without a clear understanding of the risks from those chemicals.

Administrator Jackson and I have both testified before Congress that EPA's authority is outdated and does not provide the tools to adequately protect human health and the environment. We believe there is a growing consensus that more needs to be done to improve our management of chemicals and reduce harmful exposures to chemicals.

Just this past December, the Centers for Disease Control, issued their most recent biomonitoring report on 212 chemicals, which reflects the levels of chemicals in our bodies. While this type of information does not provide a complete picture of environmental concerns and related health effects, it raises concern about exposure to harmful chemicals.

The Toxic Substances Control Act (TSCA) was signed into law in 1976 and was intended to provide protection of health and the environment against risks posed by chemicals in commerce. However, when TSCA was enacted, it authorized manufacture and use, without any evaluation, of all chemicals that were produced for commercial purposes at that time. Thus, manufacturers of these "grandfathered" chemicals weren't required to develop and produce the data on toxicity and exposure that are needed to properly and fully assess potential risks. Further compounding this problem, the statute never provided adequate authority for EPA to reevaluate existing chemicals as new concerns arose or as new scientific information became available.

As a result of the legal hurdles and procedural requirements TSCA places on EPA prior to collecting data, there are large, troubling gaps in the available data and state of knowledge on many widely used chemicals in commerce. Although there is a review process for new chemicals being introduced into commerce, chemical producers are not required to provide, without further action from EPA, the data necessary to fully assess a chemical's risks.

In the cases where EPA has adequate data on a chemical, and wants to protect the public against well-known risks to human health and the environment, there are legal hurdles that

prevent quick and effective regulatory action. Meanwhile, the public may be exposed to chemicals for which we have little understanding of the consequences.

As has been frequently cited, after years of study, EPA issued a rule in 1989 phasing out most uses of asbestos – a chemical whose health effects had been exhaustively studied and that had been demonstrated to cause lung cancer, mesothelioma and asbestosis in humans. Yet, a federal court overturned the rule because EPA failed to clear the hurdles imposed under TSCA before existing chemical risks can be controlled.

The question before all of us is how we better identify chemical risks and take effective action to eliminate harmful chemical exposures. To begin with, we need better and more comprehensive information on chemicals. Due to the legal and procedural hurdles in TSCA, over the last 30 years, EPA has only been able to require testing on around 200 of the 84,000 chemicals on the TSCA Inventory. To date, only five existing chemicals have been regulated under TSCA's ban authority.

The Obama Administration's principles for how this law should be revised and modernized call for stronger and clearer authority for EPA to collect and act upon critical data regarding chemicals risks. To summarize those principles:

First, chemicals should be reviewed against safety standards that are based on sound science and reflect risk-based criteria protective of human health and the environment. Safety standards should be driven solely by scientific evidence of risks. EPA should have the clear authority to establish safety standards that reflect the best available science while recognizing the need to assess and manage risk in the face of uncertainty.

Second, the responsibility for providing adequate health and safety information should rest on industry. Manufacturers must develop and submit the hazard, use, and exposure data demonstrating that new and existing chemicals under review are safe. If industry doesn't

provide the information, EPA should have the necessary tools to quickly and efficiently require testing, or obtain other information from manufacturers that are relevant to determining the safety of chemicals, without the delays and obstacles currently in place, or excessive claims of confidential business information.

Third, EPA should have clear authority to take risk management actions when chemicals do not meet the safety standard, with flexibility to take into account a range of considerations, including children's health, economic costs, social benefits, and equity concerns. EPA and industry must include special consideration for exposures and effects on groups with higher vulnerabilities – particularly children.

Fourth, EPA should have clear authority to set priorities for conducting safety reviews. In all cases, EPA and chemical producers must act on priority chemicals in a timely manner, with firm deadlines to maintain accountability. This will not only assure prompt protection of health and the environment, but also provide business with the certainty that it needs for planning and investment.

Fifth, we must encourage innovation in green chemistry, and support research, education, recognition, and other strategies that will lead us down the road to safer and more sustainable chemicals and processes. All of this must happen with transparency and consideration of the public's right to know.

Finally, implementation of the law should be adequately and consistently funded, in order to meet the goal of assuring the safety of chemicals, and to maintain public confidence that EPA is meeting that goal. To that end, manufacturers of chemicals should support the costs of Agency implementation, including the review of information provided by manufacturers.

Over the past few decades, the United States has negotiated and signed international agreements that have the goal of protection of human health and the environment from toxic

chemicals, but has been unable to join these Conventions because of lack of domestic legislation to implement chemicals treaty commitments. The Obama Administration has identified the Rotterdam and Stockholm conventions as priority treaties for U. S. ratification. We believe that TSCA reform provides an opportunity for the consideration of implementing legislation for the Rotterdam and Stockholm conventions.

The science underlying our understanding of chemicals has evolved substantially since TSCA was enacted. Any standards we set must allow us to take advantage of new approaches in modeling and alternative testing methods that will give us the tools to better understand risks, more quickly and efficiently. Most importantly, we must look closely at the chemicals which may present unique health effects among sensitive populations, such as children. Data suggest that many commonly used chemicals can be found in our bloodstreams, and we need to better understand the implications of this cumulative exposure to multiple chemicals. Further we are taking advantage of advances in molecular biology and computer science that have the potential to transform chemical toxicity testing and provide the ability to rapidly screen environmental chemicals. EPA's Office of Research and Development is developing robust and flexible computational tools that will assist the Agency in evaluating thousands of chemicals. The goal of EPA's Computational Toxicology Research Program is to provide decision support tools for screening and assessing chemical exposure, hazard and risk, and to inform green chemical design. One such tool is ToxCast, a cost-effective approach for screening thousands of chemicals in less time and for less cost than animal studies. Bioactivity profiles and toxicity predictions from ToxCast are providing EPA regulatory programs with science-based information helpful in prioritizing chemicals for more detailed toxicological evaluations.

As legislative reform moves forward, Administrator Jackson has committed to enhancing the Agency's chemical management program, by utilizing our current authority under TSCA to the fullest extent possible to ensure that we do everything we can to protect the American people and the environment from dangerous chemicals. Fundamental reform is needed to fully

protect against chemical risks, but until then we will move forward aggressively under the existing law.

On December 30, as part of this effort, EPA posted an initial set of four action plans addressing phthalates, long-chain perfluorinated chemicals (PFCs), polybrominated diphenyl ethers (PBDEs), and short-chain chlorinated paraffins. We are also developing action plans on benzadine dyes and bisphenol-A (BPA), although those plans are not yet ready for public release. The action plans outline the concerns that the chemicals present and the actions the Agency intends to take to address those concerns, including for the first time, utilizing TSCA's authority to list chemicals of concern.

The chemicals selected for action plan development were chosen on the basis of multiple factors, including available hazard, exposure, and use information; potential concern for children's health; use in consumer products; presence in human blood; persistent, bioaccumulative and toxic characteristics; toxicity; and production volume. We plan to use these criteria for selecting additional chemicals for future action plans as well.
Last month, EPA also announced that several U. S. companies will undertake a three-year phaseout of decabromodiphenyl ether (DecaBDE), a substance that has been used as a flame retardant in consumer and other products. Studies have shown that DecaBDE persists in the environment, potentially causes cancer, and may impact brain function. DecaBDE also can degrade to more toxic chemicals that are frequently found in the environment and are hazardous to wildlife. EPA believes that the action by these companies is an appropriate and responsible step to protect human health and environment.

As I indicated earlier, increasing transparency needs to be part of the foundation of legislative reform but we are not waiting. We intend to use the tools currently available to us, to increase the public's access to chemical information. While we understand that there are, at times, legitimate reasons why a company may need to claim confidentiality, it is also clear that CBI claims are used too often, in too many areas. For example, under TSCA, companies are

required to submit health and safety information on substantial risk, and companies have frequently claimed the chemical name as CBI in these submissions. While the Agency has the information, the public version does not include the chemical name which obviously limits the value of that information. Indeed, of the roughly 84,000 chemicals included on the TSCA inventory, the identity of more than 16,000 of these chemicals is currently classified as confidential. That makes no sense.

To begin addressing this problem, earlier this month, we announced a policy shift to alert companies that we will reject confidentiality claims for chemicals that are on the public portion of the TSCA inventory. Moreover, this past July, in one of my first acts at the new Assistant Administrator, we took action to add 530 chemicals to the public portion of the TSCA Inventory which had previously been on the confidential portion because the CBI information had been made public in one form or another. Over the coming months, we intend to announce a number of actions that will further increase transparency and assure the safety of chemicals in this country.

While we have undertaken an effort to identify and take action on a number of chemicals that are commonly used in commerce and we are beginning to increase access to information, it is clear that increased regulatory and scientific attention needs to be focused. Simply put, the existing TSCA authorities are not adequate and there can be no substitute for meaningful reform of the underlying law. It is time to bring TSCA into the 21st century. EPA looks forward to working with this committee on this very important issue. I am now happy to answer questions.

Testimony before the Subcommittee on Superfund, Toxics and Environmental Health
Committee on
Environment and Public Works
United States Senate

Current Science on Public Exposures to Toxic Chemicals

Statement of

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Centers for Disease Control and Prevention; and
Agency for Toxic Substances and Disease Registry
U.S. Department of Health and Human Services

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Introduction

Good morning Mr. Chairman and Members of the Subcommittee.

My name is Dr. Henry Falk, and I am the Acting Director of the National Center for Environmental Health at the Centers for Disease Control and Prevention (CDC), and of the Agency for Toxic Substances and Disease Registry (ATSDR).

I am pleased to appear today before the Subcommittee to discuss CDC's work in assessing people's exposure to chemicals. My testimony will focus on the biomonitoring program at CDC, and public health uses of biomonitoring.

For approximately three decades, CDC has been using biomonitoring to assess human exposure to selected chemicals (both manmade and naturally occurring). Biomonitoring is the direct measurement of chemicals and naturally occurring compounds or their metabolites in people's blood, urine or tissue. It determines which chemicals—and how much of them—get into people after they have been exposed.

CDC's Biomonitoring Program

I will describe two aspects of CDC's biomonitoring program: assessment of the U.S. population's exposure to chemicals, and targeted studies to examine exposure in vulnerable populations.

How CDC assesses the U.S. population's exposure to chemicals: CDC's Environmental Health Laboratory measures chemicals or their metabolites in blood and urine samples from participants in the National Health and Nutrition

Examination Survey (NHANES). NHANES, which is conducted by CDC's National Center for Health Statistics, involves a complete physical exam, a detailed questionnaire that collects more than 1,000 pieces of information, and the collection of blood and urine samples. The survey, which is nationally representative of the U.S. population, has been conducted multiple times since the 1970s and became a continuous survey in 1999 with two-year survey cycles. With some exceptions, most urine measurements are done in participants ages 6 years and older, and most serum measurements are done in participants age 12 years and older. Thus, the exposure information it provides on young children is limited, mostly due to the difficulty in obtaining large enough blood and urine samples from them. Currently blood levels of lead, cadmium, and mercury are measured in children aged 1 year and older, and cotinine, which is a marker for environmental tobacco smoke exposure, is measured in children aged 3 years and older.

CDC scientists publish significant biomonitoring findings from NHANES in peer-reviewed publications, and then CDC periodically publishes a summary report, the National Report on Human Exposure to Environmental Chemicals. The Fourth Exposure Report was released in December 2009, and summarizes blood and urine levels for 212 chemicals, including levels for 75 chemicals which had never before been measured in a representative sample of the U.S. population. Findings show evidence of widespread exposure in the U.S. population to some commonly-used commercial chemicals such as bisphenol-A (BPA), the perfluorinated compound known as PFOA, and a type of fire retardant known as BDE-47. The Fourth Exposure Report also notes continued progress in reducing

children's exposure to lead.

The data in the Fourth Exposure Report provide unique exposure information that can be used by scientists, physicians, and health officials for a variety of public health purposes, such as to: determine which chemicals get into Americans' bodies and at what concentrations; determine what proportion of the population has levels above those associated with adverse health effects for chemicals with a known toxicity level; establish reference values that can be used by physicians and scientists to determine whether a person or group has an unusually high exposure; track over time trends in levels of exposure of the population; assess the effectiveness of public health efforts to reduce exposure of Americans to specific chemicals; determine whether exposure levels are higher among minorities, children, women of childbearing age, or other special groups; and direct priorities for research on human health effects from exposure.

Chemicals analyzed from the NHANES samples and reported in the Fourth Report were selected based on known or hypothesized exposure in the U.S. population; scientific data on the health effects known or thought to result from some levels of exposure; the need to assess the efficacy of public health actions to reduce exposure to a chemical with known health effects; the availability of an analytical method that is accurate, precise, sensitive, and specific; the availability of adequate blood or urine samples from the NHANES survey; and the analytical cost to perform the analysis. Also, CDC has solicited suggestions for candidate chemicals from the public and other government agencies.

Targeted studies: Each year CDC's Environmental Health Laboratory works with states, other federal agencies, academic institutions and international organizations on 50-70 studies that examine vulnerable populations, particularly newborns, children, pregnant women and population groups or communities known or likely to have higher exposures. For example, one important current partnership is with the Eunice Kennedy Shriver National Institute of Child Health and Human Development at the National Institutes of Health. This partnership involves a pilot study of 525 pregnant women in which CDC is lending analytical and biomonitoring expertise. Scientists at CDC's Environmental Health Lab will measure chemicals in pregnant women's blood and urine and, after delivery, in the newborn's cord blood and mother's breast milk. Cord blood is a promising way to assess prenatal exposure to certain chemicals. However, cord blood is not the best way to measure exposures to chemicals that pass through the body more quickly; these generally are best measured in urine.

Public Health Uses

As distinguished from measurements in environmental samples, such as air, soil, water, food, and consumer products, biomonitoring measurements have the advantage of indicating the amount of a chemical that actually gets into people, rather than extrapolating from measurements of environmental media. Although biomonitoring is far ahead of the science of interpreting what exposures mean for health, biomonitoring data is valuable, and CDC uses it for a range of public health purposes, including to establish reference ranges in the population and to identify groups of people with higher levels of exposure than those typical for the

U.S. population. In addition, by tracking exposures in the U.S. population, we can detect trends in people over time, and assess whether a chemical is present in large numbers of people, or is disproportionately present in vulnerable subgroups, such as children. This information can be used by scientists and policy makers as one of the considerations in setting priorities for evaluating health impacts of chemicals. Biomonitoring thereby serves as one important tool in identifying and reducing or preventing exposures and potential health problems.

A National Research Council review of biomonitoring noted that it has been a key tool in some landmark public health actions (NRC, 2006). One example is lead. Our laboratory has been measuring lead in the NHANES blood samples since 1976. Many of the effects of lead can be benchmarked to blood lead concentrations. Lead is highly toxic, especially to young children, and can harm a child's brain, kidneys, bone marrow, and other body systems. It can cause decrements in cognitive ability and IQ and at very high levels can cause coma, convulsions, and death.[1] Our laboratory analysis of the NHANES samples, which showed that the American population's blood lead levels were declining in parallel with declining levels of lead in gasoline, provided critical support for the Environmental Protection Agency (EPA) regulations that reduced lead in gasoline (GAO, 2000). CDC and EPA have used this decline in blood lead levels over time to demonstrate that the removal of lead from gasoline had a dramatic impact on the levels of lead in the U.S. population. Today, the most common source of children's exposure to lead is from dust and soil derived from lead-based paints in older homes.[2] In the late 1970s, CDC used the NHANES data to document

that 88 percent of children had blood lead levels above 10 µg/dL, the current level of concern. Data from the Fourth Exposure Report demonstrate that collaborative public health efforts by CDC, EPA, NIEHS, the Department of Housing and Urban Development, and others reduced children's exposure to lead. For the period 1999–2004, only 1.4% of children aged 1 to 5 years had elevated blood lead levels.

Biomonitoring also can be used to monitor the effectiveness of interventions designed to reduce exposures. In the early 1990s, our laboratory analysis of cotinine data from NHANES showed that 88 percent of the nonsmoking population was exposed to secondhand tobacco smoke. This finding was used by state and local areas as a justification for restricting smoking in public places. Over the past 15 years, NHANES data have shown that exposure to secondhand smoke in nonsmokers has decreased about 70 percent, indicating that public health interventions to reduce exposure have been successful.

Conclusion

In conclusion, biomonitoring provides solid human data that can assist in making important health decisions. Better exposure information means that we can make better decisions to protect the health of the public.

CDC is fully committed to working with other federal agencies and partners to improve the uses and benefits of biomonitoring. Thank you Mr. Chairman and members of the Subcommittee. I look forward to answering any questions you may have.

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NHANES Web Site: http://www.cdc.gov/nchs/nhanes.htm

[1] *Eliminating Childhood Lead Poisoning: A Federal Strategy Targeting Lead Paint Hazards*. Washington, DC: President's Task Force on Environmental Health Risks and Safety Risks to Children; 2000.

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2009

Fourth National Report on Human Exposure to Environmental Chemicals

Executive Summary

Department of Health and Human Services
Centers for Disease Control and Prevention
National Center for Environmental Health

Background

The *National Report on Human Exposure to Environmental Chemicals (National Exposure Report)* is a series of ongoing assessments of the U.S. population's exposure to environmental chemicals by measuring chemicals in people's blood and urine, also called biomonitoring. The *Fourth National Report* on *Human Exposure to Environmental Chemicals* (*Fourth Report*) presents exposure data for 212 environmental chemicals for the civilian, noninstitutionalized U.S. population. This *Fourth Report* includes results from 2003–2004, as well as data from1999–2000 and 2001–2002 as reported in the *Second* and *Third National Report on Human Exposure to Environmental Chemicals*.

To obtain data for this *Fourth Report*, the Centers for Disease Control and Prevention (CDC)'s Environmental Health Laboratory at the National Center for Environmental Health measured chemicals or their metabolites in blood and urine from a random sample of participants from the National Health and Nutrition Examination Survey (NHANES). CDC's National Center for Health Statistics conducts NHANES, which is a series of surveys on the health status, health-related behaviors, and nutrition of the U.S. population. Since 1999, NHANES has been conducted in continuous two-year survey cycles.

For the *National Exposure Report*, an environmental chemical refers to a chemical compound or chemical element present in air, water, food, soil, dust, or other environmental media, such as consumer products. Blood and urine levels reflect the amount of the chemical that actually gets into the body from the environment. Either the chemical or its metabolite is measured. A metabolite is a substance produced when body tissues chemically alter the original compound.

The *Fourth Report* includes results for 75 chemicals measured for the first time in the U.S. population. These chemicals are in the following groups:

- acrylamide and glycidamide adducts;
- arsenic species and metabolites;
- environmental phenols, including bisphenol A and triclosan;
- perchlorate;
- perfluorinated chemicals;
- polybrominated diphenyl ethers;
- volatile organic compounds; and
- some additions to chemical groups previously measured.

A complete listing of the 75 new chemicals is given on page 10. A full listing of the chemicals included in the *Fourth Report* is available at http://www.cdc.gov/exposurereport/pdf/NER_Chemical_List.pdf.

Interpreting the Data

The presence of an environmental chemical in people's blood or urine does not mean that it will cause effects or disease. The toxicity of a chemical is related to its dose or concentration, in addition to a person's individual susceptibility. Small amounts may be of no health consequence, whereas larger amounts may cause adverse health effects. Research studies, separate from the *National Exposure Report*, are required to determine the levels of a chemical that may cause health effects and the levels that are not a significant health concern. For some chemicals, such as lead, research studies provide a good understanding of health risks associated with various blood levels. For most of the environmental chemicals included in the *Fourth Report*, more research is needed to determine whether exposure at the levels reported is a cause for health concern. CDC conducts and provides biomonitoring measurements for this type of research in collaboration with other agencies and institutions.

The *Fourth Report* presents data that provides estimates of exposure for the civilian, noninstitutionalized U.S. population. The current survey design does not permit CDC to estimate exposure on a state-by-state or city-by-city basis. For example, CDC cannot extract a subset of data and examine levels of blood lead that represent a state population.

Public Health Uses of *the Fourth Report*

The *Fourth Report* provides unique exposure information to scientists, physicians, and health officials to help prevent effects that may result from exposure to environmental chemicals. Specific public health uses of the exposure information in the *Fourth Report* are to:

- determine which chemicals get into Americans' bodies and at what concentrations;
- determine what proportion of the population has levels above those associated with adverse health effects for chemicals with a known toxicity level;
- establish reference values that can be used by physicians and scientists to determine whether a person or group has an unusually high exposure;
- assess the effectiveness of public health efforts to reduce exposure of Americans to track levels over time;
- determine whether exposure levels are higher among minorities, children, women of childbearing age, or other special groups; and
- direct priorities for research on human health effects from exposure.

First-Time Exposure Information for the U.S. Population Provided for 75 Chemicals

The *Fourth Report*, for the first time, provides population reference values in blood and urine, including 95th percentile levels, for 75 chemicals. The 95th percentile level means that 95% of the population has concentrations below that level. Public health officials use such reference values to determine whether groups of people are experiencing an exposure that is unusual compared with an exposure experienced by the rest of the population.

To provide scientists and public health officials these new data quickly, CDC published much of this exposure information on new chemicals in separate scientific peer-reviewed publications before the *Fourth Report* was released. Abstracts and links to full-text articles are available at http://www.cdc.gov/exposurereport/.

Widespread Exposure to Some Industrial Chemicals

Findings in the *Fourth Report* indicate widespread exposure to some commonly used industrial chemicals.

- Polybrominated diphenyl ethers are fire retardants used in certain manufactured products. These accumulate in the environment and in human fat tissue. One type of polybrominated diphenyl ether, BDE-47, was found in the serum of nearly all of the NHANES participants.
- Bisphenol A (BPA), a component of epoxy resins and polycarbonates, may have potential reproductive toxicity. General population exposure to BPA may occur through ingestion of foods in contact with BPA-containing materials. CDC scientists found bisphenol A in more than 90% of the urine samples representative of the U.S. population.
- Another example of widespread human exposure included several of the perfluorinated chemicals. One of these chemicals, perfluorooctanoic acid (PFOA), was a byproduct of the synthesis of other perfluorinated chemicals and was a synthesis aid in the manufacture of a commonly used polymer, polytetrafluoroethylene, which is used to create heat-resistant non-stick coatings in cookware. Most participants had measurable levels of this environmental contaminant.

Ongoing Progress in Reducing Blood Lead Levels in Children

Progress is being made in reducing children's blood lead levels. New data on blood lead levels in children aged 1 to 5 years enable estimates of the number of children with elevated levels (that is, levels greater than or equal to 10 micrograms per deciliter [µg/dL]). Figure 1 shows how the percentage of blood lead levels in children has declined since the late 1970s. For example, for the period 1999–2004, 1.4% of children aged 1 to 5 years had elevated blood lead levels, the smallest percentage of any of the prior survey periods.

These data document that public health efforts to reduce the number of children with elevated blood lead levels in the general population continue to be successful. However, the *Fourth Report* also notes that other data sources show that special populations of children at high risk for lead exposure (for example, children living in homes containing lead-based paint or lead-contaminated dust) have higher rates of elevated blood lead levels and remain a major public health concern.

First-Time Assessment of Acrylamide Exposure in the U.S. Population

Acrylamide is formed when foods containing carbohydrates are cooked at high temperatures (e.g., French fries) and as a byproduct of tobacco smoke. Most people are exposed to acrylamide through the diet and from smoking. Because acrylamide is a reactive chemical, it can bind to proteins. These reaction products are called adducts. CDC's Environmental Health Laboratory developed a new method to measure acrylamide and its metabolite, glycidamide, as adducts of hemoglobin, a major blood protein. This measure reflects the dose of acrylamide and glycidamide over the previous several months of intake. The data in the *Fourth Report* show that acrylamide exposure is extremely common in the U.S. population.

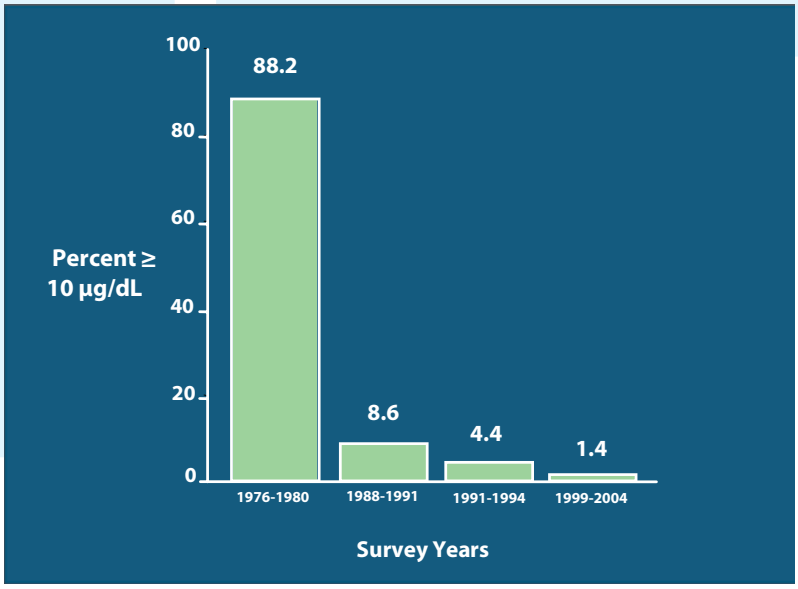


Figure 1. Percentage of children 1-5 years old in the U.S. population with elevated blood lead levels (≥ 10 µg/dL).[1]

[1] *Jones RL, Homa DM, Meyer PA, Brody DJ, Caldwell KL, Pirkle JL, Brown MJ. Trends in blood lead levels and blood lead testing among U.S. children aged 1 to 5 years, 1988–2004. Pediatrics 2009;123(3):e376-e385.*

First Available Exposure Data on Mercury in the U.S. Population

For the first time, the *Fourth Report* characterizes mercury exposure of the U.S. population aged 1 year and older. Previous *National Exposure Reports* presented mercury levels for children 1–5 years old and women 16–49 years old. Total blood mercury levels are primarily composed of one type of mercury, methyl mercury, which enters the body mainly from dietary seafood sources. Findings in the *Fourth Report* show that total blood mercury levels increase with age for all groups and begin to decline after the fifth decade of life. Compared to older women of childbearing age, younger women have higher birth rates and lower mercury levels (see Figure 2).

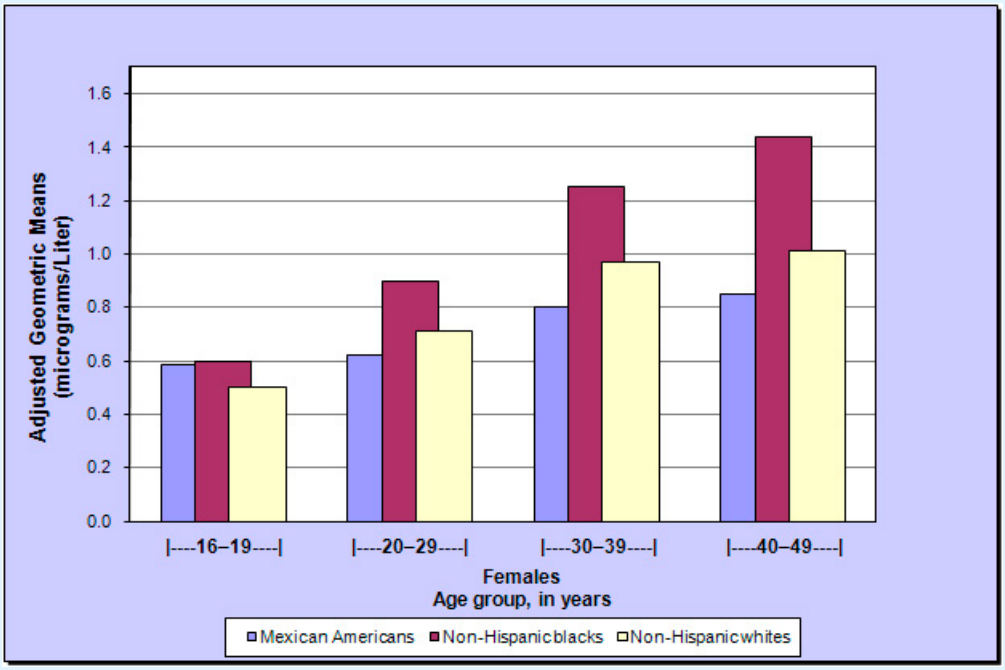


Figure 2. Age-related changes in total blood mercury levels for females aged16-49 by race/ethnicity, 1999-2006.[2]

Eight Different Species and Metabolites of Arsenic Measured

By using special laboratory methods, CDC researchers measured total arsenic and seven other forms of arsenic in the urine of NHANES participants for the first time. Some of the forms of arsenic measured are metabolites of inorganic arsenic and others are less toxic species that are formed in the environment. By differentiating these types of arsenic exposure, the *Fourth Report* helps scientists understand which forms of arsenic are important to human health.

[2] Caldwell KL, Mortensen ME, Jones RL, Caudill SP, Osterloh JD. Total blood mercury concentrations in the U.S. population: 1999-2006. Int J Hyg Environ Health 2009;212:588-598.

Perchlorate and Thyroid Function

The chemical perchlorate is both naturally occurring and manmade and is used to manufacture fireworks, explosives, flares, and rocket propellant. For decades, scientists have known that large medical doses of perchlorate affect thyroid function. Low-level exposure to perchlorate from the environment has been under investigation by many scientists in recent years. The *Fourth Report* shows that all NHANES participants have detectable perchlorate in their urine and provides reference values for urinary perchlorate levels (see Table 1). This knowledge helps scientists target the levels of human exposure for future study.

Urinary Perchlorate

Geometric mean and selected percentiles of urine concentrations (in µg/L) for the U.S. population from the National Health and Nutrition Examination Survey.

	Survey years	Geometric mean (95% conf. interval)	Selected percentiles (95% confidence interval) 50th	75th	90th	95th	Sample size
Total	01-02	**3.54** (3.29-3.81)	**3.70** (3.50-4.00)	**6.30** (5.80-6.90)	**10.0** (9.10-11.0)	**14.0** (11.0-17.0)	2820
	03-04	**3.22** (2.93-3.55)	**3.30** (2.90-3.80)	**5.50** (5.00-6.40)	**9.50** (8.40-11.0)	**13.0** (12.0-15.0)	2522
Age group							
6-11 years	01-02	**4.93** (4.22-5.76)	**5.20** (4.40-6.40)	**8.10** (6.90-9.80)	**12.0** (9.30-19.0)	**19.0** (12.0-23.0)	374
	03-04	**4.32** (3.67-5.09)	**4.60** (4.00-5.20)	**7.90** (5.70-9.50)	**13.0** (8.81-16.0)	**16.0** (11.0-29.0)	314
12-19 years	01-02	**3.80** (3.44-4.20)	**4.40** (3.80-4.80)	**6.80** (6.30-7.30)	**10.0** (8.90-11.0)	**13.0** (11.0-17.0)	828
	03-04	**3.62** (3.19-4.12)	**3.80** (3.20-4.40)	**6.40** (5.50-7.10)	**9.80** (7.90-12.0)	**13.0** (10.0-18.0)	721
20 years and older	01-02	**3.35** (3.08-3.65)	**3.50** (3.20-3.70)	**5.90** (5.30-6.60)	**10.0** (8.70-11.0)	**13.0** (11.0-17.0)	1618
	03-04	**3.05** (2.75-3.38)	**3.20** (2.70-3.60)	**5.20** (4.70-6.10)	**9.10** (7.90-10.0)	**12.0** (11.0-14.0)	1487
Gender							
Males	01-02	**4.19** (3.93-4.46)	**4.40** (4.20-4.60)	**7.10** (6.40-7.90)	**11.0** (9.70-12.0)	**14.0** (11.0-19.0)	1335
	03-04	**3.75** (3.39-4.16)	**3.90** (3.40-4.40)	**6.40** (5.60-7.50)	**11.0** (9.20-12.0)	**14.0** (13.0-17.0)	1229
Females	01-02	**3.01** (2.74-3.31)	**3.10** (2.70-3.40)	**5.40** (5.00-6.00)	**9.20** (8.20-11.0)	**13.0** (11.0-17.0)	1485
	03-04	**2.79** (2.49-3.11)	**2.90** (2.50-3.20)	**4.90** (4.40-5.50)	**8.20** (6.90-9.84)	**11.0** (8.80-15.0)	1293
Race/ethnicity							
Mexican Americans	01-02	**4.02** (3.47-4.66)	**4.40** (3.70-5.00)	**7.10** (5.80-8.40)	**12.0** (9.40-13.0)	**14.0** (12.0-18.0)	708
	03-04	**3.76** (3.45-4.11)	**3.96** (3.50-4.40)	**6.20** (5.30-7.50)	**11.0** (9.10-12.0)	**15.0** (12.0-17.0)	617
Non-Hispanic blacks	01-02	**3.51** (3.07-4.03)	**3.70** (3.10-4.10)	**5.90** (5.10-7.00)	**9.20** (7.80-12.0)	**15.0** (11.0-20.0)	681
	03-04	**3.21** (2.90-3.56)	**3.20** (2.87-3.50)	**5.40** (4.60-6.30)	**8.60** (7.50-11.0)	**13.0** (9.30-17.0)	652
Non-Hispanic whites	01-02	**3.51** (3.18-3.88)	**3.70** (3.40-4.10)	**6.30** (5.70-7.10)	**10.0** (8.90-11.0)	**14.0** (11.0-18.0)	1228
	03-04	**3.26** (2.89-3.68)	**3.30** (2.80-4.00)	**5.60** (4.90-6.80)	**9.40** (8.10-11.0)	**13.0** (11.0-15.0)	1092

Limit of detection (LOD, see Data Analysis section in full *Report*) for Survey years 01-02 and 03-04 are 0.05 and 0.05. For the 2001-2002 Survey period, surplus samples were used, and data are unavailable at NHANES website.

***Table 1. Urinary Perchlorate as provided in the* Fourth Report.**

Reduced Exposure to Environmental Tobacco Smoke

Environmental tobacco smoke (ETS) has significant health effects on cardiovascular and respiratory disease. Cotinine is a metabolite of nicotine, and for nonsmokers, levels of cotinine in people's blood tracks exposure to ETS. In the past 15 years, data show that blood cotinine levels for nonsmokers in the U.S. population have decreased about 70%, indicating that public health interventions to reduce ETS exposure have been successful.

U.S. Population's Exposure to Volatile Organic Compounds

People are exposed every day to volatile chemicals in the air we breathe. The *Fourth Report* provides measurements on 33 of these hydrocarbon and halohydrocarbon-type chemicals. One example is the gasoline additive methyl *tert*-butyl ether (MTBE). Exposure to this chemical can occur through the air we breathe or from contaminated water sources. A high percentage of the NHANES participants representing the U.S. population showed detectable levels of MTBE.

Exposure to Cadmium

Recent research studies show that urine cadmium levels as low as 1 microgram per gram of creatinine in people may be associated with subtle markers of effects on the kidney and with an increased risk for low bone-mineral density. The *Fourth Report* shows that about 5% of the U.S. population aged 20 years and older has urinary cadmium levels at or near these levels. Cigarette smoking is the most likely source for these higher cadmium levels. These findings should promote further research on the public health consequences of cadmium in people.

Selection of Chemicals for the *Fourth Report*

Chemicals presented in the *Fourth Report* were selected on the basis of scientific data that suggested exposure in the U.S. population; the seriousness of health effects known or suspected to result from exposure; the need to assess the efficacy of public health actions to reduce exposure to a chemical; the availability of a biomonitoring analytical method with adequate accuracy, precision, sensitivity, specificity, and speed; the availability of sufficient quantity of blood or urine samples; and the incremental analytical cost to perform the analyses. More information is available at http://www.cdc.gov/exposurereport/chemical_selection.htm.

Plans for Future *National Exposure Reports*

CDC's goal is to make new biomonitoring exposure information available as soon as possible to the public and scientific community. To meet this goal, CDC periodically releases the *National Exposure Report* and also publishes biomonitoring exposure information in peer-reviewed publications. The *National Exposure Report* is cumulative, providing biomonitoring exposure data starting in 1999 through the latest available data at the time of the report release. Future plans include releasing data on additional chemicals and providing more information on exposure in population groups defined by age, sex, and race or ethnicity. Peer-reviewed journal articles published since the latest release of the *National Exposure Report* provide more recent and supplementary biomonitoring data for the U.S. population. These peer-reviewed publications typically also contain more extensive data analysis than that provided in the *National Exposure Report*.

About CDC's Environmental Health Laboratory

By using advanced laboratory science and innovative techniques, CDC's Environmental Health Laboratory at the National Center for Environmental Health has been at the forefront of efforts to assess people's exposure to environmental chemicals. CDC's laboratory scientists have built on more than three decades of experience in measuring chemicals directly in people's blood or urine, a process known as biomonitoring. Biomonitoring measurements are the most health-relevant assessments of exposure because they measure the total amount of the chemical that actually gets into people from all environmental sources (e.g., air, soil, water, dust, or food). With a few exceptions, the concentration of the chemical in people provides the best exposure information for public health officials to evaluate the potential for adverse health effects.

New Chemicals in the *Fourth Report*

Acrylamide
Acrylamide hemoglobin adducts
Glycidamide hemoglobin adducts

Perchlorate

Total and Speciated Arsenic
Arsenic, Total
Arsenic (V) acid
Arsenobetaine
Arsenocholine
Arsenous (III) acid
Dimethylarsinic acid
Monomethylarsonic acid
Trimethylarsine oxide

Environmental Phenols
Benzophenone-3 (2-Hydroxy-4-methoxybenzophenone)
Bisphenol A (2,2-*bis* [4-Hydroxyphenyl] propane)
4-*tert* -Octylphenol (4-[1,1,3,3-Tetramethylbutyl] phenol)
Triclosan (2,4,4′-Trichloro-2′-hydroxyphenyl ether)

Phthalate Metabolite
Mono-(2-ethyl-5-carboxypentyl) phthalate (MECPP)

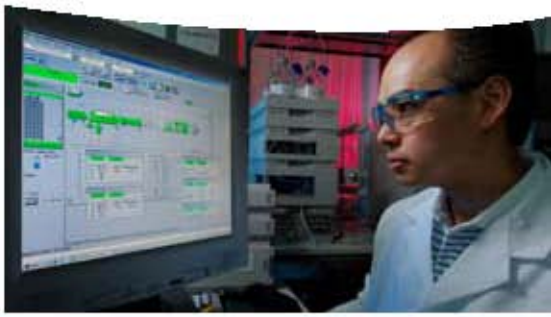
Perfluorochemicals
Perfluorobutane sulfonic acid (PFBuS)
Perfluorodecanoic acid (PFDeA)
Perfluorododecanoic acid (PFDoA)
Perfluoroheptanoic acid (PFHpA)
Perfluorohexane sulfonic acid (PFHxS)
Perfluorononanoic acid (PFNA)
Perfluorooctane sulfonamide (PFOSA)
Perfluorooctane sulfonic acid (PFOS)
2-(N-Ethyl-perfluorooctane sulfonamido) acetic acid (Et-PFOSA-AcOH)
2-(N-Methyl-perfluorooctane sulfonamido) acetic acid (Me-PFOSA-AcOH)
Perfluorooctanoic acid (PFOA)
Perfluoroundecanoic acid (PFUA)

Non-Dioxin-Like Polychlorinated Biphenyls
2,2′,3,5′-Tetrachlorobiphenyl (PCB 44)
2,2′,4,5′-Tetrachlorobiphenyl (PCB 49)
2,2′,3,3′,4,4′,5,5′,6,6′-Decachlorobiphenyl (PCB 209)

Brominated Fire Retardants
2,2′,4-Tribromodiphenyl ether (BDE 17)
2,4,4′-Tribromodiphenyl ether (BDE 28)
2,2′,4,4′-Tetrabromodiphenyl ether (BDE 47)
2,3′,4,4′-Tetrabromodiphenyl ether (BDE 66)
2,2′,3,4,4′-Pentabromodiphenyl ether (BDE 85)
2,2′,4,4′,5-Pentabromodiphenyl ether (BDE 99)
2,2′,4,4′,6-Pentabromodiphenyl ether (BDE 100)
2,2′,4,4′,5,5′-Hexabromodiphenyl ether (BDE 153)
2,2′,4,4′,5,6′-Hexabromodiphenyl ether (BDE 154)
2,2′,3,4,4′,5′,6-Heptabromodiphenyl ether (BDE 183)
2,2′,4,4′,5,5′-Hexabromobiphenyl (BB 153)

Disinfection By-Products (Trihalomethanes)
Bromodichloromethane
Dibromochloromethane (Chlorodibromomethane)
Tribromomethane (Bromoform)
Trichloromethane (Chloroform)

Volatile Organic Compounds
Benzene
Chlorobenzene (Monochlorobenzene)
1,2-Dibromo-3-chloropropane (DBCP)
Dibromomethane
1,2-Dichlorobenzene (*ortho*-Dichlorobenzene)
1,3-Dichlorobenzene (*meta*-Dichlorobenzene)
1,4-Dichlorobenzene (*para*-Dichlorobenzene)
1,1-Dichloroethane
1,2-Dichloroethane (Ethylene dichloride)
1,1-Dichloroethene (Vinylidene chloride)
cis-1,2-Dichloroethene
trans-1,2-Dichloroethene
Dichloromethane (Methylene chloride)
1,2-Dichloropropane
2,5-Dimethylfuran (DMF)
Ethylbenzene
Hexachloroethane
Methyl *tert*-butyl ether (MTBE)
Nitrobenzene
Styrene
1,1,2,2-Tetrachloroethane
Tetrachloroethene (Perchloroethylene)
Tetrachloromethane (Carbon tetrachloride)
Toluene
1,1,1-Trichloroethane (Methyl chloroform)
1,1,2-Trichloroethane
Trichloroethene (Trichloroethylene, TCE)
meta- and *para*-Xylene
ortho-Xylene



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United States Government Accountability Office

GAO

Testimony

Before the Subcommittee on Superfund, Toxics and Environmental Health, Committee on Environment and Public Works, U.S. Senate

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BIOMONITORING

EPA Could Make Better Use of Biomonitoring Data

Statement of John Stephenson, Director
Natural Resources and Environment

GAO
Accountability * Integrity * Reliability

GAO-10-419T

February 4, 2009

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Highlights

Highlights of GAO-10-419T, a testimony before the Subcommittee on Superfund, Toxics, and Environmental Health, Committee on Environment and Public Works, U.S. Senate

BIOMONITORING

EPA Could Make Better Use of Biomonitoring Data

Why GAO Did This Study

Biomonitoring, which measures chemicals in people's tissues or body fluids, has shown that the U.S. population is widely exposed to chemicals used in everyday products. Some of these have the potential to cause cancer or birth defects. Moreover, children may be more vulnerable to harm from these chemicals than adults.

The Environmental Protection Agency (EPA) is authorized under the Toxic Substances Control Act (TSCA) to control chemicals that pose unreasonable health risks. One crucial tool in this process is chemical risk assessment, which involves determining the extent to which populations will be exposed to a chemical and assessing how this exposure affects human health

This testimony, based on GAO's prior work, reviews the (1) extent to which EPA incorporates information from biomonitoring studies into its assessments of chemicals, (2) steps that EPA has taken to improve the usefulness of biomonitoring data, and (3) extent to which EPA has the authority under TSCA to require chemical companies to develop and submit biomonitoring data to EPA.

View GAO-10-419T or key components.
For more information, contact John Stephenson at (202) 512-3841 or stephensonj@gao.gov.

What GAO Found

EPA has made limited use of biomonitoring data in its assessments of risks posed by commercial chemicals. One reason is that biomonitoring data relevant to the entire U.S. population exist for only 212 chemicals. In addition, biomonitoring data alone indicate only that a person was somehow exposed to a chemical, not the source of the exposure or its effect on the person's health. For most of the chemicals studied under current biomonitoring programs, more data on chemical effects are needed to understand if the levels measured in people pose a health concern, but EPA's authorities to require chemical companies to develop such data is limited. However, in September 2009, the EPA Administrator set forth goals for updated legislation to give EPA additional authorities to obtain data on chemicals.

While EPA has initiated several research programs to make biomonitoring more useful to its risk assessment process, it has not developed a comprehensive strategy for this research that takes into account its own research efforts and those of the multiple federal agencies and other organizations involved in biomonitoring research. EPA does have several important biomonitoring research efforts, including research into the relationships between exposure to harmful chemicals, the resulting concentration of those chemicals in human tissue, and the corresponding health effects. However, without a plan to coordinate its research efforts, EPA has no means to track progress or assess the resources needed specifically for biomonitoring research. Furthermore, according to the National Academy of Sciences, the lack of a coordinated national research strategy has allowed widespread chemical exposures to go undetected, such as exposures to flame retardants. While EPA agreed with GAO's recommendation that EPA develop a comprehensive research strategy, the agency has not yet done so.

EPA has not determined the extent of its authority to obtain biomonitoring data under TSCA, and this authority is untested and may be limited. The TSCA section that authorizes EPA to require companies to develop data focuses on health and environmental effects of chemicals. However, biomonitoring data indicate only the presence of a chemical in the body, not its impact on health. It may be easier for EPA to obtain biomonitoring data under other TSCA sections, which allow EPA to collect existing information on chemicals. For example, TSCA obligates chemical companies to report information that reasonably supports the conclusion that a chemical presents a substantial risk of injury to health or the environment. EPA asserts that biomonitoring data are reportable if a chemical is known to have serious toxic effects and biomonitoring data indicates a level of exposure previously unknown to EPA. EPA took action against a chemical company under this authority in 2004. However, the action was settled without an admission of liability by the company, so EPA's authority to obtain biomonitoring data remains untested. GAO's 2009 report recommended that EPA clarify this authority, but it has not yet done so. The agency did not disagree, but commented that a case-by-case explanation of its authority might be more useful than a global assessment.

Mr. Chairman, Ranking Member, and Members of the Subcommittee:

I am pleased to appear here today to discuss EPA's use of biomonitoring data. Biomonitoring, which measures chemicals in people's tissues or body fluids, has shown that the U.S. population is widely exposed to chemicals used in everyday products. Some of these have the potential to cause cancer or birth defects. Moreover, children may be more vulnerable to harm from these chemicals than adults because their biological functions are still developing and their size and behavior may expose them to proportionately higher doses.

The mission of the Environmental Protection Agency (EPA) is to protect human health and the environment. To help EPA achieve this objective, the Toxic Substances Control Act (TSCA) authorizes it to regulate the manufacture, processing, and distribution of chemicals. A crucial tool in this regulatory process is chemical risk assessment, which involves determining the extent to which populations will be exposed to a chemical and assessing how this exposure affects human health. EPA uses such risk assessments to determine if it needs to take any risk management actions, such as prohibiting or restricting the manufacture, processing, or distribution of a chemical.

A recent proliferation of biomonitoring data has provided new insights into the general population's exposure to chemicals. Biomonitoring studies for certain chemicals, such as lead, have been ongoing for decades, but recent advances in analytic methods have allowed scientists to measure more chemicals in smaller concentrations. This is a promising development. According to the Centers for Disease Control and Prevention (CDC), "biomonitoring measurements are the most health-relevant assessments of exposure because they measure the amount of the chemical that actually gets into people from all environmental sources, such as the air, soil, water, dust, or food combined." The CDC conducts the most comprehensive biomonitoring program in the country, and in December 2009 it published the fourth in a series of reports on the concentrations of certain chemicals or their by-products in a representative sample of the U.S. population. For example, the CDC reported that 90 percent of the people tested had detectable levels of Bisphenol A (BPA). BPA is an industrial chemical that has been present in many hard plastic bottles and metal-based food and beverage cans since the 1960s. On the basis of results from recent studies using novel approaches to test for subtle effects, the Food and Drug Administration announced in January of this year that it and the National Toxicology Program at the National Institutes of Health (NIH) have some concern

about the potential effects of BPA on the brain, behavior, and prostate gland in fetuses, infants, and young children.

My testimony today is based on our prior work on federal biomonitoring efforts and discusses EPA's use of current biomonitoring studies, EPA's biomonitoring research strategy, and EPA's authorities under TSCA to obtain biomonitoring data.[1] Specifically, my statement addresses (1) the extent to which EPA incorporates information from biomonitoring studies into its assessments of chemicals, (2) steps that EPA has taken to improve the usefulness of biomonitoring data, and (3) the extent to which EPA has the authority under TSCA to require chemical companies to develop and submit biomonitoring data to EPA. Our prior work was conducted in accordance with generally accepted government auditing standards. Those standards require that we plan and perform the audit to obtain sufficient, appropriate evidence to provide a reasonable basis for our findings and conclusions based on our audit objectives. We believe that the evidence obtained provides a reasonable basis for our findings and conclusions based on our audit objectives.

Background

Biomonitoring—one technique for assessing people's exposure to chemicals—involves measuring the concentration of chemicals or their by-products in human specimens, such as blood or urine. While, biomonitoring has been used to monitor chemical exposures for decades, more recently, advances in analytic methods have allowed scientists to measure more chemicals, in smaller concentrations, using smaller samples of blood or urine. As a result, biomonitoring has become more widely used for a variety of applications, including public health research and measuring the impact of certain environmental regulations, such as the decline in blood lead levels following declining levels of gasoline lead.

CDC conducts the most comprehensive biomonitoring program in the country under its National Biomonitoring Program and published the first, second, third and fourth National Report on Human Exposure to Environmental Chemicals—in 2001, 2003, 2005, and 2009, respectively—which reported the concentrations of certain chemicals or their by-products in the blood or urine of a representative sample of the U.S. population. For each of these reports, the CDC has increased the number

[1]GAO, *Biomonitoring: EPA Needs to Coordinate Its Research Strategy and Clarify Its Authority toObtain Biomonitoring Data*, GAO-09-353, (Washington, D.C.: Apr. 30, 2009).

of chemicals studied—from 27 in the first report, to 116 in the second, to 148 in the third, and to 212 in the fourth. Each report is cumulative (containing all the results from previous reports). These reports provide the most comprehensive assessment to date of the exposure of the U.S. population to chemicals in our environment including such chemicals as acrylamide, arsenic, BPA, triclosan, and perchlorate. These reports have provided a window into the U.S. population's exposure to chemicals, and the CDC continues to develop new methods for collecting data on additional chemical exposures with each report.

For decades, government regulators have used risk assessment to understand the health implications of commercial chemicals. Researchers use this process to estimate how much harm, if any, can be expected from exposure to a given contaminant or mixture of contaminants and to help regulators determine whether the risk is significant enough to require banning or regulating the chemical or other corrective action. Biomonitoring research is difficult to integrate into this risk assessment process, since estimates of human exposure to chemicals have historically been based on the concentration of these chemicals in environmental media and on information about how people are exposed. Biomonitoring data, however, provide a measure of internal dose that is the result of exposure to all environmental media and depend on how the human body processes and excretes the chemical.

EPA Has Made Limited Use of Biomonitoring Data in Assessing Risks Posed by Chemicals

EPA has made limited use of biomonitoring data in its assessments of risks posed by chemicals. As we previously reported,[2] one major reason for the agency's limited use of such data is that, to date, there are no biomonitoring data for most commercial chemicals. The most comprehensive biomonitoring effort providing data relevant to the entire U.S. population includes only 212 chemicals, whereas EPA is currently focusing its chemical assessment and management efforts on the more than 6,000 chemicals that companies produce in quantities of more than 25,000 pounds per year at one site.[3] Current biomonitoring efforts also provide little information on children. Large-scale biomonitoring studies generally omit children because it is difficult to collect biomonitoring data

[2]GAO-09-353.

[3]Companies must report on most chemicals covered by TSCA that they produce above this 25,000-pound threshold during every fifth year. EPA's estimate of more than 6,000 is based on data chemical companies submitted during the 2005 calendar year.

from them. For example, some parents are concerned about the invasiveness of taking blood samples from their children, and certain other fluids, such as umbilical cord blood or breast milk, are available only in small quantities and only at certain times. Thus, when samples are available from children, they may not be large enough to analyze.

A second reason we reported for the agency's limited use of biomonitoring data is that EPA often lacks the additional information needed to make biomonitoring studies useful in its risk assessment process. In this regard, biomonitoring provides information only on the level of a chemical in a person's body but not the health impact. The detectable presence of a chemical in a person's blood or urine does not necessarily mean that the chemical causes harm. While exposure to larger amounts of a chemical may cause an adverse health impact, a smaller amount may be of no health consequence. In addition, biomonitoring data alone do not indicate the source, route, or timing of the exposure, making it difficult to identify the appropriate risk management strategies. For most of the chemicals studied under current biomonitoring programs, more data on chemical effects are needed to understand whether the levels measured in people pose a health concern, but EPA's ability to require chemical companies to develop such data is limited. As a result, EPA has made few changes to its chemical risk assessments or safeguards in response to the recent proliferation of biomonitoring data. For most chemicals, EPA would need additional data on the following to incorporate biomonitoring into risk assessment: health effects; the sources, routes, and timing of exposure; and the fate of a chemical in the human body. However, as we have discussed in prior reports, EPA will face difficulty in using its authorities under TSCA to require chemical companies to develop health and safety information on the chemicals. In January 2009, we added transforming EPA's process for assessing and controlling toxic chemicals to our list of high-risk areas warranting attention by Congress and the executive branch.[4] Subsequently, the EPA Administrator set forth goals for updated legislation that would give EPA the mechanisms and authorities to promptly assess and regulate chemicals.

EPA has used some biomonitoring data in chemical risk assessment and management, but only when additional studies have provided insight on the health implications of the biomonitoring data. For example, EPA was able to use biomonitoring data on methylmercury—a neurotoxin that

[4]GAO, *High-Risk Series: An Update*, GAO-09-271 (Washington, D.C.: January 2009).

accumulates in fish—because studies have drawn a link between the level of this toxin in human blood and adverse neurological effects in children. EPA also used both biomonitoring and traditional risk assessment information to take action on certain perfluorinated chemicals. These chemicals are used in the manufacture of consumer and industrial products, including nonstick cookware coatings; waterproof clothing; and oil-, stain-, and grease-resistant surface treatments.

EPA Has Taken Steps to Improve the Usefulness of Biomonitoring Data but Lacks a Comprehensive Research Strategy

EPA has several biomonitoring research projects under way, but the agency has no system in place to track progress or assess the resources needed specifically for biomonitoring research. For example, EPA awarded grants that are intended to advance the knowledge of children's exposure to pesticides through the use of biomonitoring and of the potential adverse effects of these exposures. The grants issued went to projects that, among other things, investigated the development of less invasive biomarker than blood samples—such as analyses of saliva or hair samples—to measures of early brain development. Furthermore, EPA has studied the presence of an herbicide in 135 homes with preschool-age children by analyzing soil, air, carpet, dust, food, and urine as well as samples taken from subject's hands. The study shed important light on how best to collect urine samples that reflect external dose of the herbicide and how to develop models that simulate how the body processes specific chemicals. Nonetheless, EPA does not separately track spending or staff time devoted to biomonitoring research. Instead, it places individual biomonitoring research projects within its larger Human Health Research Strategy. While this strategy includes some goals relevant to biomonitoring, EPA has not systematically identified and prioritized the data gaps that prevent it from using biomonitoring data. Nor has it systematically identified the resources needed to reach biomonitoring research goals or the chemicals that need the most additional biomonitoring-related research.

Also, EPA has not coordinated its biomonitoring research with that of the many agencies and other groups involved in biomonitoring research, which could impair its ability to address the significant data gaps in this field of research. In addition to the CDC and EPA, several other federal agencies have been involved in biomonitoring research, including the U.S. Department of Health and Human Service's Agency for Toxic Substances and Disease Registry, entities within the U.S. Department of Health and Human Service's NIH, and the U.S. Department of Labor's Occupational Safety and Health Administration. Several states have also initiated biomonitoring programs to examine state and local health concerns, such

as arsenic in local water supplies or populations with high fish consumption that may increase mercury exposure. Furthermore, some chemical companies have for decades monitored their workforce for chemical exposure, and chemical industry associations have funded biomonitoring research. Finally, some environmental organizations have conducted biomonitoring studies of small groups of adults and children, including one study on infants.

As we previously reported, a national biomonitoring research plan could help better coordinate research and link data needs with collection efforts.[5] EPA has suggested chemicals for future inclusion in the CDC's National Biomonitoring Program but has not gone any further toward formulating an overall strategy to address data gaps and ensure the progress of biomonitoring research. We have previously noted that to begin addressing the need for biomonitoring research, federal agencies will need to strategically coordinate their efforts and leverage their limited resources.[6] Similarly, the National Academies of Science found that the lack of a coordinated research strategy allowed widespread exposures to go undetected, including exposure to flame retardants known as polybrominated diphenyl ethers—chemicals which may cause liver damage, among other things, according to some toxicological studies. The academy noted that a coordinated research strategy would require input from various agencies involved in biomonitoring and supporting disciplines. In addition to EPA, these agencies include the CDC, NIH, the Food and Drug Administration, and the U.S. Department of Agriculture. Such coordination could strengthen efforts to identify and possibly regulate the sources of the exposure detected by biomonitoring, since the most common sources—that is, food, environmental contamination, and consumer products—are under the jurisdiction of different agencies.

We have recommended that EPA develop a comprehensive research strategy to improve its ability to use biomonitoring in its risk assessments.[7] However, though EPA agreed with our recommendation, the agency still lacks such a comprehensive strategy to guide its own research efforts. In addition, we recommended that EPA establish an interagency

[5]GAO-09-353.

[6]GAO, *Toxic Chemicals: Long-Term Coordinated Strategy Needed to Measure Exposures in Humans*, GAO/HEHS-00-80 (Washington, D.C.: May 2, 2000).

[7]GAO-09-353.

task force that would coordinate federal biomonitoring research efforts across agencies and leverage available resources. If EPA determines that further authority is necessary, we stated that it should request that the Executive Office of the President establish an interagency task force to coordinate such efforts. Nonetheless, EPA has not established such an interagency task force to coordinate federal biomonitoring research, nor has it informed us that it has requested the Executive Office of the President do so.

EPA's Authority to Obtain Biomonitoring Data under TSCA Is Untested and May Be Limited

EPA has not determined the extent of its authority to obtain biomonitoring data under TSCA, and this authority is generally untested and may be limited. Several provisions of TSCA are potentially relevant. For example, under section 4 of TSCA EPA can require chemical companies to test chemicals for their effects on health or the environment.[8] However, biomonitoring data indicate only the presence of a chemical in a person's body and not its impact on the person's health. EPA told us that biomonitoring data may demonstrate chemical characteristics that would be relevant to a chemical's effects on health or the environment and that the agency could theoretically require that biomonitoring be used as a methodology for developing such data. EPA's specific authority to obtain biomonitoring data in this way is untested, however, and EPA is only generally authorized to require the development of such data after meeting certain threshold risk requirements that are difficult, expensive, and time-consuming.[9] EPA may also be able to indirectly require the development of biomonitoring data using the leverage it has under section 5(e) of TSCA, though it has not yet attempted to do so.[10] Under certain circumstances, EPA can use this section to seek an injunction to limit or prohibit the

[8] 15 U.S.C. § 2603(a) (2006).

[9] To require testing, EPA must determine that there are insufficient data to reasonably determine or predict the effects of the chemical on health or the environment, and that testing is necessary to develop such data. The agency must also make one of two additional findings. The first is that a chemical may present an unreasonable risk of injury to human health or the environment. The second is that a chemical is or will be produced in substantial quantities, and that either (1) there is or may be significant or substantial human exposure to the chemical or (2) the chemical enters or may reasonably be anticipated to enter the environment in substantial quantities.

[10] 15 U.S.C. § 2604(e) (2006).

manufacture of a chemical.[11] As an alternative, EPA sometimes issues a consent order that subjects manufacture to certain conditions, including testing, which could include biomonitoring. While EPA may not be explicitly authorized to require the development of such test data under this section, chemical companies have an incentive to provide the requested test data to avoid a more sweeping ban on a chemical's manufacture. EPA has not indicated whether it will use section 5(e) consent orders to require companies to submit biomonitoring data.

Other TSCA provisions allow EPA to collect existing information on chemicals that a company already has, knows about, or could reasonably ascertain.[12] For example, section 8(e) requires chemical companies to report to EPA any information they have obtained that reasonably supports the conclusion that a chemical presents a substantial risk of injury to health or the environment.[13] EPA asserts that biomonitoring data are reportable as demonstrating a substantial risk if the chemical in question is known to have serious toxic effects and the biomonitoring data indicate a level of exposure previously unknown to EPA. Industry has asked for more guidance on this point, but EPA has not yet revised its guidance. Confusion over the scope of EPA's authority to collect biomonitoring data under section 8 (e) is highlighted by the history leading up to an EPA action against the chemical company E. I. du Pont de Nemours and Company (DuPont). Until 2000, DuPont used the chemical PFOA to make Teflon®. In 1981, DuPont took blood from several female workers and two of their babies. The levels of PFOA in the babies' blood showed that PFOA had crossed the placental barrier. DuPont also tested the blood of twelve community members, 11 of whom had elevated levels

[11] Under section 5(e), when a company proposes to begin manufacturing a new chemical or to introduce an existing chemical for a significant new use, EPA may determine (1) that the available information is not sufficient to permit a reasoned evaluation of the health and environmental effects of that chemical and (2) that in the absence of such information, the manufacture of the chemical may meet certain risk or exposure thresholds. If the agency does so, the Administrator can issue a proposed order limiting or prohibiting the manufacture of the chemical. If a chemical company objects to such an order, the matter becomes one for the courts. If a court agrees with the Administrator, it will issue an injunction to the chemical company to limit or prohibit manufacture of the chemical. If and when the chemical company submits data to EPA sufficient for the Administrator to make a reasoned determination about the chemical's health and environmental effects, which may include test data, the injunction can be dissolved. Thus, an injunction would provide an incentive for the chemical company to develop testing data.

[12] 15 U.S.C. §§ 2604(a), 2604(b), 2607(a), 2607(d), 2607(e) (2006).

[13] 15 U.S.C. § 2607(e) (2006).

of PFOA in their blood. DuPont did not report either set of results to EPA. After EPA received the results from a third party, DuPont argued that the information was not reportable under TSCA because the mere presence of PFOA in blood did not itself support the conclusion that exposure to PFOA posed any health risks. EPA subsequently filed two actions against DuPont for violating section 8(e) of TSCA by failing to report the biomonitoring data, among other claims. DuPont settled the claims but did not admit that it should have reported the data. However, based on the data it had received, EPA conducted a subsequent risk assessment, which contributed to a finding that PFOA was "likely to be carcinogenic to humans." In turn, this finding contributed to an agreement by DuPont and others to phase out the use of PFOA by 2015. However, EPA's authority to obtain biomonitoring data under section 8(e) of TSCA remains untested in court.

Given the uncertainties regarding TSCA authorities, we have recommended that EPA should determine the extent of its legal authority to require companies to develop and submit biomonitoring data under TSCA. We also recommended that EPA request additional authority from Congress if it determines that such authority is necessary. If EPA determines that no further authority is necessary, we recommended that it develop formal written policies explaining the circumstances under which companies are required to submit biomonitoring data. However, EPA has not yet attempted a comprehensive review of its authority to require the companies to develop and submit biomonitoring data. The agency did not disagree with our recommendation, but commented that a case-by-case explanation of its authority might be more useful than a global assessment. However, we continue to believe that an analysis of EPA's legal authority to obtain biomonitoring data is critical.

Mr. Chairman, this concludes my prepared statement. I would be pleased to respond to any questions that you or other Members of this Subcommittee may have.

GAO Contact and Staff Acknowledgements

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Testimony
Before the Subcommittee on Superfund, Toxics and Environmental Health
Committee on Environment and Public Works
United States Senate

Statement for hearing entitled, "Current Science on Public Exposures to Toxic Chemicals"

Statement of

Linda Birnbaum, Ph.D., DABT, ATS

Director, National Institute of Environmental Health Sciences, National Institutes of Health, and Director, National Toxicology Program
U.S. Department of Health and Human Services

For Release on Delivery
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Mr. Chairman and distinguished members of the Subcommittee, I am pleased to appear before you today to present testimony on recent science related to exposure assessment.

Since human disease sometimes results from the interactions of our genetic susceptibilities and our environmental exposures,[1] having reliable data on exposure is essential to planning for and carrying out research on how environmental exposures initiate or promote disease.

From the days when one outdoor monitor in a city would be used to measure air pollution for a study to backpack monitors to badges pinned on a shirt, our ability to measure exposure continues to improve significantly.

In this statement, I shall describe some examples of research where exposure in the U.S. population or a vulnerable subgroup drove or changed the research agenda, some studies exploring the initiation or promotion of disease related to environmental exposures and the efforts made by researchers to use the best possible exposure data, and some of the technologies to assess exposures under development by the National Institute of Environmental Health Sciences (NIEHS), which is part of the National Institutes of Health, an agency of the U.S. Department of Health and Human Services (HHS).

The importance of biomonitoring cannot be underestimated. It can tell us three things: whether exposure to humans is actually occurring; at what levels; and how widespread the exposure is in the population. Sometimes the information is new; other times it confirms something we already suspected based on what we know about how a manmade or naturally occurring compound is used and where it is found in the environment. And occasionally, we are surprised by the results.

Findings from biomonitoring studies often trigger new research, either toxicology or population-based studies to investigate potential adverse health outcomes. One example is the surprising finding by HHS's Centers for Disease Control and Prevention (CDC) in 2002 of high levels of tungsten in urine of residents of Churchill County, Nevada, the site of a childhood leukemia cluster. Since we do not know enough about tungsten to understand whether this is a health risk, this discovery prompted the National Toxicology Program (NTP) to initiate studies on tungsten. More recently, CDC national blood and urine data showing widespread U.S. population exposure to parabens, triclosan and oxybenzone were an important factor in the decision to conduct additional toxicology studies for these compounds. What biomonitoring cannot tell us is the source of the exposure. For example, bisphenol A exposure is widespread in the population as evidenced by urinary levels in biomonitoring studies. We suspect that much of the bisphenol A exposure is coming from food and beverage containers. The findings from a small CDC study that premature infants in neonatal intensive care units (NICUs) had substantial levels of bisphenol A in urine indicated other sources of exposure. In a recently published study, the authors suspect the source was the presence of bisphenol A in polyvinyl chloride-containing medical devices used in NICUs.[2] Since premature infants represent a uniquely sensitive

[1] The World Health Organization defines exposure as the contact between an agent and a target. The target may be an individual or a population; it can be an organ, a tissue, or a cell. The agent of exposure can be a biological, physical, or psychosocial stressor.

[2] Exposure to Bisphenol A and Other Phenols in Neonatal Intensive Care Unit Premature Infants. Environ Health Perspect 117:639–644 (2009).

subpopulation, additional research is being carried out to understand the health risks of such exposures. Many if not all of the substances in CDC's biomonitoring program are included because of evidence that they pose potential human health hazards. For example, DEHP and other phthalates were included in CDC's first National Exposure Report because of known adverse developmental and reproductive effects in rodents, as identified in NTP and other toxicological studies. The same is true for heavy metals, certain pesticides, polycyclic aromatic hydrocarbons (PAHs), etc. As new hazards are identified from toxicological research, these compounds become good candidates for inclusion in national biomonitoring studies, e.g., brominated flame retardants. Once biomonitoring studies show us the range and nature of exposures occurring in the general U.S. population, the cycle continues as additional toxicological and epidemiological research is triggered to increase our knowledge on specific adverse health risks.

The Agricultural Health Study is a cohort study of 57,000 licensed pesticide applicators and 32,000 of their spouses in Iowa and North Carolina. NIEHS scientists, in collaboration with colleagues from NIH's National Cancer Institute (NCI), the Environmental Protection Agency (EPA) and CDC's National Institute for Occupational Safety and Health (NIOSH), have carried out biomonitoring studies of subgroups of pesticide applicators using specific chemicals and have used these data to validate and refine their questionnaire-based exposure algorithms for the much larger study population. In other studies conducted within the cohort, researchers have collected house/farm dust and biological samples to assess exposure to pesticides, endotoxins, and metals as well as gene variants that may affect risk of specific health outcomes.

In preparation for studies to assess health-related risks to mothers and their offspring, NIEHS researchers are assessing the validity of using blood and urine samples at a single time during pregnancy. They are assessing the agreement between measures from samples taken at three points during pregnancy to determine if a single sample is reliable enough to assess risk related to phthalates, pesticides, bisphenol A, and perfluorooctanoic acid (PFOA).

An example of biomonitoring where surprising results triggered additional research occurred in one of the NIEHS/NCI Breast Cancer and Environment Centers studies. Researchers found unexpectedly high levels of PFOA in girls in one school district. The source of the exposure could not be determined. The researchers worked with their community partners who had been involved in the program from the beginning to survey families about how they wanted to receive study results. In response to the survey, the researchers and the community partners produced a newsletter to provide updates to the families. The researchers have since received a second grant to identify possible sources and health effects of PFOA.

Sometimes an event changes an exposure in a population allowing a "before" and "after" comparison. In a study of infants born before the EPA's regulatory actions to phase out residential use of chlorpyrifos and diazinon, the association between birth weight and length and cord blood levels of these insecticides was highly significant. Among infants born after January

2001, exposure levels were substantially lower and no association with fetal growth was apparent.[3]

Concern about widespread exposure in the U.S. population often guides the NIEHS research agenda. In 1974, NIEHS launched a classic study in six cities to explore the associations between air pollution produced by fossil fuels and respiratory health in large cohorts of adults and children which provided a wealth of information. Collaboration with EPA led to expansion of this study to include more cities and confirmed the negative effects of air pollution on human health. In 1978, the NTP tested yellow paint on pencils; there was no evidence of carcinogenicity in rats or mice.[4] In a 2009 study of the effects of PAHs on children's IQ in New York City (Washington Heights, Harlem, and the South Bronx), the mothers' exposure as measured during their pregnancies by wearing backpack monitors was associated with a decrease in IQ among the more exposed children. The extent of this effect was similar to that of low-level lead exposure.[5]

By 2015, the use of engineered nanomaterials (ENMs) and nano-enabled devices is expected to exceed $3 trillion, resulting in exposures with possible unknown consequences to health and the environment. A key first step in understanding risk is to develop ways to measure exposures. NIEHS is supporting studies with funding from the American Recovery and Reinvestment Act of 2009 (ARRA) to conduct real-time, on-site measurement of exposures with a suite of instruments to characterize ENMs during different phases of the production process. The next step is to understand their interactions with biological systems and the resulting health risks. Again using ARRA funding, NIEHS is supporting studies on ENM-induced inflammatory and oxidative stress responses in multiple cell culture systems with the goal of finding biomarkers of response. This information will provide cell-specific and ENM-specific toxicity profiles. Other studies are looking at the fundamental interactions of ENMs at the cellular and molecular levels. ARRA funding is also supporting studies using animals to determine organ specific health effects and to evaluate human health risks of ENMs. Studies planned include research on the following:

- The effect of inhaled ENMs on the respiratory tract, brain, liver, and other organs;
- Whether inhaled cadmium nanoparticles can cross the placenta and influence fetal stability and development; and
- Pulmonary effects of ENMs to understand whether they modify the effects of other agents (e.g., drugs, vaccines) while having minimal effects on their own, enhance allergen sensitization, or alter innate immunity.

NIEHS is also supporting research on nanotechnologies to improve environmental monitoring. Detection devices under development include:

- a microsensor for detecting formaldehyde in air
- nanobiosensors for probing chemical exposures and their effects on individual cells

[3] Prenatal insecticide exposures and birth weight and length among an urban minority cohort. Environ Health Perspect 112:1125-1132 (2004).
[4] Bioassay of diarylanide yellow for possible carcinogenicity CAS No. 6358-85-6. National Cancer Institute Carcinogenesis Technical Report Series No. 30 (1978) http://ntp.niehs.nih.gov/ntp/htdocs/LT_rpts/tr030.pdf - 2234.3KB
[5] Prenatal airborne polycyclic aromatic hydrocarbon exposure and child IQ at age 5 years. Pediatrics 124:e195-e202 (2009).

- wearable nanosensors (approximately 4”x4”) for real-time monitoring of diesel and gasoline exhaust
- Low-cost, portable sensors for measuring metals such as arsenic and mercury at hazardous waste sites.

Investigators studying the interplay of genetic and environmental factors in the risk for Parkinson’s disease have developed a new model to estimate residential exposure of individuals to pesticide drift from nearby farms. The exposure model uses a geographic information system that combines data on home addresses, land use, and pesticide applications. By measuring the proximity of residences to the fields where pesticides were applied, this model allows estimation of exposures that occur with drift from application sites and/or travel through soil to water wells. One of their studies showed that residential exposure to a combination of the herbicide paraquat and the fungicide maneb increases the risk of Parkinson’s disease.[6] Another study revealed that estimated pesticide exposures from drift and from well water contamination combined to increase risk of Parkinson’s disease.[7]

An investigator in California is using ARRA funding to improve exposure modeling in a study of birth outcomes related to exposure to pollution from traffic. Using real-time global positioning system (GPS) tracking and detailed activity questionnaires to determine locations more accurately, the model will assess pregnant women’s exposure to traffic-related air pollution with greater precision.

In the NIEHS Sister Study of 51,000 women whose sisters have breast cancer, researchers have collected urine, blood, toenail, and dust samples to provide a snapshot of environmental exposures at the time of enrollment in the study. The study will assess exposure to pesticides, other hormonally active compounds such as bisphenol A and phthalates, toxic metals, trace metals, vitamin D, specific micronutrients, and hormones. Samples will also be used to measure gene variants that may be related to disease risk. The study design will allow researchers to assess the associations between breast cancer and other diseases with these markers of exposure, nutrition and health status. Ultimately these data will be used in studies of gene-environment interactions. These data will also be used in conjunction with self-reported questionnaire data to develop questionnaire-based exposure measures and to validate both questionnaire-based methods and the use of single biological samples. Lastly, these data will support mechanistic studies of specific pathways leading to breast cancer risk and to develop markers for early detection or for predicting progression of disease.

Determining actual levels of exposure for use in research, risk assessment, and risk management is an ongoing challenge, and NIEHS is actively pursuing many research approaches to help solve this problem and thus promote more accurate science and better decision making. For example, the NIEHS is supporting development and testing of a robot called PIPER[8] capable of mimicking children’s floor activities while collecting better estimates of young children’s

[6] Parkinson’s Disease and residential exposure to maneb and paraquat from agricultural applications in the central valley of California. Am J Epidemio 169(8):919-926 (2009)
[7] Well-water consumption and Parkinson’s Disease in rural California. Environ Health Perspect 117:1912-1918 (2009).
[8] Pretoddler Inhalable Particulate Environmental Robotic

exposure to indoor air pollutants (particulate matter, pesticides, allergens, endotoxins and airborne fungi). A study of asthma and indoor environmental contaminants is currently underway to test PIPER in the homes of 200 children. The study will compare measurements of particulates obtained by PIPER with those from standard adult height monitoring stations and examine their association with asthma symptoms.

The NIEHS has the lead for the Exposure Biology Program of the trans-NIH Genes, Environment and Health Initiative. The Program is funding 32 projects focusing on the development of innovative technologies to measure environmental exposures, diet, physical activities, psychosocial stress, and others factors that contribute to disease development. In addition to developing new measures of exposure, the program also supports the development of markers of biological response and DNA damage, as well as the development of biosensors based on monitoring biological responses. A critical aspect is the integration of these technologies to enable a more accurate understanding of exposure. For example, the combination of physical activity measurements with particulate matter exposure allows for an improved estimate of individual dose. With the additional inclusion of GPS analysis, this information can potentially be used to identify the sources of these exposures and guide the development of interventions to improve public health.

In summary, understanding the connection between our health and our environment, with its mixture of chemicals, diet and lifestyle stressors, is no less complex than understanding the intricacies of the human genome. At NIEHS, we remain committed to helping the field of exposure science evolve to meet emerging public health challenges. We look forward to the increased contributions of exposure scientists as we work to understand the role of environment in the etiology of disease.

Mr. Chairman and members of the Subcommittee, I am pleased to present testimony on this important issue and would be happy to answer any questions.

Testimony of Molly Jones Gray,
To the Senate Environment and Public Work's Committee
February 4th 2010

It is my pleasure to be here. My name is Molly Jones Gray. I come here before you today as a concerned mother. I recently participated as one of the women in a Washington Toxics Coalition study called "Earliest Exposures." This was a study of pregnant women to investigate what toxins our developing fetuses were exposed to during pregnancy.

Earliest Exposures- A research study by WA Toxics Coalition
The study tested for phthalates, mercury, PFC "Teflon chemicals", flame-retardants, and BPA. Many of these substances are known to cause adverse health effects such as reproductive problems, cancer, hormone disruption, and impaired neurodevelopment. My results were higher than the national average in many of the substances tested. In fact, I had the highest mercury of all the pregnant women tested.

During the five years preceding the study, I struggled with fertility and repeated miscarriages. As I searched for an answer to why I was having such a hard time bringing a child to term, I discovered the connection between our external environment, chemical exposures and their effect on our health, particularly reproductive systems. At that time, I made reasonable changes in my life to reduce my exposure to toxic chemicals from all routes of entry- air, food, drink, and skin. I did my very best to eat organic food, low mercury seafood and use personal care products without phthalates and fragrances.

Personal Reflection on My Test Results
When I first heard of the study about chemicals in pregnancy, I was extremely interested in participating. I wanted to see if my best intentions made a difference. The answer I received was incredibly disheartening. I was shocked that my levels were as high as they were. I learned that this fight to avoid toxins is larger than one person alone! These chemicals are ubiquitous in the environment and as clean as I tried to be, it was not enough to protect my baby boy.

Mother's- to- be can make many choices to ensure a healthy baby- we can take prenatal vitamins, exercise, avoid cigarettes and alcohol, and eat healthy diets. I am disappointed that with all of the choices we are able to make we do not have a choice to protect our children from the powerful influence of toxic chemicals on their developing bodies.

Now that my son is 7 months old, people often ask me if my son is healthy. My answer to that is as far as I know he is a healthy happy boy. My concerns are of the unknown. We have no idea what the long-term health implications of these

results are and I do not want my precious son or other children to be our scientific experiment.

Changes Needed to Protect All Children
Developing babies are uniquely vulnerable to insult as they are developing at a rapid pace. Toxic exposures at crucial points in development could affect the wellbeing for a lifetime. In addition, fetuses have been found to have immature detoxification pathways. They cannot clear toxins as well as adults.

Something is wrong when I, as an educated consumer, am unable to protect my baby from toxic chemicals. I and all other parents should be able to walk into stores and buy what we need without winding up with products that put our families' health at risk. Now that I've learned that companies can put chemicals into products without ever testing for whether they harm our health, I think we need to change our laws.

On behalf on my son Paxton and all other children, I am asking for your help to lower our body burdens of chemicals that come between us and our health.

In order to do that I am asking Congress to take immediate steps to eliminate the use of persistent toxic chemicals — those that build up in our bodies or are passed on to the next generation. Legislation should also reduce the use of chemicals that have known serious health effects and ensure that only the safest chemicals are created and used in everyday products. Finally, we need standards that protect our most vulnerable populations like pregnant women and developing fetuses.

Conclusion
I am disappointed that toxic chemicals like the ones found in my body in pregnancy are in our environment, our personal care products, our clothes, our furniture, our baby toys, and our food. Babies deserve to grow and develop in a healthy environment, in utero and out. But babies are born everyday already exposed to toxins linked to serious health problems. Safe until proven harmful is not good enough for my baby or me. I want our country to value the lives of its children the same way I value and love my son. It will take time to rid our population of this burden on our bodies- we need to start now. This is not my story alone—this is the story of all of our children, grandchildren and future generations. Thank you for this opportunity to tell my story.

Testimony of Kenneth A. Cook

President
Environmental Working Group

Before the

SUBCOMMITTEE ON
SUPERFUND, TOXICS AND ENVIRONMENTAL HEALTH
U.S. SENATE COMMITTEEE ON ENVIRONMENT & PUBLIC WORKS

On

"Current Science on Public Exposures to Toxic Chemicals"

Thursday, February 4, 2010

Mr. Chairman and distinguished Members of the Subcommittee: My name is Kenneth A. Cook, and I am the President and Co-Founder of Environmental Working Group (EWG), a nonprofit research and advocacy organization based here in Washington, DC, with offices in Ames, Iowa, and Oakland, California. I thank the members of the subcommittee for holding this important hearing and for the opportunity to testify.

Emerging science on human exposure has transformed the debate over toxic chemicals policy. This morning, I would like to talk to you about 10 Americans whose exposure to toxic chemicals has had an important impact on that policy debate. EWG tested these 10 Americans in 2004 and found more than 200 synthetic industrial chemicals in their blood, including dioxins and furans, flame retardants, and active ingredients in stain removers and carpet protectors. We also found lead, polychlorinated biphenyls (PCBs), and pesticides that the federal government banned more than 30 years ago.

We do not know much about these 10 Americans, but we do know a little about how they were exposed. Their chemical exposures did not come from the air they breathed, the water they drank, or the food they ate. They were not exposed at work or at school. They did not encounter these chemicals in personal care products or cleaning agents they used.

How do we know? These 10 Americans were newborns. The more than 200 chemicals we found in their umbilical cord blood crossed the placenta to contaminate the babies before birth. Our research uncovered a startling truth — babies are coming into the world pre-polluted with toxic chemicals.

EWG commissioned this biomonitoring study and obtained cord blood samples from the American Red Cross. We tested ten of them at a cost of $10,000 per sample. Then last year, we examined the cord blood of another group of 10 Americans — children of African American, Asian-Pacific, and Latino heritage. We found similar unsettling results, including the first national detections in cord blood in the United States of the endocrine-disrupting chemical bisphenol A (BPA) and the thyroid toxin perchlorate.

We found no significant differences in results between the two studies. Instead, we discovered that we are all united by the disturbing reality that toxic pollution begins in the womb.

The current science makes clear that we must reform the Toxic Substances Control Act (TSCA) to ensure that industry submits pre-market evidence that its chemicals are safe for kids, our most vulnerable population. Each day brings another jarring headline as new research documents the health dangers of these exposures.

My testimony focuses on the current science of human exposure to toxic chemicals. But I want to thank you, Mr. Chairman, for your leadership over the past five years to put children's' exposure to toxic chemicals at the forefront of a policy debate that is long overdue — the debate over how to reform the 34-year-old Toxic Substances Control Act. You may not have realized it at the time, but when you named your reform proposal the "Kid-Safe Chemicals Act", Mr. Chairman, you instantly engaged millions of people in the debate over toxic chemicals.

Conduct a Google search for the (exact) phrase "Kid-Safe Chemicals Act" today and you find an extraordinary 554,000 links on the Web, including literally tens of thousands of entries about your bill in blogs, newspaper articles, discussion groups, and other online publications, written by parents, journalists, medical professionals, educators, and scientists. State legislators from Maine to Washington and numerous other states in between subsequently followed your lead and used the phrase "child-safe" or "kid-safe" in naming their initiatives for chemical policy.

And research shows time and again something that you have known throughout your career, Mr. Chairman: focus an issue through its impacts on children, their health and well being, and the American people get it. For anyone who wants proof, I would point to the current struggle to arrive at bipartisan consensus on health care reform. And I would contrast it to the successful, bipartisan effort that ultimately resulted in a major health care reform in 2009 after years of strong bipartisan support: the major expansion of the State Children's Health Insurance Program.

BIOMONITORING REVEALS EXPOSURES TO HUNDREDS OF CHEMICALS

The Centers for Disease Control and Prevention (CDC) calls biomonitoring "the most health-relevant assessment of exposure" and warns that "[f]or children age 5 years and younger, minimal information exists on exposure to priority environmental chemicals, and this lack of information is a major gap in protecting children from harmful exposures"(CDC 2010). EWG's umbilical cord study set out to address this gap. Our researchers conducted a comprehensive survey of the published scientific literature, identifying every study in which scientists had tested umbilical cord blood for industrial chemicals. EWG's findings agree with CDC's — the peer-reviewed literature contains surprisingly little biomonitoring information for newborns. The vast majority of chemicals found in cord blood were first identified in EWG-led research. Altogether, biomonitoring studies have found up to 358 chemicals in cord blood from U.S. newborns (see ATTACHMENT A).

Detection of a chemical in umbilical cord blood does not prove that it will cause harm. As researchers have mapped more and more of the "human toxome," however, scientists, public health experts, and policymakers have embraced biomonitoring as the logical foundation for changing the way government regulates industrial chemicals. There is now widespread agreement that cord blood monitoring should be the starting point. The Kid-Safe Chemicals Act, S. 3040, introduced in the 110th Congress would prioritize safety assessments by focusing first on the chemicals that show up in people. The law would require phasing out production and use of toxic chemicals found in umbilical cord blood unless rigorous, expedited testing showed them to be safe.

CHEMICALS AT PARTS-PER-BILLION LEVELS ARE LINKED TO DISEASE

CDC's biomonitoring studies have revealed the presence of scores of chemicals in the blood and urine of Americans, often at concentrations as low as a few parts-per-billion (ppb). Such low levels may sound trivial, but science shows that chemicals can be biologically active even in the ppb range. In fact, many commonly prescribed medications are biologically active at concentrations in that range and below. Two examples are Cialis, which is active in the body at levels as low as 30 ppb, and the birth control device, Nuvaring, whose estrogen component is clinically effective at 0.035 ppb. At these tiny doses, these drugs can initiate procreation or prevent it. The fact that pharmaceuticals can exert their clinical effects at very low concentrations makes clear that industrial chemicals may do the same. In addition, an increasing number of toxicity studies are done at concentrations that mimic environmental exposures. If animal studies find effects at very low exposures, we must strongly consider the possibility that there are biological effects in humans. Simply put, low doses do matter.

Epidemiological studies have long since established critical links between environmental exposures and adverse health effects, including the relationship between tobacco exposure and lung cancer (Blair et al 2009). Recent biomonitoring studies have discovered associations between exposure to various industrial and consumer chemicals and adverse health effects, including reduced birth weight and head circumference in newborns, thyroid disease, aggressive behavior in children, and difficulty conceiving (Table 1). In just the last year, researchers using data from the National Health and Nutrition Examination Surveys (NHANES) have linked thyroid and heart disease to exposures to compounds such as perfluorochemicals (PFCs) and BPA respectively (Melzer et al 2010a, Melzer et al 2010b).

Table 1: Studies show everyday chemical exposures are linked to serious adverse health effects

Chemical	Study Population	Finding	Range of concentrations in population studied (ppb)
Phthalates	Infant boys (n=85)	Boys with higher prenatal exposure to phthalates (measured in maternal urine) had decreased anogenital distance (Swan et al 2005).	Mono-isobutyl phthalate (MiBP): Not detected (ND) to >7.7 Mono-benzyl phthalate (MBzP): ND to >25.8 Mono-n-butyl phthalate (MBP): ND to >38.7 Mono-ethyl phthalate (MEP): ND to >1076
Bisphenol A (BPA)	Children (n=249)	Parents of children with higher exposure to BPA during early pregnancy (as measured in maternal urine) report higher incidence of behavioral effects in daughters, including increased aggression and hyperactivity (Braun et al 2009).	ND to >37.3
Bisphenol A (BPA)	Adults (n=2,605)	Adults with higher BPA levels in urine reported higher prevalence of cardiovascular disease (Melzer et al 2010a).	ND to 80.1
Brominated flame retardants (PBDEs)	Newborns (n=288)	Newborns with higher levels of certain PBDEs in cord blood serum had decreased levels of thyroid hormones	Bromodiphenyl ether congener 47 (BDE-47): 1.1 to 311 BDE-100: 0.5 to 77

		critical to normal brain development (Herbstman et al 2008).	
Perfluorochemicals (PFCs)	Newborns (n=293)	Newborns with higher levels of two PFCs in cord blood serum, PFOA and PFOS, were found to have lower birth weight and head circumference (Apelberg et al 2007).	Perfluorooctane sulfonate (PFOS): ND to 34.8 Perfluorooctanoic acid (PFOA): 0.3 to 7.1
Perfluorochemicals (PFCs)	Adults (n=3,974)	Adults with higher levels of two PFCs in their blood serum, PFOA and PFOS, reported higher prevalence of thyroid disease (Melzer et al 2010b).	PFOA: 0.1 to 123 PFOS: 0.1 to 435
Brominated flame retardants (PBDEs)	Adult women (n=223)	Women with higher levels of certain PBDEs in their blood serum were found to have significant decreases in their ability to conceive (Harley et al 2010).	BDE-47: ND to >25.2 BDE-100: ND to >4

THE TOLL OF CHEMICAL POLLUTION ON HEALTH AND HEALTH COSTS

The last ten years have produced an avalanche of credible studies documenting the costs of diseases associated with toxic pollution. Our failure to protect the American people, and especially America's kids, from contamination by toxic chemicals has taken a tremendous toll on Americans' health and resulted in significant health care costs.

As of 2009, 182 human diseases in all had been linked to chemical exposures, according to researchers at the University of California-San Francisco and Boston Medical Center (Janssen 2008). These range from autism to birth defects to asthma to childhood cancer. Take, for example, neuro-developmental disease, which includes autism and autism spectrum disorders, speech and language disorders, learning disorders, and neurological and psychiatric disease. A Canadian study in 2001 estimated that as much as half of these afflictions may be the result of chemical exposures. The cost of treating and caring for the affected children was estimated at up to $83.5 billion a year (Muir 2001).

Toxic pollution has been linked to a variety of other childhood diseases. In 2002, researchers at the Mount Sinai School of Medicine calculated that all lead poisoning cases, 30 percent of all asthma cases, 10 percent of neurobehavioral disorders, and five percent of pediatric cancers were traceable to chemical exposures. The financial cost topped $55 billion annually as of 2002, which was nearly three percent of U.S. health care costs at the time (Landrigan 2002).

We also know that low dose chemical exposures can affect brain development in utero, in infants, and in children even when these exposures do not cause diagnosable disease. One result is lower IQ, which has huge implications for the future productivity and earning potential of affected children (Mendola 2002). Researchers at the National Institutes of Environmental Health Sciences and Mt. Sinai estimated that the figure for mercury poisoning alone is nearly $9 billion a year (Trasande 2005).

Other data suggests that toxic pollution may contribute to 80 percent of chronic childhood diseases. Mount Sinai's Philip Landrigan estimates that genetics account for only 10 to 20 percent of cases of chronic disease in childhood in the U.S. and other industrialized nations (Landrigan 2001). These include birth defects, the leading cause of infant death; developmental disorders such as attention deficit hyperactivity disorder and autism; asthma, which more than doubled in incidence from 1980

to 1996, according to the CDC (Moorman 2007); and childhood leukemia and brain cancer, on the rise since the 1970s (Gurney 1996; Linabery 2008). Dr. Landrigan's team and other specialists have determined that many diseases, from respiratory illness to immune, thyroid and neuropsychological deficits, are likely linked to environmental toxins (Etzel 2004; Sly 2008; Wigle 2008).

THE U.S. SPENDS MORE TO TEST FOR TOXIC CHEMICALS IN SOIL AND FISH THAN IN INFANTS

The federal government budgets far more to monitor soil, water, and air for chemical contamination than it spends to test for chemicals in people. The disparity is great. In 2008, for example, the government funded the CDC's human biomonitoring program, part of the National Health and Nutrition Examination Survey, at $13.6 million. Compare this to the $12 million spent on testing wildlife, including peregrine falcons in Alaska and the Arctic, for toxic chemicals (McClure 2009 and US Fish & Wildlife Service 2009). In 2008, the government paid $22.5 million to test large mouth bass, Charr, herring, eels, lamprey, minnows, and shad for persistent organic contaminants (USGS 2009). In 2008, EPA spent an estimated $300 million for soil and water testing under Superfund (EPA 2009a). Even the expansive National Children's Study, which EWG strongly supports, only includes a small fraction of its $179 million budget for the biomonitoring of 525 pregnant women. And until very recently, the federal budget for biomonitoring of cord blood was zero. We should allocate more resources to biomonitor the pollution in people.

EXTENT OF EXPOSURE IS LIKELY FAR GREATER THAN STUDIES HAVE SHOWN

Current biomonitoring studies cover just a small percentage of the chemicals that could be in our bodies. More than 80,000 chemicals have been registered for use in the U.S. since 1976, and more than 15,000 have been manufactured or imported in medium-to-high amounts in the past 25 years. Biomonitoring tests to date have involved less than one percent of those compounds. In its own work, EWG has tested more than 200 people over the past 15 years. We tested for 540 chemicals and detected up to 482 of them. The more chemicals we test for, the more we find. Meanwhile the research on biologically active low doses of toxic chemicals has exploded.

Some chemicals EWG found were banned 30 years ago. Scientists tend to rigorously investigate chemicals only after they are banned. The unfortunate reality is that we often know little about more recently introduced chemicals that are in our bodies now.

In addition to the need for more research, a recent EWG investigation showed that the identities of many new chemicals are kept hidden from the public (EWG 2010). EWG found that industry has placed "confidential business information" (CBI) claims on the identity of 13,596 new chemicals produced since 1976—nearly two-thirds of the 20,403 chemicals added to commerce in the past 34 years. A significant number of these secret chemicals are used everyday in consumer products, including artists' supplies, plastic products, fabrics and apparel, furniture, and items intended for use by children. EPA data shows that at least 10 of the 151 high volume confidential chemicals produced or imported in amounts greater than 300,000 pounds a year are used in products specifically intended for use by children.

TSCA's overbroad secrecy provisions threaten public health. Under section 8(e) of TSCA, companies must turn over all data showing that a chemical may present "a substantial risk of injury to health or the environment." By definition, compounds with 8(e) filings are the chemicals of the greatest health concern. In the first eight months of 2009, industry concealed the identity of the chemicals

in more than half the studies submitted under 8(e). Non-industry scientists and the public simply do not know how many of the chemicals that have been flagged as "posing a significant risk of injury to health or the environment" by industry, but are not identified by name because of CBI protections, could also be present in our bodies and in newborns.

RECOMMENDATIONS

In conclusion, we commend Administrator Jackson's call for TSCA reform and the steps that Assistant Administrator Owens has taken to address abuses of confidential business information claims. To protect our children's health, however, EPA needs strong authority from Congress to put the burden on industry to show a chemical is safe before it goes on the market. EPA must have express authority to require more transparency of chemical health and safety data. The federal government should use biomonitoring of cord blood to prioritize which of the 80,000 chemicals registered for use we should tackle first. Therefore, EWG looks forward to the re-introduction of the Kid-Safe Chemicals Act and urges Congress to take quick action to pass this necessary TSCA reform legislation.

We strongly support the CDC's existing biomonitoring programs and urge full funding of the national children's study. We urge CDC to consider umbilical cord monitoring as part of an expanded biomonitoring program. More funding for large, population-scale biomonitoring studies could fill this critical gap in data. Such studies could help scientists and policymakers to determine how infant exposure to chemicals in the womb varies across populations; what other industrial compounds may be present in umbilical cord blood; and what health risks those pollutants may pose, alone or in combination, to developing babies.

Thank you for your time. I welcome the opportunity to answer any questions you may have.

Attachments

ATTACHMENT A: Results of Select Cord Blood Biomonitoring Studies of American Infants

ATTACHMENT B: Government Spending on Testing Soil, Water & Air vs. Human Biomonitoring

ATTACHMENT C: Chart of Public Health Costs of Toxic Chemical Exposures

References

ATTACHMENT A

RESULTS OF SELECT CORD BLOOD BIOMONITORING STUDIES OF AMERICAN INFANTS
Nationally, cord blood biomonitoring studies have detected up to 358 chemicals

Chemical class	Chemical subclass	Summary of representative study	No. of newborns tested	Place of birth	No. of Chemicals found
Dioxin & Furan	Brominated dioxin	EWG tested cord blood from 10 newborns for 12 brominated dioxins and furans and found at least one of these chemicals in 7. In the 7 newborns, 6 to 7 different congeners were found. Mean total level was 12 pg/g lipids in blood serum. (EWG 2005)	10	U.S. hospitals	6-7
Dioxin & Furan	Brominated dioxin	EWG tested cord blood from 10 newborns of minority background for 12 brominated dioxins and furans and found at least one in 4 of the subjects. Six different congeners were found. Mean total level was 10.7 pg/g lipids in blood serum. (EWG 2009)	10	Mich. Fla. Wis. Mass. Calif.	6
Dioxin & Furan	Chlorinated dioxin	Researchers from the SUNY Health Science Center tested cord blood from 5 babies delivered via C-section from late 1995 to early 1996 for dioxins, dibenzofurans, and coplanar PCBs. Mean measured levels of total PCDDs, PCDFs, and coplanar PCBs were 165 pg/g for cord blood. (EWG 2005)	5	N.Y.	1
Dioxin & Furan	Chlorinated furan	EWG tested cord blood from 10 newborns for 17 chlorinated dioxins and furans and found at least one in all 10 subjects. Eleven different congeners were found. Mean total level was 56.3 pg/g lipids in blood serum. (EWG 2005)	10	U.S. hospitals	11
Dioxin & Furan	Chlorinated furan	EWG tested cord blood from 10 newborns of minority background for 17 chlorinated dioxins and furans and found at least one in all 10 subjects. Fifteen (15) different congeners were found. Mean total level was 59.7 pg/g lipids in blood serum. (EWG 2009)	10	Mich. Fla. Wis. Mass. Calif.	15
Fire Retardant	Brominated Fire Retardant	EWG measured TBBPA levels in cord blood from 10 newborns of minority background. TBBPA was found in 3 samples with a mean level of 11 ng/g lipids in blood serum. (EWG 2009)	10	Mich. Fla. Wis. Mass. Calif.	1
Metal	Cadmium	Researchers from Harvard measured cord blood concentrations of cadmium in 94 healthy babies, finding concentrations ranging from 0.003 to 0.210 ug/dl, with mean of 0.045 ug/dl. (Rabinowith 1984)	94	Boston, Mass.	1
Metal	Lead	Researchers from SUNY Oswego, the New York State Department of Health, the University of Albany, and Penn State University measured cord blood lead levels in 154 children and correlated lead levels with adrenocortical responses to acute stress in children. They divided cord blood levels into the following 4 quartiles: < 1.0 (1st quartile; n = 37),	154	N.Y.	1

Chemical class	Chemical subclass	Summary of representative study	No. of newborns tested	Place of birth	No. of Chemicals found
		1.1–1.4 ?g/dL (2nd quartile; n = 39), 1.5–1.9 ?g/dL (3rd quartile; n = 36), and 2.0–6.3 ?g/dL (4th quartile; n = 42). (Gump 2008)			
Metal	Lead	Researchers from Harvard University, Emory University, and University of Massachusetts at Amherst tested lead levels in cord blood from 527 babies born between 1993 and 1998 and found mean levels of 1.45 ug/dL. (Sagiv 2008)	527	New Bedford, Mass.	1
Metal	Mercury	Researchers from Columbia University and the CDC tested for cord blood levels of mercury in women who live and or work close to the World Trade Center site between Dec. 2001 and June 2002. The researchers found a mean cord mercury level of 7.82 ug/L. (Lederman 2008)	289	New York City, N.Y.	1
Musk	Musk	EWG measured nitro and polycyclic musk levels in cord blood from 10 newborns of minority background. Galoxolide was found in 6 samples at a mean level of 0.483 ng/g, and tonalide was found in 4 samples at a mean level of 0.147 ng/g. (EWG 2009)	10	Mich. Fla. Wis. Mass. Calif.	2
PAH	Polyaromatic hydrocarbons (PAHs)	Researchers from Columbia University measured levels of benzoA-pyrene DNA adduct levels in 203 babies from New York City mothers who were pregnant during 9/11. (Perera 2005)	203	New York City, N.Y.	1
PAH	Polyaromatic hydrocarbons (PAHs)	EWG tested cord blood from 5 newborns for 18 polyaromatic hydrocarbons and found at least one in all 5 subjects. Nine (9) different chemicals were found with total mean concentration of 279 ng/g lipids in blood serum. (EWG 2005)	5	U.S. hospitals	9
PBDE	Polybrominated diphenyl ether (PBDE)	Researchers from Columbia University and Johns Hopkins tested 297 cord blood samples from babies born at Johns Hopkins Hospital from Nov. 26, 2004 to March 16, 2005 for 8 PBDE congeners. They report that 94% of the samples contained at least one of the tested congeners. (Herbstman 2007)	297	Baltimore, Md.	7
PBDE	Polybrominated diphenyl ether (PBDE)	Researchers from Indiana University measured levels of 6 PBDEs in 12 paired samples of maternal and cord blood from live births that occurred from Aug. to Dec., 2001. They found that concentrations of PBDEs in both sets of samples were 20-to-106 fold higher than levels reported in a similar study from Sweden, leading them to conclude "human fetuses in the United States may be exposed to relatively high levels of PBDEs." (Mazdai 2003)	12	Indianapolis, Ind.	6
PBDE	Polybrominated diphenyl ether (PBDE)	EWG tested cord blood from 10 newborns for 46 polybrominated diphenol ethers (PBDEs) and found at least one of these chemicals in 10 out of 10 participants. Among all 10	10	U.S. hospitals	27-32

Chemical class	Chemical subclass	Summary of representative study	No. of newborns tested	Place of birth	No. of Chemicals found
		participants who tested positive for the chemicals, 27 to 32 different congeners were found. Mean total level was 4.53 ng/g lipids in blood serum. (EWG 2005)			
PBDE	Polybrominated diphenyl ether (PBDE)	EWG tested cord blood from 10 newborns of minority background for 46 polybrominated diphenyl ethers (PBDEs) and found at least one in all 10 samples. Among all 10 participants who tested positive for the chemicals, 26 to 29 different congeners were found. Mean total level was 72.9 ng/g lipids in blood serum. (EWG 2009)	10	U.S. hospitals	26-29
PBDE	Polybrominated diphenyl ether (PBDE)	Researchers at Columbia University and Johns Hopkins tested 288 cord blood samples from babies born at Johns Hopkins Hospital from Nov. 26, 2004 to March 16, 2005 for 3 PBDE congeners. In all the 288 subjects, all three congeners were found. (Herbstman 2008)	288	Baltimore, Md.	3
PBDE	Polybrominated diphenyl ether (PBDE) Metabolite	Researchers from the School of Public and Environmental Affairs at Indiana University tested PBDE and PBDE metabolities in 20 pregnant women and their newborn babies who had not been intentionally or occupationally exposed. They noted that metabolites in humans seem to be accumulating. (Qiu 2009)	20	Indianapolis, Ind.	10
PCB	Polychlorinated biphenyl (PCB)	Researchers at Columbia University and Johns Hopkins tested 297 cord blood samples from babies born at Johns Hopkins Hospital from Nov. 26, 2004 to March 16, 2005 for 35 PCB congeners. They report levels for 4 of the 35 but note that ">99% (of samples) had at least one detectable PCB congener." (Herbstman 2007)	297	Baltimore, Md.	18
PCB	Polychlorinated biphenyl (PCB)	Researchers from SUNY Oswego investigated cord blood levels of PCBs in children born between 1991 and 1994 and correlated levels with response inhibition when the children were 4.5 years of age. The researchers found that "results indicated a dose-dependent association between cord blood PCBs and errors of commission." (Stewart 2003)	293	Great Lakes states	7
PCB	Polychlorinated biphenyl (PCB)	EWG tested cord blood from 10 newborns for 209 polybrominated diphenol ethers (PBDEs) and found at least one of these chemicals in 10 out of 10 participants. Among all 10 participants who tested positive for the chemicals, 98 to 147 different congeners were found. Mean total level was 6.2 ng/g lipids in blood serum. (EWG 2005)	10	U.S. hospitals	98-147
PCB	Polychlorinated biphenyl (PCB)	EWG tested cord blood from 10 newborns of minority background for 209 polychlorinated biphenyls and found at least one in all 10 samples. Among all 10 participants who tested positive for the chemicals, 98 to 144 different congeners were found. Mean total level was 22.1 ng/g lipids in blood serum. (EWG 2009)	10	Mich. Fla. Wis. Mass. Calif.	98-144

Chemical class	Chemical subclass	Summary of representative study	No. of newborns tested	Place of birth	No. of Chemicals found
PCB	Polychlorinated biphenyl (PCB)	Researchers from Harvard, Emory, and the University of Massachusetts at Amherst tested levels of 51 PCB congeners in cord blood from 542 babies born between 1993 and 1998. No information on levels of individual congeners is given; however, the mean sum of PCB congeners 118,138,153, and 180 is 0.25 ng/g and the TEF-weighted sum of mono-ortho PCB congeners 105, 118, 156, 167, and 189 is 6.75 pg/g lipid. (Sagiv 2008)	542	New Bedford, Massachusetts	>4
PCN	Polychlorinated naphthalene (PCN)	EWG tested cord blood from10 newborns for 70 polychlorinated naphthalenes and found at least one in all 10 subjects. In all, 31 to 50 different congeners were found with total mean concentration of 0.574 ng/g lipids in blood serum. (EWG 2005)	10	U.S. hospitals	31-50
PCN	Polychlorinated naphthalene (PCN)	EWG tested cord blood from10 newborns of minority background for 70 polychlorinated naphthalenes and found at least one in all 10 subjects. In all, 17 to 24 different congeners were found, with total mean concentration of 0.637 ng/g lipids in blood serum. (EWG 2009)	10	Mich. Fla. Wis. Mass. Calif.	17-24
Pesticide	Carbamate	Researchers from Columbia University, the CDC, and the Southwest Research Institute measured the levels of 29 pesticides in cord plasma from 211 babies born into an urban community in New York City between Sept.1998 and May 2001. 48% of the babies had exposure to 2-Isopropoxyphenol, 45% to carbofuran, and 36% to bendiocarb. All of the babies were exposed to at least one carbamate. (Whyatt 2003)	211	New York City, N.Y.	5
Pesticide	Fungicide	Researchers from Columbia University, the CDC, and the Southwest Research Institute measured the levels of 29 pesticides in cord plasma from 211 babies born into an urban community in New York City between Sept. 1998 and May 2001. 83% of the babies had exposure to dicloran, 70% to phthalimide. All of the babies had exposure to at least one fungicide. (Whyatt 2003)	211	New York City, N.Y.	4
Pesticide	Herbicide	Researchers from Columbia University, the CDC, and the Southwest Research Institute measured the levels of 29 pesticides in cord plasma from 211 babies born into an urban community in New York City between Sept. 1998 and May 2001. 38% had exposure to chlorthal-dimethyl and 20% had exposure to Alachor. All had exposure to at least one herbicide. (Whyatt 2003)	211	New York City, N.Y.	5
Pesticide	Imide	Researchers from Columbia University, the CDC, and the Southwest Research Institute measured the levels of 29 pesticides in cord plasma from 211 babies born into an urban community in New York City between Sept. 1998 and May 2001. 83% had exposure to dicloran and 70% had exposure to phthalimide. All had exposure to at least one	211	New York City, N.Y.	1

Chemical class	Chemical subclass	Summary of representative study	No. of newborns tested	Place of birth	No. of Chemicals found
		fungicide. (Whyatt 2003)			
Pesticide	Mosquito Repellent	Researchers from Columbia University, the CDC, and the Southwest Research Institute measured the levels of 29 pesticides in cord plasma from 211 babies born into an urban community in New York City between September 1998 and May 2001. 33% of the babies had exposure to diethyltoluamide. (Whyatt 2003)	211	New York City, N.Y.	1
Pesticide	Organochlorine Pesticide (OC)	Researchers from Harvard, Emory, and the University of Massachusetts at Amherst tested levels of 2 organochlorine pesticides in cord blood from 542 babies born between 1993 and 1998. Mean DDE levels were 0.48 ng/g serum. Levels of HCB were not given. (Sagiv 2008)	542	U.S. hospitals	1
Pesticide	Organochlorine Pesticide (OC)	EWG tested cord blood from10 newborns for 28 organochlorine pesticides and found at least one in all 10 subjects. In all, 21 different pesticides were found. (EWG 2005)	10	U.S. hospitals	21
Pesticide	Organophosphate Pesticides and Metabolites	Researchers from Columbia University, the CDC, and the Southwest Research Institute measured the levels of 29 pesticides in cord plasma from 211 babies born into an urban community in New York City between Sept. 1998 and May 2001. 71% had exposure to chlorpyrifos (mean 4.7 pg/g) and 49% had exposure to diazinon (mean 1.2 pg/g), the two most commonly detected pesticides. All other pesticides were found in 4% or less of the samples and all babies had exposure to at least one of the organophosphates. (Whyatt 2003)	211	New York City, N.Y.	8
Pesticide	Pyrethroid	Researchers from Columbia University, the CDC, and the Southwest Research Institute measured the levels of 29 pesticides in cord plasma from 211 babies born into an urban community in New York City between Sept 1998 and May 2001. 7% had exposure to trans-permethrin and 13% had exposure to cis-permethrin. (Whyatt 2003)	211	New York City, N.Y.	2
PFC	Perfluorochemical (PFC)	Researchers from CDC, Columbia University, and Johns Hopkins tested cord blood from 299 babies born at Johns Hopkins Hospital between Nov. 26, 2004 and March 16, 2005 for 10 PFCs. They detected PFOS in 99% and PFOA in 100% of samples. Eight other PFCs were detected at lesser frequency. (Apelberg 2007)	299	Baltimore, Md.	9
PFC	Perfluorochemical (PFC)	EWG tested cord blood from 10 newborns for 12 perfluorochemicals and found at least one of these chemicals in 10 out of 10 participants. Among all 10 participants who tested positive for the chemicals, 9 of 12	10	U.S. hospitals	9

Chemical class	Chemical subclass	Summary of representative study	No. of newborns tested	Place of birth	No. of Chemicals found
		different chemicals were found with total mean concentration of 5.86 ng/g in whole blood. (EWG 2005)			
PFC	Perfluorochemical (PFC)	EWG tested cord blood from10 newborns of minority background for 13 perfluorochemicals and found at least one of these chemicals in 10 out of 10 participants. Among all 10 participants who tested positive for the chemicals, 6 of 13 different chemicals were found with total mean concentration of 2.38 ng/g in whole blood. (EWG 2009)	10	Mich. Fla. Wis. Mass. Calif.	6
Plastic	Bisphenol A & BADGE	Researchers from the Environmental Working Group measured BPA levels in cord blood from 10 newborns of minority background. BPA was found in 9 of 10 samples with a mean level of 2.18 ng/L. (EWG 2009)	10	Mich. Fla. Wis. Mass. Calif.	1
Rocket fuel	Perchlorate	Researchers from the Environmental Working Group measured perchlorate levels in cord blood from 10 newborns of minority background. Perchlorate was found in 9 of 10 samples with a mean level of 0.209 ug/L. (EWG 2009)	10	Mich. Fla. Wis. Mass. Calif.	1

ATTACHMENT B: U.S. Spending on Testing Soil, Water & Air vs. Human Biomonitoring

Agency/Program	**Program Description**	**Annual Budget/ Applicable Year**
Centers for Disease Control and Prevention (CDC) – National Health and Nutrition Examination Survey (NHANES)	The NHANES program is designed to assess the health and nutritional status of adults and children in the United States. The program includes biomonitoring participants ages 6 and above for environmental contaminants.	$13.6M/ 2009 $13.3M/ 2008 (McClure 2009)
US Fish & Wildlife Service - Environmental Contaminant Program	The Environmental Contaminant Program involves monitoring the nation's fish and wildlife for contaminants. The program's research includes, for example, monitoring Artic and American Peregrine Falcons in Alaska and organochlorine residues in Alaskan peregrines.	$13.2M/ 2009 $11.98 M/ 2008 (USFWS 2009)
U.S. Geological Survey (USGS) - Fisheries and Aquatic Resources Program	The Fisheries and Aquatic Resources Program involves testing and monitoring aquatic species for various contaminants. Research includes testing the large mouth bass for persistent organic contaminants, and assessing bioaccumulation of mercury in fish and bioaccumulation of PCBs in Atlantic Charr.	$22.5M/ 2008 (USGS 2009)
U.S. Environmental Protection Agency (EPA) - Superfund	The Superfund remediation program involves the clean up and long-term monitoring of Superfund sites, including testing of soil and water.	$591M/ 2008: Total remedial budget (U.S. EPA 2009a) ~$300M/ 2008: EPA estimated budget for soil and water testing (EPA 2009b)
U.S. Environmental Protection Agency (EPA) - Healthier Outdoor Air Program	The Healthier Outdoor Air Program is designed to provide healthier outdoor air for all Americans. The program includes EPA testing outdoor air for chemical contaminants.	$587M/ 2008: Total program budget (EPA 2009) ~$235M- $294M: EPA estimated budget for air testing and monitoring (EPA 2009c)

ATTACHMENT C: Public Health Costs of Chemical Exposures

Disease	Cost or burden associated with chemical exposures	Finding
Childhood Diseases	$55 billion	An authoritative 2002 study attributed all lead poisoning cases, 30 percent of asthma cases, 10 percent of neurobehavioral disorders and 5 percent of pediatric cancers to chemical pollution. The study, led by pediatrician Philip J. Landrigan, director of the Children's Environmental Health Center at Mount Sinai School of Medicine, estimated the annual costs of this toxic disease burden at $55 billion, nearly 3 percent of U.S. health care costs at the time (Landrigan 2002).
Neurodevelopmental Disease	Up to $83.5 billion	The annual cost of neurodevelopmental disease is estimated at $81-to-167 billion per year. As much as half may be due to exposure to toxic chemicals, according to a 2001 study led by economist Tom Muir of Environment Canada (Muir 2001).
Mercury-linked IQ Loss	$8.7 billion	Low-dose exposure to mercury and other neurotoxic chemical pollution can cause severe and sometimes lifelong neurobehavioral and cognitive problems, according to the National Institutes for Environmental Health Studies (Mendola 2002). A 2005 study by Mount Sinai researchers estimated the costs of this loss of intelligence and productivity from childhood mercury poisoning at $8.7 billion a year (Trasande 2005). Mercury is just one of 201 chemicals known to be neurotoxic in humans (Grandjean 2006).
Chronic Childhood Disease	Up to 80-90%	Mount Sinai's Landrigan estimates that genetics account for only 10-20 percent of cases of chronic disease in childhood in the U.S. and other industrialized nations (Landrigan 2001). This includes: birth defects, the leading cause of infant death; developmental disorders such as attention deficit hyperactivity disorder and autism; asthma, which more than doubled in incidence from 1980 to 1996, according to the Centers for Disease Control and Prevention (Moorman 2007); and childhood leukemia and brain cancer, on the rise since the 1970s (Gurney 1996; Linabery 2008). Landrigan's team and other specialists say that many diseases, from respiratory illness to immune, thyroid and neuropsychological deficits, are likely linked to environmental toxins (Etzel 2004; Sly 2008; Wigle 2008).
Developmental Problems	28 percent	An expert committee of the National Academy of Sciences concluded in 2000 that a combination of environmental and genetic factors cause 25 percent of American children's developmental problems, including low birth weight, neurobehavioral deficits and pre- and post-natal death. The report estimated that another 3 percent are caused by toxic environmental exposures alone (NRC 2000).
Children on Medication	26 percent of all children (irrespective of link to chemical exposures)	In 2007, 26 percent of Americans age 19 and under took prescription drugs for chronic health problems, according to a major pharmaceutical benefit provider. The most commonly dispensed medications were treatments for asthma and allergy, followed by attention deficit/hyperactivity disorder (ADHD) and depression (Medco 2008). No one knows for sure how much chemical exposures contribute to this disease burden, but a wide range of compounds have been linked to the most common children's health problems, including 82 types of chemicals or pollution linked to asthma (Janssen 2009).
Lifetime Disability		Chemical injury to developing organs in a young child or an infant can cause lifelong disability (NRC 1993, U.S. EPA 1998). Numerous studies have linked early exposure to chemical pollutants to later health problems, including: asthma and respiratory disorders; thyroid deficits; cardiovascular disease; learning disabilities, intellectual delay, loss of IQ points and corresponding loss of earning potential; and neurodegenerative conditions such as Parkinson's disease (Boyd 2008; Etzel 2004; Landrigan 2002; Muir 2001; Weiss 2000).
Indirect Costs		The U.S. EPA and the European Organization for Economic Cooperation and Development (OECD) say the true costs of chronic childhood illnesses include: parents' earnings forgone to care for child; value of missed school days; child's foregone earnings; effects of reduced educational attainment on child's future earnings; reduced labor force associated with developmental disabilities. (OECD 2006, U.S. EPA 2002).
Human Diseases Linked to Exposures	182 diseases	Based on a comprehensive review of scientific literature, researchers at the University of California, San Francisco and Boston Medical Center documented 182 human diseases and health problems, including birth defects, asthma, and childhood cancers, associated with chemical exposures (Janssen 2008).
		At the 2004 international summit on chemicals and health at the United Nations

"Serious Threat to Children"		Educational, Scientific and Cultural Organization (UNESCO) in Paris, 154 prominent scientists, physicians and other experts from the U.S. and 18 other nations signed a statement asserting that chemical exposures are a "serious threat to children" (PA 2005).

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ENVIRONMENTAL WORKING GROUP

Kenneth A. Cook

ONE collection day

TEN blood samples

413 chemicals

2 8 7

toxic chemicals

detected

Video

10 more Americans

358

Selected U.S. Studies of Industrial Chemicals in Umbilical Cord Blood

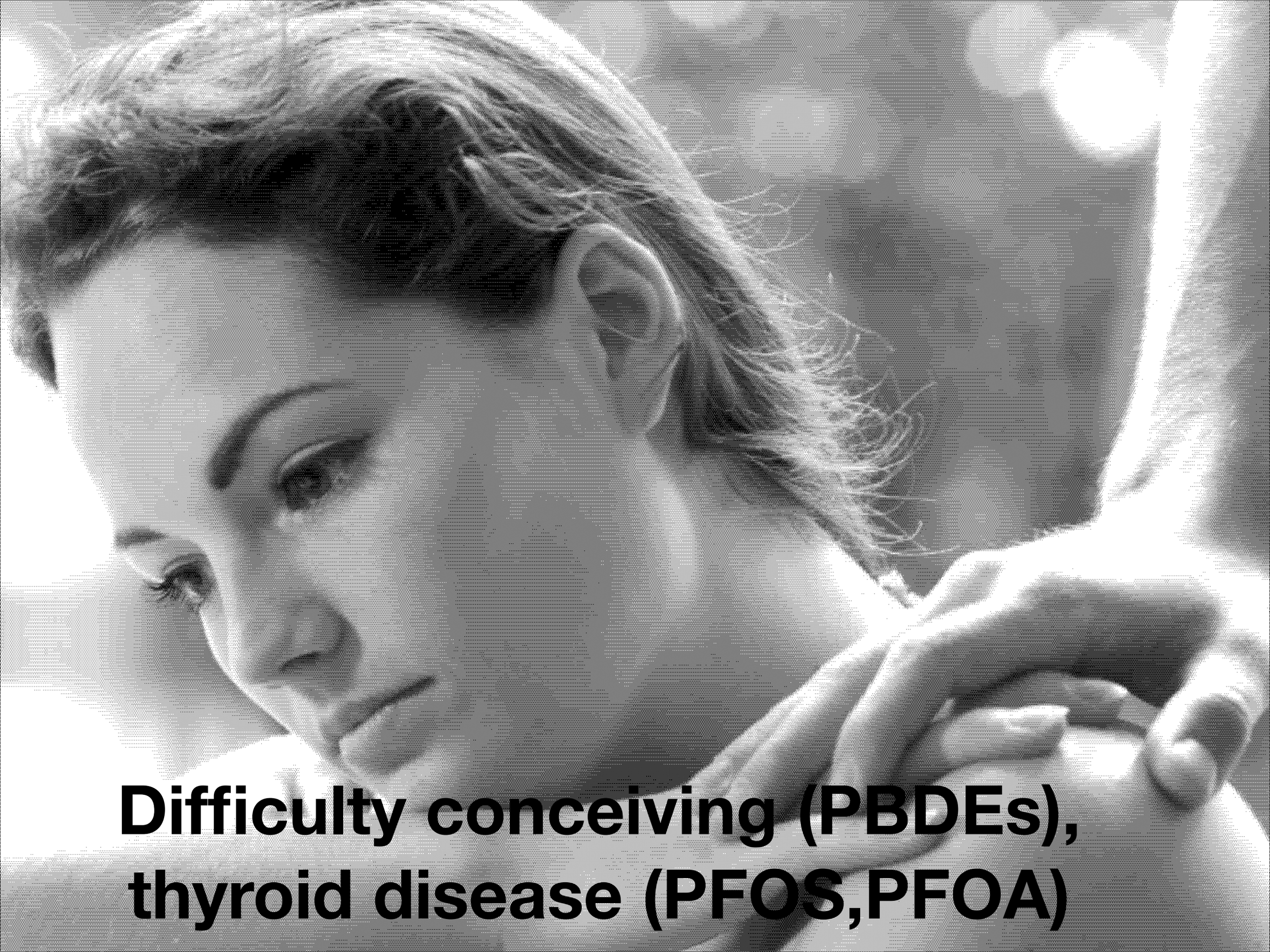
Low Doses Matter

Cialis (tadalafil) tablets — 30 ppb

NUVARING (etonogestrel/ethinyl estradiol vaginal ring) — 0.035 ppb

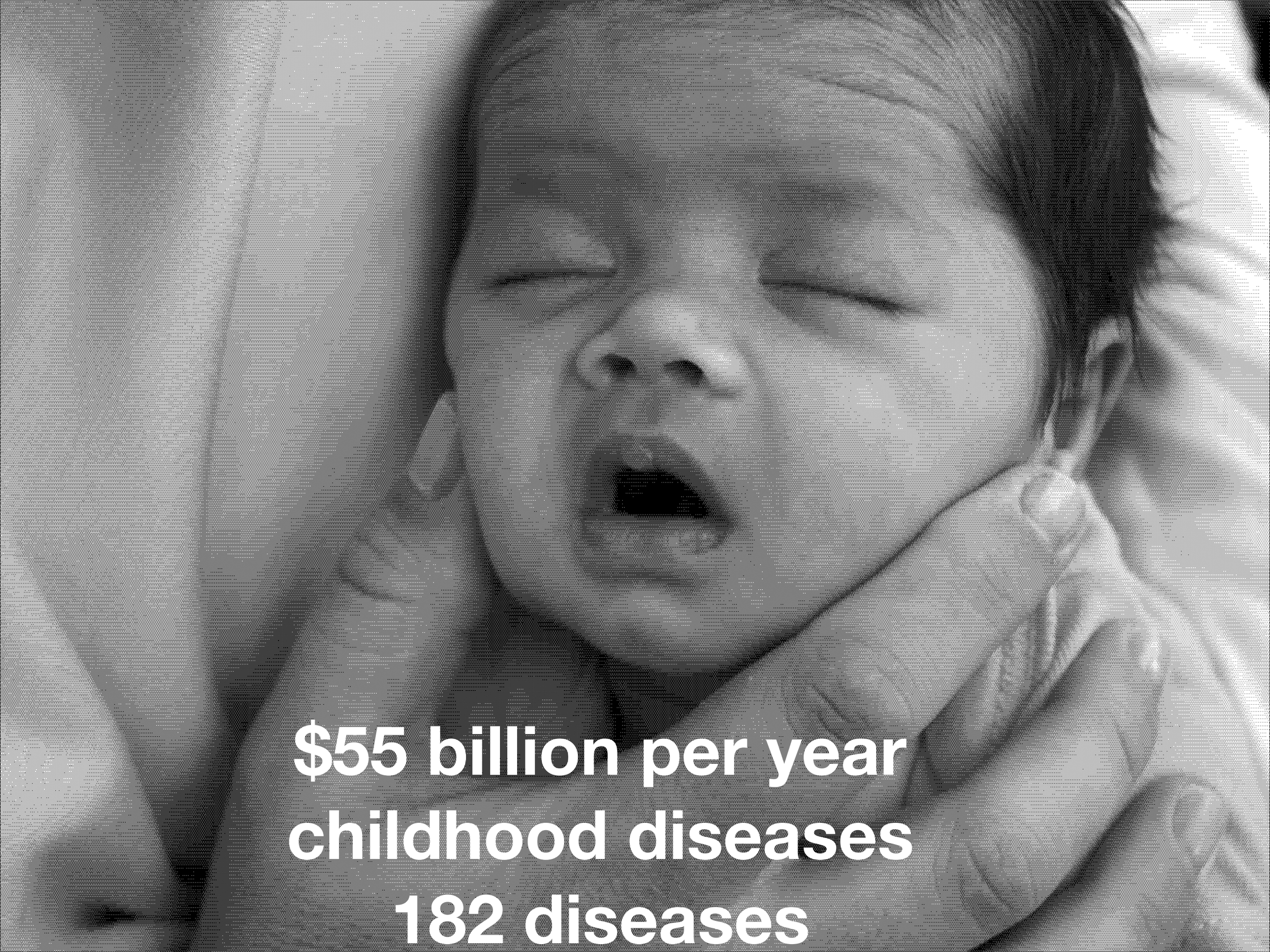
PAXIL PAROXETINE HCl — 30 ppb

Birth weight and head circumference (PFOS, PFOA)



Difficulty conceiving (PBDEs), thyroid disease (PFOS,PFOA)

Heart disease (BPA)


$55 billion per year
childhood diseases
182 diseases

$300 million
$0 million

200 people 493 chemicals 15,000 chemicals

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09 APR 20 AM 6:03

April 6, 2009

Document Control Officer 8(e) Coordinator
U. S. Environmental Protection Agency – East
Confidential Business Information Center
Mail Code: 7407M
1200 Pennsylvania Avenue, N.W.
Washington, DC 20460

Dear Sir:

In accordance with TSCA 8(e) requirements, [redacted] is submitting [redacted].

The purpose of the study was to determine the acute inhalation toxicity of the test article [redacted]

The information submitted in this study is considered "Confidential Business Information". A sanitized, as well as a confidential version, is being submitted.

Please contact me if you have any questions.

Sincerely,

Video

January 30, 2010

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Re. Hearing: Current Science on Public Exposures to Toxic Chemicals
Scope: Examination of recent science analyzing public exposures to toxic chemicals

Senate Committee: Environment and Public Works
Subcommittee on Superfund, Toxics and Environmental Health

Chairwoman Boxer, Chairman Lautenberg, Ranking Member Inhofe, esteemed Members of the Committee, and guests:

Thank you for the opportunity and honor of presenting testimony to this subcommittee on the "current science on public exposures to toxic chemicals." I trust that this hearing on health exposures and biomonitoring will be useful, and an important component to the process of improving public health through the intended reform of the Toxic Substances Control Act of 1976 (TCSA).

Biomonitoring is a tool with clear benefits. The ability to actually measure the amount of any given chemical in the body is an important step beyond – or test of – modeling assumptions. The ability to identify lower and lower concentrations of an increasing number of substances has allowed us to recognize potential problems much earlier than in the past and has provided the impetus to act before harm occurs. The use of biomonitoring for research to investigate potential new interactions on multiple fronts is an important new area for investigation. Many of the witnesses before this committee have discussed these points in the past. I want to focus my remarks on the impact of biomonitoring on medical care and public perceptions, particularly in the area of risk communication. I leave my written comments to be read into the record, along with associated references. I am happy to respond to any questions from the committee.

Personal Background:

I am a physician, trained and board-certified in Internal Medicine, Emergency Medicine, Medical Toxicology, with additional experience in Pathology, Occupational Health, and laboratory interpretation. I have been an attending physician in Connecticut for 22 years. I am the Medical Director of Occupational Health Services for Hartford Hospital and the Connecticut Children's Medical Center. I am the Associate Medical Director of the Connecticut Poison Control Center (CPCC), one of about 60 regional poison centers certified by the American Association of Poison Control Centers. The CPCC receives more than 30,000 calls every year from the public and medical personnel regarding possible or known toxic exposures. I am an Associate Professor at the University of Connecticut School of Medicine, and the Director of the Medical Toxicology training program at UConn, one of about 24 such programs in the country. In that role and as an educator, I am responsible for training some of the next generation of medical providers. I am a consultant to the Connecticut Department of Public Health and was a member of the Environmental Health Public Tracking Program Planning Committee. I participate in our state's biopreparedness activities. I also serve as a reviewer for 6 peer-reviewed medical journals, and am a member of the Editorial Board of the Journal of Medical Toxicology. I am a member of the Scientific Advisory Council of the Environmental Health Research Foundation, at whose invitation I agreed to testify today. I am a member of the Board of Directors of the American College of Medical Toxicology (ACMT), which is the member organization representing most of the 500 board-certified Medical Toxicologists in the country. In that capacity, I serve on the Practice Committee and am the National Director of a network between ACMT and the Agency for Toxic Substances and Disease Registry (ATSDR) of the Centers for Disease Control and Prevention (CDC). The purpose of this network is to provide the regional expertise of physician medical toxicologists to the regional ATSDR representatives and their public health partners in order to address concerns about human exposure to chemicals in the environment (either naturally-occurring or arising from human activity).

My comments are my own, and do not necessarily reflect opinions of the ACMT, its Board of Directors, or its members. I have attached for the written record an editorial published in our on-line journal (Appendix A), and a position statement of the College (Appendix B) relevant to some of the issues discussed today.

The mission of the American College of Medical Toxicology is to advance quality care of poisoned patients and public health through physicians who specialize in consultative, emergency, environmental, forensic, and occupational toxicology. Previous contracts and cooperative agreements with ATSDR have allowed ACMT to present material on chemicals as potential terrorist weapons (Toxic Industrial Chemicals and Toxic Industrial Materials) to more than 6000 public health, prehospital and medical personnel, emergency planners, and military personnel; and material on the health effects of clandestine methamphetamine laboratories to more than 1100, as well as recurring conferences at regional and national meetings.

Potential Benefits of Biomonitoring:

Medical Toxicology is a medical subspecialty focusing on the diagnosis, management and prevention of poisoning and other adverse human health effects due to medications, occupational and environmental toxins, and biological agents.

Biomonitoring is an important tool for use in toxicology. In the current setting of unwarranted or uncertain fear about "all things chemical", it can also be used to focus or alleviate concerns. Specifically, a robust biomonitoring program can be used to a greater or lesser extent to:

- Identify the concentration of chemicals actually taken up by the human body and the metabolic fate of those chemicals;
- Improve the accuracy or test the validity of assumptions in physiologically-based pharmacokinetic modeling or regulatory models;
- Identify susceptible populations or particular at-risk groups (e.g. genetic polymorphisms) for chemical toxicity;
- Track trends of exposure over time and in the setting of various interventions;
- Validate reference ranges for chemical exposure;
- Inform discussions regarding levels of exposure consistent with no adverse effects (thresholds);
- Provide a framework in which to evaluate individuals' concerns about chemical exposure.

Need for Support of Currently Existing Mechanisms to Conduct Biomonitoring:

While the viewpoints and worldview of the multiple participants in the 2006 National Research Council's (NRC) report on "Human Biomonitoring for Environmental Chemicals" (http://www.nap.edu/catalog.php?record_id=11700) may differ, their recommendations identify not only potential benefits and research utility, but also the shortcomings and the practical difficulties of using biomonitoring to answer questions about environmental exposures and human health.

These difficulties are to be expected, given the different dosing scenarios, genetic polymorphisms, and impact of other diseases and confounders on an individual's or population's response to any single or mixture of substances. As reform of TSCA is considered, please bear in mind the recommendations of this group, as well as the need for funding to reach the goals espoused by this committee. The NRC's recommendations include the need for:

- Coordinated strategy for population biomonitoring based on potential for exposure and public-health concerns;
- Development of biomonitoring-based hazard and exposure assessments and public-health; surveillance to interpret the risks posed by low-level exposure to environmental chemicals, enhancing where possible existing efforts by adding biomonitoring in order to improve interpretation;
- Focus on strategies for reporting results of biomonitoring studies;
- Review of bioethical issues inherent to biomonitoring efforts;

These are in fact the ultimate goal of such efforts as the recurring National Health and Nutrition Evaluation Survey (NHANES) and the goal of the Environmental Public Health Tracking

programs at the state and regional levels. Unfortunately, funding for state-based biomonitoring efforts, building on years of public health activities and medical concerns at the state, regional, and national levels, has been cut drastically, resulting – for example – in a 67% decrease in allocated funding this year and a reduction from a possible 33 states to only 3 states funded. The National Association of Public Health Laboratories (APHL) has issued a document identifying the priority needs of the state laboratories and emphasizing the need for coordinated funding of existing infrastructure to improve and regionalize what is now a fragmented system (http://www.aphl.org/policy/priorities/Documents/HillDayFactSheets2009.pdf). Utilizing improved capabilities and capacity developed through biopreparedness efforts over the last 8 years, it is very possible to utilize the expertise and resources of state-based public health laboratories for biomonitoring projects of public health importance. I was able to attend the National Biomonitoring Planning Conference held by the APHL in Atlanta last fall (http://www.aphl.org/aphlprograms/eh/Documents/NBMSummary2009.pdf). This meeting of state and federal laboratorians generated the framework for a 5 year plan to generate a data- and expertise-sharing biomonitoring program. However, this can only occur through funding and education of qualified personnel to make use of purchased equipment.

Limitations of Biomonitoring:

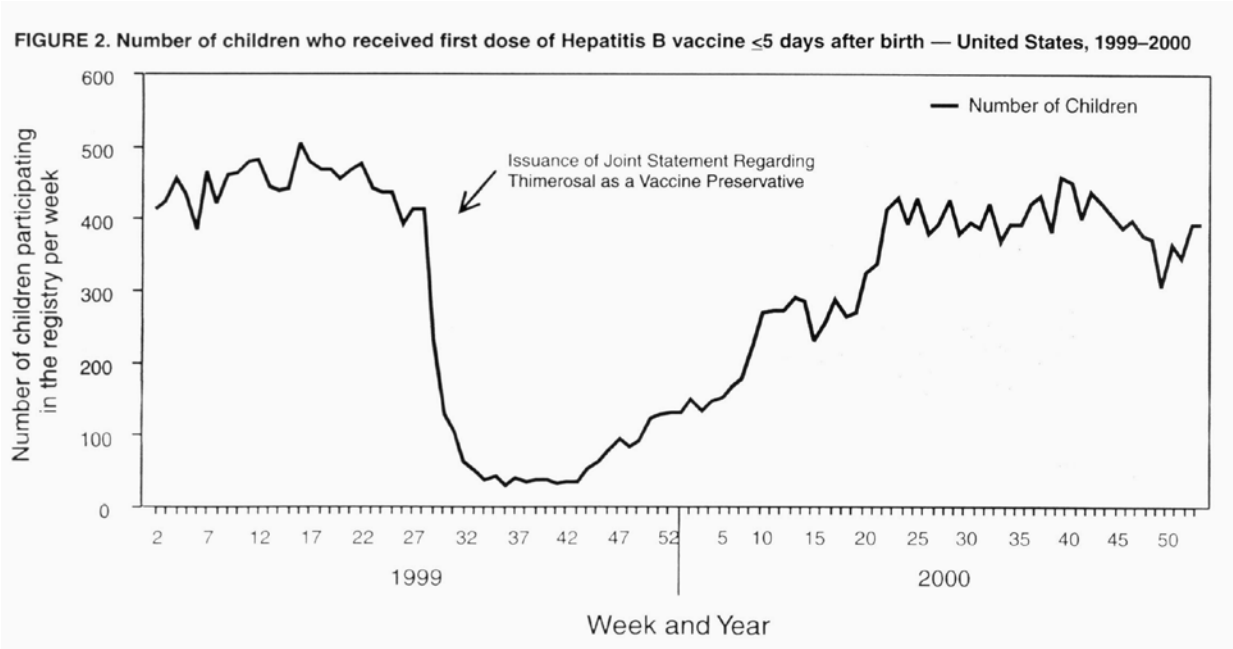
It is important to recognize the limitations of biomonitoring. Biomonitoring is a tool. It is not an answer. It does not, in and of itself, eliminate potential confounders or alternative explanations for identified associations between chemical exposure and disease. As perhaps needs reiterating, the identification of a substance confirms its presence; it does not indicate whether that substance is causing harm or benefit. Any environmental chemical will be present to some extent in those who ingest, inhale or otherwise are exposed to it. Thus, the statements that have been made in this committee and other venues that "neurotoxins", "endocrine disruptors", or other "harmful chemicals" are present in our (and our childrens') bodies is meaningless, without specific relationship to dose, exposure timing, and comparison to appropriate control populations. While it is frequently stated that "scientists have developed a more refined understanding of how some chemicals can cause and contribute to serious illness", it is also true that our ability to measure substances at very low concentrations has outstripped our ability to determine causation. In other words, scientists are able to identify spurious associations with environmental chemicals, while having difficulty accounting for confounders, thus proffering disease causations that do not, in fact, exist.

The Precautionary Principle (United Nations, 1992)

Where there are threats of serious or irreversible environmental damage, lack of full scientific certainty shall not be used as a reason for postponing cost-effective measures to prevent degradation.

Unfortunately, biomonitoring can be – and has been – abused as a tool. The practical problem with overstating exposure-disease associations is seen every day by medical professionals who evaluate people who are fearful of being "poisoned" by the latest chemical touted in a study as the cause of the same disease blamed on another compound the month before. Unfortunately, there are also a number of practitioners who prey on such patients, offering therapies that are not

indicated for conditions the patient does not have. On a weekly, if not more frequent basis, I am contacted by patients or media desiring assistance in interpretation or personal application of data reported in the scientific literature or obtained from ill-considered or inappropriately-performed laboratory testing. This does not just affect the small portion of the population with fixed delusions. It potentially impacts every woman considering pregnancy, every parent wondering about their children's health, and every worker and employer. The incessant drumbeat that environmental chemicals are the source of all ills is hyperbole that should fall in the face of the evidence supported by biomonitoring.



When the message is not communicated clearly or correctly, we end up with inappropriate response and harm, rather than the prevention of harm. This is demonstrated in the drop in vaccinations (figure above) and neonatal deaths from Hepatitis B secondary to unfounded concerns about thimerosal-preserved multi-dose vials of vaccine.

Similarly, increases in unvaccinated measles cases and persistently lower rates of vaccination are attributed to the unethical and dishonest study published in The Lancet by Wakefield et al., based on 12 patients.

Practical Risk Communication Issues Concerning Exposure to Chemicals in the Environment:

How do I as a practicing toxicologist provide a scientific, understandable, and appropriate message to my patients and other concerned parties, both professionals and lay public? I have used the following criteria in my evaluation of the literature and communication with others. I would respectfully suggest that these be considered when communicating biomonitoring data to

Americans, whether at the patient-physician, scientist-peer-review literature, policy or regulatory levels.

- Identifying a substance as being of public health concern is not the same as stating it is causing individual harm. Appropriately obtained or extrapolated biomonitoring data can be used to gauge an individual's exposure compared to population norms.

- Decisions about exposure need to incorporate information about at-risk populations (and whether an individual is a member of such a group), as well as the benefits gained by use of the product or availability and potential adverse effects associated with alternatives. Biomonitoring data alone does not answer this question, but common sense should play an important role.

- Claims of association of a medical condition with historic exposures to some substance need to be evaluated in the face of current exposures. Biomonitoring data that identifies decreasing – or increasing – population exposure to chemical compounds should be incorporated into all research publications touting disease associations and should be required by editors prior to acceptance for publication.

- Using a study population to data dredge for associations is reasonable for hypothesis generation. A statistical association generated post-hoc from multiple comparisons is shaky ground from which to draw conclusions, particularly when the conclusions fly in the face of existing information or known facts, or do not take into account reasonable confounders.

- It is intellectually dishonest to claim that effects of chemical exposure are so small as to be clinically unrecognizable, then attribute major clinical effects to these same exposures.

If we consider everything a risk, then we can't avoid true dangers.

My point is that spurious associations and contradictory positions on regulation of chemicals in our environment are not going to be resolved solely by the use of biomonitoring. However, appropriate focus on those substances or exposures of most concern can be greatly influenced by the results of carefully considered, appropriately conducted and correctly interpreted biomonitoring studies.

As a practicing physician toxicologist, it is my responsibility to interpret the basic science, animal and human exposure data for people who are concerned about their risk, and to educate physicians and others who provide care for patients or information for people. Those who co-opt the biomonitoring process for their own advancement and political aims do a disservice to the entire medical and lay community with generalizations about "chemicals", "cancer", "neurotoxins", "endocrine disruptors", and other terms that are used without specific and detailed reference to dose, effect, and risk/benefit considerations, applied to both the products in use and their alternatives.

Biomonitoring is a very useful tool for documenting human exposure to environmental chemicals of concern, tracking trends in exposure, and prioritizing chemicals of most concern for possible regulation, restriction or substitution, consistent with "green chemistry" principles. Chemicals with declining prevalence or concentration in the population, as demonstrated by biomonitoring, should be treated as the historical success or cautionary stories they provide in terms of public health improvement or lack thereof. Attention and funding should be focused on those compounds that display biopersistence, bioaccumulation, biotransformation, or that generate sentinel signals from high-dose exposure (e.g. occupational) or high-risk populations (e.g. fetal/neonate); and for which concern for significant public health effects exist.

I thank the committee for this opportunity to present the views of a practicing medical toxicologist and educator on the important issues of biomonitoring, public health, and risk communication.

Sincerely,

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References:

American College of Medical Toxicology Position Statement: Post-Chelator Challenge Urinary Metal Testing. http://www.acmt.net/cgi/page.cgi?aid=2999&_id=52&zine=show (last accessed 1/30/10)

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CDC Funding 2009 Biomonitoring grants http://www.cdc.gov/biomonitoring/state_grants.htm

CDC. Fire Deaths and Injuries: Fact Sheet. http://www.cdc.gov/HomeandRecreationalSafety/Fire-Prevention/fires-factsheet.html

CDC National Exposure Report: (chemicals in 4th report): http://www.cdc.gov/exposurereport/pdf/NER_Chemical_List.pdf [212 chemicals, including 75 being checked (retrospectively – 2003-4; 1999-2000, 2001-2) for the fist time]

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APPENDIX A: Internet Journal of Medical Toxicology Editorial

A Call To Arms For Medical Toxicologists: The Dose, Not The Detection, Makes The Poison
Internet Journal of Medical Toxicology 2003; 6(1):1 (archived)
http://www.acmt.net/cgi/page.cgi?aid=1543&_id=52&zine=show

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Introduction

Over the last several decades, the analytic capability to measure very small concentrations of an increasingly vast array of chemical structures has increased dramatically. Analytic chemists can now measure certain purported toxicants at a fraction of a part per trillion.[1] To give some idea of this level of detection, the proverbial "drop in a bucket" would be measuring things at the parts per million range; parts per trillion is equivalent to a "drop in a lake"!

Unfortunately, our ability to determine what to do with this data has not progressed as fast as the analytic technology. Although a tenet of toxicology is that "the dose makes the poison", many people inappropriately fear that the very detection of a substance must equate with toxicity. As medical toxicologists, we focus on the patient's symptoms and signs and their association with exposure and delivered dose. However, many of us are faced with patients coming from other practitioners with laboratory data from a multi-element panel indicating toxicity by mercury, arsenic, or other heavy metals or excesses or deficiencies of a wide array of trace elements or hydrocarbons (so-called environmental pollutants). These laboratory tests are often presented as *de facto* evidence of toxicity or "systemic imbalance or insufficiency" without any evidence of excessive dose or exposure. Furthermore these test results are then considered the cause of a variety of poorly characterized or general symptoms. Unfortunately, "environmental ecologists" and other practitioners[2] often use these test results, which we consider clinically irrelevant, as support for a variety of scientifically unproven or clinically non-indicated treatments.

We define esoteric testing to be uncommonly performed laboratory analyses for trace elements, environmental contaminants, or endogenous enzymes obtained from samples of blood, urine, hair or other body tissue. These tests or matrices generally lack a published reporting of validated reference ranges or suffer from significant procedural difficulties. While a large number of potentially valid analytes or methods may fall into this broad definition, the widespread use of certain testing panels and laboratories by certain groups of practitioners present obvious examples of aberrant practices with which we are all familiar. (the so-called "know it when you see it" definition of quackery).

We present the following composite case and a rationale for a proposed set of criteria to assist physicians in the decision to perform esoteric testing and in the interpretation and application of results already obtained.

Case Example

A 52 year old woman presents to the toxicology clinic complaining of generalized fatigue, difficulty with memory, and anxiety. There is a history of some weight loss over the last few months and difficulty sleeping. The patient is an ex-smoker and consumes occasional ethanol. A general physical exam is unremarkable, as is a neurological and mini-mental status exam. During

further questioning, as the toxicologist formulates a wide differential (including a number of non-toxicologic diagnoses), the patient declares, "My other doctors found I was out of balance and have too much mercury in my system. I want to know if I should have my dental fillings removed because I don't feel much better after chelation." With further discussion, it becomes clear that the patient has been to a number of practitioners, some of whom have used "alternative practices" such as kinesiology to determine she has an excess of heavy metal contamination, while others have given courses of dimercaptopropane sulphonate (DMPS) followed by urinary mercury collection and hair mercury analysis.

Discussion

While poisoning by a wide variety of naturally-occurring heavy metals or industrial contaminants is well-described, the "low-level" toxicity of mercury, arsenic, and other heavy metals is more problematic. Even for elements, such as mercury, where it is generally accepted that hair analysis is a valid analytic technique[3], proper collection, analysis and interpretation is still necessary. Furthermore, the distinction between public health concerns and individual toxicity is very important. For example, it is generally accepted that mercury contamination of the environment has contributed to an increase in the mercury concentration in marine animals. All states have health advisories regarding the consumption of fresh-water fish because of concerns about mercury (and PCB) contamination. Yet these advisories are focused on the possible risk for neurotoxicity for the unborn child of a pregnant woman. While various studies have raised questions about subtle population neurodevelopmental effects from amounts of mercury 10-100 times that of the average American diet (resulting in maternal hair mercury measurements far above what is commonly reported as abnormal by hair analysis laboratories), even these authors state that none of their subjects demonstrated clinical mercury poisoning.[4] Can we reassure the vast majority of patients with vague symptoms and abnormal heavy metal screens without glossing over the patient who is truly poisoned? We believe such a balance is possible and should be one component of the medical toxicologists' practice. On an individual basis, we can educate practitioners and the general populace in our area regarding some of the cautions to take with available laboratory testing. Each of the following points deserves careful consideration:

1) The decision to perform laboratory testing should be based on a differential diagnosis, rather than indiscriminate testing.

It is often tempting to run a large battery of tests on patients with poorly characterized or complex presentations. Patients who carry diagnoses such as chronic fatigue syndrome, multiple chemical sensitivity, fibromyalgia are especially prone to this type of testing, since these "conditions" are essentially symptom complexes and have no known organic or toxic etiology. Also, patients with chronic, progressive or incurable disorders such as multiple sclerosis and autism may be tested for toxicants. Some physicians will order trace mineral analyses searching for a cause of these syndromes, but many unscrupulous practitioners order these tests to "prove" to patients the need for chelation or other unnecessary, and potentially dangerous, "treatments". Unfortunately, this reliance on analytic testing is often misplaced. By pure chance, the statistical likelihood of finding a test result outside a population norm will increase as the number of tests increases. In the absence of good clinical correlation, these results are usually meaningless, but can cause a good deal of confusion and concern in both patient and physician.[5] As mentioned above, the dose determines toxicity. In addition, most toxicants produce a characteristic pattern of effects; this

specificity of effect should be carefully sought in the history and physical exam, which then should guide testing patterns.

2) Critical methodological steps regarding specimen collection and laboratory analysis must be heeded.
All of these tests measure very small amounts of chemical compounds. As such, even low-level contamination of collection materials or procedures can result in false positive reports. This problem is well described with lead biomonitoring, where elevated capillary blood measurements from fingerstick testing must be confirmed with a venous sample because skin contamination with lead may result in falsely elevated blood levels. This can also occur with heavy metal testing of hair, due to external contamination by metals found in hair treatments, public water supplies or air pollution.[6] Similar problems arise with blood or urine collections.[7] In addition, dietary restrictions are necessary when analyzing body burden of heavy metals or trace elements to prevent false elevations from such agents as dietary supplements or seafood. As an example, the presence of largely non-toxic arsenobetaine and arsenocholine - "fish arsenic" - from seafood interferes with the assessment of arsenic exposure.[8] Although a further testing refinement (i.e. speciation of arsenic type) can be used for this element if there are concerns about the patient's dietary contribution, few laboratories provide this expensive service. Furthermore, this would not distinguish the contribution of arsenosugars that are present in marine algal products (often present in supplements).[9] Finally, many labs will analyze a urine specimen collected for six hours after a chelation challenge, and then compare this result with a norm based on a non-challenged collection. This result will almost always be higher than the non-challenged test but does not reflect an abnormal body burden of the presumed toxicant.[10,11,12] As an example, normal subjects may excrete several fold more mercury post-chelation than in their own pre-chelation test.[12] The results then are "flagged" as abnormal when in fact the testing has done little more than document a normal response to the chelator.

3) Laboratory tests should have well-validated reference ranges. These are lacking for many esoteric tests.
Population norms are often not standardized or are based on small numbers. In fact, some of these laboratories have developed their own reference ranges that are much lower than widely accepted ranges such as that published for hair mercury by the National Centers for Environmental Health of the CDC. This represents their belief that these toxins are more poisonous than mainstream medical science believes. The end result is many patients' results will be flagged as abnormal. In addition, accuracy is very poor for some analyses, such as hair testing by popular laboratories.[13] Many of these laboratories claim Clinical Laboratory Improvement Amendments (CLIA) certification, a federal standard for certain analytic tests, yet no such certification specifically exists for hair mineral analyses. Proficiency testing standards for hair testing do not exist, and individual labs devise their own verification methods and criteria for accuracy. Analytic laboratories should demonstrate some validity of testing, both internal (precision) and compared to standards (accuracy). Even when this is done,[14] information regarding measurements in a target population, such as those with known clinical effects from excesses or deficiencies of the given analyte, should be included.

4) Exceedance of a reference value does not necessarily imply that a patient is poisoned.

Interpretation of laboratory tests is best done in the clinical setting. Often additional clinical, epidemiological and laboratory data are necessary to establish a scientific basis for linking an elevated lab value with the presence or future risk of an adverse health outcome. In fact, for some elements and enzymes, the biologic or physiologic human health effects are not well characterized. As with the heavy metals, the effects of gross deficiencies (e.g. selenium)[15] or excesses (e.g. manganese)[16] are well described, while the effects of smaller variations from a population norm are less clear. Indeed, the experiences of certain unusual populations, such as two-three fold increases in serum manganese in patients receiving total parenteral nutrition, suggest no clinical adverse effects from these excesses.[17,18] Again, laboratories will often report determinations, usually in hair or red blood cells, compared to an unvalidated population norm, rather than as correlated with health or disease. Laboratories should provide normal ranges based on validated control populations. It is inappropriate for a laboratory to provide treatment recommendations. This is particularly true when the laboratory is associated with industries that distribute or otherwise promote treatments for the purported intoxications or deficiencies they claim to document.

Summary

In general, testing for heavy metals, nutritional elements present at extremely low concentrations, or so-called environmental contaminants, should only be obtained in the following situations and with the indicated precautions:

- A properly performed clinical history and physical exam suggests the lack or excess of these chemicals or minerals/metals.
- Proper patient preparation may include dietary avoidance of food and supplements that contain the substance of interest for several days prior to the sample collection.
- The use of collections after chelation is usually unwarranted.
- If post-chelation collections are used, the range of normals must be adjusted accordingly, and the results must be interpreted with extreme caution.
- Collection should be done through a certified laboratory that is experienced in the collection and handling of these specimens to avoid contamination.
- Analysis should be at a reputable laboratory that provides data on their normative population, including the selection and number of controls, and validation of their analytic procedures.
- The laboratory should not provide treatment recommendations or sell therapy to the patient.

Conclusion

There are many factors to consider before ordering a large array of esoteric laboratory tests and a number of important considerations in the interpretation of these tests. The current popularity of broad trace element or pollutant screening with subsequent "detoxification" treatment, is often inappropriate. At this time, many of these tests are best utilized as research tools, such as the current population evaluations by the National Center for Environmental Health of the Centers for Disease Control and Prevention.[19] Application of these test results to individual patients is fraught with problems. Current concerns about environmental-related illness have been misappropriated by a number of practitioners to vindicate non-indicated treatments. A large portion of our toxicology clinic population is convinced their symptoms are due to poisoning, when neither their symptom complex nor laboratory testing justify such a conclusion. It is our

contention that medical toxicologists should be at the forefront in the discussion regarding the appropriateness of toxicologic testing and its interpretation. In addition, we should be active in protecting patients from the misapplication of these tests.

Addendum

The proceedings of an ATSDR panel on hair analysis have been published recently. The reference is: Harkins DK, Susten AS. Hair Analysis: Exploring the State of the Science. Environ Health Persp 2003;111:576-578.

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APPENDIX B: American College of Medical Toxicology Position Statement

Post-Chelator Challenge Urinary Metal Testing

by Nathan Charlton, MD and Kevin L. Wallace, MD FACMT, posted on 10:35 AM, July 27, 2009
http://www.acmt.net/cgi/page.cgi?aid=2999&_id=52&zine=show

Heavy metals, such as lead and mercury, are ubiquitous in the environment [1-4]. Exposure in human populations is constantly occurring, and detectable levels of lead and mercury are commonly found in blood and urine of individuals who have no clinical signs or symptoms of toxicity and may be considered background or reference values [1-5]. Although urine testing for various metals in an appropriate clinical context, using proper and validated methods, is common and accepted medical practice, the use of post-challenge (a.k.a., post-provocation) urine metal testing, wherein specimens are typically collected within 48 hours of chelation agent administration, is fraught with many misunderstandings, pitfalls and risks. The American College of Medical Toxicology issues this position statement in disapproval of the use of post-challenge urinary metal testing in clinical practice and the use of such test results as an indication for further administration of chelating agents.

In current evidence-based medical practice, urinary testing is commonly used in the biomonitoring of exposure to certain metals such as arsenic and inorganic mercury and the severity of their associated toxicity. It is accepted practice to conduct such testing, e.g., in exposed individuals with clinical evidence of peripheral neuropathy, as long as validated collection and analytical methods are employed prior to, or after, a sufficiently long time interval (e.g., 3-5 days) following administration of a chelating agent, i.e., applied to non-challenge urine specimens, and the results are compared to appropriate reference values [5, 6]. In some non-evidence-based medical practices, however, assessment of metal poisoning is frequently based on non-validated post-challenge urine metal testing, which invites inappropriate comparison to normal urine reference ranges [4-7].

Chelating agents such as dimercaptosuccinic acid (DMSA), dimercaptopropanesulfonic acid (DMPS), dimercaprol (BAL), and edetate calcium disodium (CaNa2-EDTA) bind metallic and metalloid elements and have been shown to increase their elimination from the body. Chelating agents have been found to mobilize metals in healthy individuals who have a body burden considered normal for a standard reference population, as well as in those who are determined to have a high body burden of the same metallic species [4, 8-11]. More specifically, urine specimens collected in relatively close temporal proximity to administration of chelating agents, i.e., post-challenge specimens, are expected to have increased concentrations of metallic elements. This includes elements, such as zinc, that are essential to normal physiologic functions and maintenance of good health.

Normal reference values for non-challenge urine metal test results vary among and within different populations. Ranges for these values have been established in nationally certified laboratories that meet proficiency standards for urinary metal testing [5]. However, scientifically acceptable normal reference values for post-challenge urine metal testing have not been established [10]. In addition, scientific investigation to date has failed to establish a valid correlation between prior metal exposure and post-challenge test values [10]. Despite the lack of

scientific support to do so, it is also a common practice of some laboratories and care providers to provide or apply non-challenge normal reference values as a comparative means of interpreting results of post-challenge urine metal testing [5]. Currently available scientific data do not provide adequate support for the use of post-challenge urine metal testing as an accurate or reliable means of identifying individuals who would derive therapeutic benefit from chelation.

Unfortunately, the practice of post-challenge urine metal testing and its application to assessment of metal poisoning often leads to unwarranted and prolonged oral and/or intravenous administration of chelating agents, in response to the results of serial post-challenge testing that remain elevated above non-challenge reference values. Chelation therapy based on such laboratory values, in addition to being of no benefit to patient outcome, may actually prove harmful [5, 12]; catastrophic outcomes such as acute fatal hypocalcemia have been reported following the improper use of a chelating agent, edetate disodium (Na2-EDTA) [13]. In addition, the safer formulation of this agent, CaNa2-EDTA, has been demonstrated to increase urinary excretion of essential minerals such as iron, copper and zinc [8, 14]. There is published experimental evidence that deleterious effects may occur when chelation is applied in the absence of prior lead exposure. [15] Other chelating agents such as DMSA and DMPS may also increase the elimination of certain essential elements, as well as promote target organ redistribution of metallic elements of concern such as mercury [16-18].

It is, therefore, the position of the American College of Medical Toxicology that post-challenge urinary metal testing has not been scientifically validated, has no demonstrated benefit, and may be harmful when applied in the assessment and treatment of patients in whom there is concern for metal poisoning.

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Testimony of Tracey J. Woodruff, PhD, MPH
U.S. Senate Committee on Environment and Public Works on

"Current Science on Public Exposures to Toxic Chemicals"

Thursday, February 4, 2010
Dirksen Senate Office Building, Washington DC

Senator Frank R. Lautenberg, Chairman
Senator James M. Inhofe, Ranking Member

Good morning, Chairman Lautenberg, Senator Inhofe, committee members and guests. I am Dr. Tracey Woodruff, Associate Professor and Director of the Program on Reproductive Health and the Environment, Department of Obstetrics, Gynecology, and Reproductive Sciences at the University of California, San Francisco. Thank you for the opportunity to testify at this important hearing. The Program on Reproductive Health and the Environment is dedicated to creating a healthier environment for human reproduction and development by advancing scientific inquiry, clinical care and health policies that prevent exposures to harmful chemicals in our environment. Today I shall focus on concerning trends in reproductive health and development, current chemical exposures and policy needs.

Trends. There are numerous concerning trends in the developmental health of the United States population, which have been reported in the scientific literature [1, 2]. These include:

- **More women in the U.S., particularly women under the age 25, the time of peak fertility, are reporting difficulty conceiving and maintaining their pregnancies**. Between 1982-2002, the percent of women reporting that they had difficulty in conceiving and maintaining pregnancy, doubled from 4.3% to 8.3% in a national survey conducted by the National Center for Health Statistics [3, 4].
- **Increasing numbers of babies are born too early – before the 37th week of gestation – putting them at greater risk for death, learning and behavior problems, and developmental delays** [5]. One out of every eight babies is born prematurely, a rate that has increased 36% since the early 1980s [6].
- **Birth weights are declining, even among normal, healthy, full-term infants, putting more infants at risk for short and long-term health complications and chronic disease** [7]**.** A new study reports that U.S. birth weights declined 1.5% between 1990 and 2005, a drop that was not explained by maternal and neonatal risk factors or obstetrics practices. During the same period, the number of infants born small for gestational age increased by nearly 1% [8].
- In my own state of California, gastroschisis, a birth defect where the abdominal wall does not form completely and the intestines protrude outside of the body, has increased by over 300% between 1987 and 2003 [9].

- Increasing rates of childhood diseases, including autism [10], certain childhood cancers [11], and obesity [12].

These are among a number of adverse trends in health outcomes that have been summarized in "The Health Case for Reforming the Toxic Substances Control Act," a new report highlighting the growing concern about chemicals and increases in adverse health effects in the population [13].

We also have growing scientific evidence that environmental contaminants can impact early development, particularly if exposures occur prior to conception, during pregnancy or early in life -- periods of development that are more vulnerable to disruption by environmental chemicals [14]. In particular, disruptions during the prenatal period can increase the risk of effects during the immediate, short and long term. Some examples:

- immediate term: birth defects, pre-term birth, low birth-weight
- short term: learning disabilities and childhood cancers
- long term: diabetes, cardiovascular disease, and cancers as adults.

Chemical Exposures and policy needs: Since World War II, chemical production in the U.S. has increased more than twenty-fold [15]. As of 2006, there are over 80,000 chemical substances registered for use in U.S. commerce, and about 3,000 chemicals manufactured or imported in excess of 1 million pounds each [16]. Environmental contaminants are ubiquitous in our air, water, food and drink, personal care products, pesticides and everyday household items.

Biomonitoring – a growing area of research that measures the types and levels of chemicals in our bodies – now demonstrates irrefutably that these chemicals are contaminating our bodies in addition to our environments. For example, the National Health and Nutrition Examination Survey, an annual nationally based representative survey of the U.S. population, consistently finds measurable amounts of hundreds of environmental contaminants in people's bodies. For example, over 75% of people have triclosan in their body, up to 100% of people have some type of PCB measured in their body, over 98% of people have polyfluoroalkyl chemicals, and over 90% of people have measureable levels of bisphenol A [17]. Many of these exposures come from the everyday use of products in our lives – such as personal care products, cookware and containers – sources that most people consider to be inert.

Such high frequencies of chemical detection mean that, as a population, we are exposed to a multitude of chemicals simultaneously. As a population, we also vary in our biological susceptibility to harm by chemical exposure. This susceptibility can be due to age (prenatal, infant, child, puberty or elderly), health status (pre-existing health conditions such as immune compromise, diabetes, asthma), or socioeconomic stressors.

Therefore, when we consider the risk of adverse health effects from exposure to any ***one*** chemical reported through biomonitoring studies, the National Academy of Sciences recommends that we consider this exposure in the context of the existing chemical exposures and biological susceptibilities of the U.S. population. Given the lack of data on the impacts of

cumulative exposure to chemicals, the National Academy of Sciences also concludes that we should not assume that there is a safe level of exposure to any individual chemical unless proven otherwise [18].

Thyroid hormones and thyroid disrupting chemicals illustrate reasons to be concerned about the pattern of chemical exposure that biomonitoring studies reveal. Thyroid hormones are essential for fetal brain development during pregnancy [19]. Even small reductions in maternal thyroid hormone levels are associated with neurological deficits in the children [20, 21]. In addition, there already are conditions in the U.S. population that put pregnant women at risk for perturbations of thyroid hormone levels: 16% of U.S. women report having any thyroid disease [22] and about 1/3 of U.S. pregnant women have insufficient iodine intake [23], which is critical to maintaining sufficient levels of thyroid hormones.

Biomonitoring studies are, for the first time, demonstrating that women of childbearing age are carrying a body burden of multiple chemicals which have been shown to disrupt the thyroid system, including PCBs, perfluorinated compounds, perchlorate and triclosan. Body burdens of these chemicals can be at least 300 to 1,500 times higher than the levels of thyroid hormone circulating in our bodies, indicating that our current interactions with our environment are exposing us to biologically relevant levels of chemicals. Indeed, separate studies have found a relationship between PCBs and perchlorate and thyroid hormone levels [24, 25].

But, the value of biomonitoring is not just in the observations of exposure. Biomonitoring studies also indicate where our chemicals policies have failed to protect us from exposures that can put us at risk of reproductive and developmental effects.

What to do.

Our current approach of using biomonitoring data as a demonstration of a problem means that it is potentially too late for those people who have already been exposed.

There are many chemicals with sufficient scientific data for the government to take action to reduce exposures. And, for the many more chemicals for which we have insufficient information, we need policies that require chemical manufacturers to provide sufficient evidence that the chemicals they want to produce do not pose undue health risks to our population.

The scientific data clearly show that every child in the U.S. is born with a burden of multiple chemicals in their body that can impact their future health. By acting now, we can improve our health and the health of generations to come.

Thank you

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