

**LEAD CONTAMINATION IN THE DISTRICT OF
COLUMBIA WATER SUPPLY**

**OVERSIGHT HEARING BY THE COMMITTEE ON
GOVERNMENT REFORM**

MARCH 5, 2004

**TESTIMONY
OF
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I am Ellen Silbergeld, and I am pleased to respond to your invitation to participate as a witness in this hearing on lead contamination in the District of Columbia water supply, and responsibilities of the District of Columbia Water and Sewer Authority and the federal government in this matter. I am testifying at your invitation, as a private citizen. For identification purposes only, I am Professor of Environmental Health Sciences at the Bloomberg School of Public Health of the Johns Hopkins University in Baltimore Maryland. I have attached a short professional resume. I have served on several state, national, and international committees and advisory boards related to preventing childhood and adult lead poisoning, including service as chair of the Maryland state panel on lead paint poisoning prevention, the CDC Scientific Advisory Committee on Childhood Lead Poisoning, and as an advisor to the World Health Organization, the International Labor Organization, the World Bank, the National Research Council, the Department of Housing and Urban Development, and the US EPA. I was a member of EPA's Science Advisory Board committee evaluating the scientific basis for EPA's current regulations concerning lead in drinking water.

I have conducted research on lead toxicity, exposures, and mechanisms for 30 years at Johns Hopkins, the National Institutes of Health, and the University of Maryland Medical School. Most recently, my research group has published three major papers on the associations between lead and cardiovascular disease, the role of genetics in lead toxicity, and the mechanisms by which lead can increase risks of cancer. This work has been recognized by several awards, notably the Barsky Award from the APHA and a MacArthur Fellowship. In 1988, the lead industry referred to me as "an ardent anti-lead activist" and I am proud of that characterization, which reflected my participation in what has been called the major environmental health achievement of the last century, the bipartisan effort to eliminate lead from gasoline.

I live in a city that last year had its own problems with lead in drinking water, including failures to disclose information and inexcusable delays in taking needed actions to protect the health of children in our schools. As in DC, it took the attention of media and pressure from parents to elicit compliance with the law. From this experience, I would suggest to this Committee that the issue before you today is unlikely to be limited to the District of Columbia. There are likely to be many public water supplies in this country where water is not being tested, or if it is tested where the information is not promptly or fully communicated to consumers, and where appropriate actions are not being taken. I urge the committee to examine this issue nationally, with the research resources of the Congress. The DC situation exemplifies the failures of a public health program based upon testing and notification by the agency that is also responsible for taking action. This in no way excuses the DC Water and Sewer Authority from its responsibilities, and I did not excuse the Baltimore City Health Department from a similar dereliction of its responsibilities. Even as of last week (www.dcwasa.com, February 27, 2004), WASA was not forthcoming in its disclosures to DC consumers: first, WASA suggests that there are never any sources of lead in the mains, and second, its survey results are presented in an incomplete and misleading fashion:

In this testimony, I will provide answers primarily to your question concerning the health risks of lead poisoning, on which I can claim expertise, and I will make some comments in response to your other questions.

1. Hazards of lead exposure

Lead is an entirely toxic metal, and its hazards have been described in medical writings for over 2000 years. Effective actions to protect the health of the general population were primarily initiated over the last 100 years although concerns over water contamination by lead were noted as long ago as the late 1700s in England and in colonial America (Schock et al 2002).

It is now the overwhelming consensus of both medical and public health experts that lead is highly toxic to multiple organ systems, including the central and peripheral nervous systems, the cardiovascular system, the kidney, and the reproductive system of both males and females. We understand that at the molecular level lead can substitute for calcium and other essential trace elements to interfere with the molecular biology of the cell at the level of DNA integrity, intra- and intercellular signaling, differentiation and development of complex systems like the nervous and reproductive systems, synaptic formation and memory storage in the brain, and vascular endothelial function. Lead is toxic to the fetus and it is carcinogenic (Silbergeld 2003). Just two weeks ago an expert group of the International Agency for Research on Cancer upgraded lead to a probable human carcinogen. In public health policy and medical guidance, we correctly focus upon preventing exposures of the developing child, pre- and postnatally, because of its effects on the developing brain, which have now been shown to persist through at least early adulthood (Silbergeld 1997). We know that lead exposures of pregnant women are quickly transferred to the fetus, and we know that prenatal exposures to lead are associated with adverse effects on early childhood growth, hearing, and cognitive development. Lead is a recognized cause of attention deficit hyperactivity disorder.

Lead exposures are also toxic to adults, increasing risks of stroke and hypertension as well as the risks of early mortality due to cardiovascular disease and cancer (Lustberg and Silbergeld 2003; Silbergeld 2003).). These risks are particularly important for women, since there is evidence that later in life bone lead stores may be mobilized back into blood over the menopause, with associated increases in blood pressure (Nash et al 2003).

The toxic effects of lead can only be prevented by preventing exposure. A large multisite study funded by NIH, including researchers at Hopkins, reported that treating children after exposure did not reverse lead toxic effects on neurocognitive function (Rogan et al 2001). We have found that earlier exposures to lead are associated with later cardiovascular disease, at a point long after the time of exposure (Lustberg and Silbergeld 2003; Navas Acien, et al , 2004)

This consensus has driven remarkable policy change in this country, including bans on certain uses of lead and controls on many other sources. As a consequence, lead exposures in the general public have decreased significantly over the past 20 years

(Silbergeld 1997). *But more recent studies suggest that our work is not over, that the toxic effects of lead on children and adults may occur even at the blood lead levels that are currently accepted in regulation and public health* (Canfield et al 2003). Moreover, recent data from the CDC national surveillance indicate that significant numbers of US children and adults are still overexposed to lead at current guidance levels, and thus continued vigilance and intervention remain important. According to the CDC (MMWR 2003), *DC failed to meet the PHS Year 2000 goal of eliminating childhood lead toxicity since in 2001, out of the 30% of children <6 yrs who were tested, nearly 3% had blood lead levels above 10 ug/dL*. It is in this context that we should consider the current issues of lead in drinking water. This source adds onto all the other sources of lead in the environment, providing an estimated 20% of total daily exposures to most US populations (Shannon and Graef 1992; Maas et al 2002). *But lead exposure via drinking water alone can by itself be sufficient to induce toxicity, especially in young infants*. In a landmark paper in 1967, Sir Abraham Goldberg and his colleagues traced the etiology of a cluster of mentally retarded children in Glasgow to the storage of drinking water in lead lined tanks (Gibson et al 1967). Shannon and Graef (1989) reported the case of an infant poisoned by drinking water with a lead concentration of 130 ppb. EPA considers that *“lead at concentrations of 40 ppb or higher poses an imminent and substantial endangerment to the health of children and pregnant women”* (www.epa.gov, drinking water information site; see Sherlock et al (1984) for data supporting this statement). This is the basis of my concern that by providing its survey data in only three categories – that is, >15 ppb; 15 up to 300 ppm, and \geq 300 ppb -- the WASA is still concealing information of great importance to consumers and to the DC Health Department.

In the 1980s, as the levels of medical concern for lead exposures were lowered, it was recognized that drinking water could be a source of increased lead exposures, primarily from the long use of lead in many parts of the water distribution system. The Safe Drinking Water Act was amended to cover the national problem of lead in drinking water. It is important to recognize that lead rarely if ever contaminates drinking water at the source -- reservoirs or groundwater -- but it can enter our drinking water through the entire distribution system including mains, service lines, in-house plumbing, water coolers, and plumbing fixtures within our houses, schools, and other buildings. National surveys undertaken at this time confirmed the widespread nature of the problem of lead in drinking water. Certain water systems were identified as presenting increased risk, including portions of the DC water system. *I served on the EPA's expert committee to evaluate a health-based standard for lead in drinking water and the EPA promulgated a health-based guidance level of zero for lead in drinking water*. Its subsequent regulations (using an enforceable level greater than zero) included a ban on the use of lead in water systems, including use of lead containing solders and brass fixtures with excess lead in residential plumbing. EPA required actions to be taken by public water systems when the enforceable standard was exceeded. The EPA also required public drinking water systems to test and communicate test results to water consumers.

Has the SDWA lead program been effective as a public health measure? The bans on lead solder and lead-adulterated brass fixtures have been largely effective at keeping new lead sources out of our water systems, but the Congress might investigate the

implementation of these portions of the SDWA regulations nationally. I myself have seen lead solder still on sale in hardware stores in Baltimore, and the city of Los Angeles has brought a major lawsuit against a supplier for using banned products in the municipal water supply. In a study conducted by Berkowitz in New Jersey (1995), a significant number of schools and daycare centers were found to have used lead solder in their plumbing *after* EPA's promulgation of the ban on new use. It is unlikely that this experience is confined to New Jersey.

The effectiveness of water treatment as a means of dealing with lead in drinking water, and the public notification programs have a much less encouraging record. The SDWA required actions to be taken when the lead guidance was exceeded, but *too much reliance was placed upon controlling simply by adjusting the parameters of water treatment*, specifically corrosion control through changing pH and alkalinity as calcium carbonate as well as the use of other additives. In fact, the EPA and our committee were pressured by the water industry to permit this "fix" in order to avoid the burden of actually replacing water lines. Water treatment controls are appropriate only to some extent, and only in some systems. Information from WASA indicates that some of the lead problems are extensive and likely to be related to lead in the mains, which will be difficult to deal with simply by flushing the lines. Moreover, there are other problems with the treatment only strategy, related to ensuring effective control over pathogens in water, also a problem in the DC system.

The other "tool" in the SDWA lead regulation is public notification and risk communication. As a public health professor, I am generally wary of relying upon these tools as extensively as the SWDA does, because they only work when testing and notification are both prompt and accurate and when consumers are able to use the information to effectively reduce their risks. As I noted above, we have unfortunate experience in Baltimore, as you do in DC, that *we cannot rely upon prompt testing and accurate notification*. In fact, placing this burden on the water system sets up a conflict of goals and responsibilities: if their tests indicate that water treatment is needed, then they must spend the money for adjusting this process; if their tests indicate that water treatment is not enough, then they must spend even more money for replacing lead sources in the system. This part of the problem could be avoided if the Congress were to appropriate sufficient funds to the states to cover these costs, but like many infrastructure problems, we have not done so even while we can apparently pay to fix the water systems of Iraq.

Does consumer notification work? Consumer information can be a powerful source for voluntary change and health promotion, but only if consumers can actually and feasibly reduce risks by individual actions. Thus, even if consumers are being properly notified, we have to examine if and how consumers can respond to notifications and effectively reduce their exposures to lead in drinking water. *EPA suggests that consumers avoid consumption of first draw water*, since if the problem of lead is confined to the proximal part of the water distribution system, then running the tap will result in purging those gallons of water that contain the highest lead levels. However, if the problem is not confined to this part of the system, then running water will not work. It does not deal

with extensive lead problems within the system, because even extensive flushing (>15 min) does not reduce the contribution of lead from lead pipes. Moreover, *the lowering effects of flushing are only temporary and lead levels rise again after as little as 15-30 min of no use* (Schock et al 1996; Murphy 1993; Gulson et al 1997). The latter authors concluded that this effect could result in unacceptable exposures to both young children and pregnant women, when water lead concentrations remained in the range of 35-50 ppb. Finally, placing the burden of risk reduction on consumers can result in unacceptable exposures to those most at risk -- for example, a young child getting up in the middle of the night for a drink of water, or a crying infant whose parents are quickly mixing formula. For the DC health department to go even further, to recommend *no* consumption of tap water to thousands of DC residents is unconscionable, as if Marie Antoinette were running public health – let them drink Evian! Given the general socioeconomics of lead risks (which are the exemplar of environmental racism), *it is likely that many of those at greatest risk will have the least means to purchase their own water*. An analysis of WASA's maps suggests this. Nonetheless, it may well be the case that for many consumers it is prudent to recommend no consumption of drinking water, including use for pets and use in humidifiers, but the burden of ensuring their safety by private purchase of bottled water should not be placed upon them. Moreover, not all bottled water is without risk including lead (Misund et al 1999; Dabeka et al 2002). The assurance of a safe and healthful water supply has been a jewel in our society for almost 100 years; this is not a matter of public health to be outsourced.

In conclusion, Mr. Chairman, in my opinion *the issue of lead in the DC water supply is a serious and immediate public health problem* for many consumers who reside or work in DC. Moreover, it is probably a signal that *similar problems may exist in many other water systems nationwide*. Second, *elements of the SDWA are demonstrably ineffective in protecting our drinking water from lead*: lack of full enforcement on product bans appears to be significant; local testing and notification programs are not monitored by the EPA; and reliance upon consumer action is not sufficient to protect the public's health. I hope you will consider this issue in its national perspectives and also in the context of the urgent need for substantial support for the states and localities in dealing with this and many other crises in our infrastructure that together belie our assumptions of a safe and healthful environment. I am happy to answer your questions on this testimony or other topics that I may be able to discuss.

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NIH BIOGRAPHICAL SKETCH

Provide the following information for the key personnel in the order listed on Form Page 2.

NAME Ellen K. Silbergeld	TITLE Professor
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EDUCATION/TRAINING (*Begin with baccalaureate or other initial professional education, such as nursing, and include postdoctoral training.*)

INSTITUTION AND LOCATION	DEGREE (if applicable)	YEAR(s)	FIELD OF STUDY
Vassar College, Poughkeepsie, N.Y.	A.B.	1967	Modern History
The Johns Hopkins University, Baltimore, MD	Ph.D.	1972	Environmental Engineering

RESEARCH AND PROFESSIONAL EXPERIENCE: Concluding with present position, list, in chronological order, previous employment, experience, and honors. Include present membership on any Federal Government public advisory committee. List, in chronological order, the titles, all authors, and complete references to all publications during the past three years and to representative earlier publications pertinent to this application. If the list of publications in the last three years exceeds two pages, select the most pertinent publications.

DO NOT EXCEED TWO PAGES.**PROFESSIONAL EXPERIENCE**

- 1972-75 Postdoctoral Fellow in Neurotoxicology: The Johns Hopkins Univ Sch of Hygiene & Public Health, Baltimore, Maryland
- 1975 Assistant Professor: Department of Environmental Medicine, The Johns Hopkins Univ Sch of Hygiene & Public Health
- 1975-79 Staff Fellow/Head: Behavioral Neuropharmacology Unit, Experimental Therapeutics Branch, NINCDS, NIH
- 1979-81 Chief: Section of Neurotoxicology, NINDS, NIH, Bethesda, MD
- 1982-84 Guest Scientist: Reproductive Toxicology Section, Pregnancy Research Branch, NICHD, NIH, Bethesda, MD
- 1982-91 Chief Toxics Scientist: Environmental Defense, Washington, D.C.
- 1987- Adjunct Faculty: Department of Health Policy & Management, The Johns Hopkins Univ Sch of Public Health
- 1990-01 Adjunct Professor: Dept of Environmental Health Sciences, The Johns Hopkins Univ Sch of Public Health
- 1991- Professor: Program in Toxicology, University of Maryland School of Medicine, Baltimore, MD
- 1992- 01 Professor: Dept of Epidemiology & Preventive Medicine, Univ of Maryland School of Medicine, Baltimore, MD
- 1993- Senior Consultant/Toxicologist: Environmental Defense Fund, Washington, D.C.
- 1996-01 Director: Program in Human Health & the Environment, Univ of Maryland School of Medicine, Baltimore, MD
- 2002- Professor, Dept of Environmental Health Sciences, Health Policy and Management, and Dept of Epidemiology, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD

PUBLIC ADVISORY COMMITTEE MEMBERSHIPS (Selected)

- 1983-89 National Research Council Board on Environmental Sciences and Toxicology
- 1983-89 EPA Science Advisory Board Executive Committee
- & 93-99
- 1987-89 Member, NSF EVIST Study Section
- 1989-92 NIH-NTP Board of Scientific Counselors
- 1990-94 Maryland Panel on Lead Poisoning (Chair)
- 1992- EPA Expert Scientific Group on Risk Assessment of Dioxins
- 1994-95 Epidemiology Panel, U.S. Army Breast Cancer Research Program
- 1994-95 Secretary of Energy Advisory Board
- 1994- Editor-in-Chief, Environmental Research
- 1996-99 HHMI Fellowship selection panel, Epidemiology (Chair, 1999)
- 1996-98 US EPA Endocrine Disruptors Screening and Testing Advisory Committee
- 1997-02 CDC Advisory Board, Center for Environmental Health
- 2000- American Cancer Society Environmental Carcinogenesis Committee
- 2000 - Member, Commission on Agricultural Biotechnology, Health and the Environment, NAS-NRC
- 1999-01 Pew Commission on Environmental Health
- 2000-01 member, expert workgroups on agricultural antibiotic use and human health, FAO-CODEX (UN) and WHO

2002- NRC Committee on Environmental Technology
 2002-03 Environmental Health Consultant, PAHO-WHO
 2002 NIEHS Site Visit Chair
 2003 NIEHS Special Interest Study Sections

HONORS & AWARDS (Selected)

1967 Graduate: *Summa cum laude* (Vassar College), Phi Beta Kappa
 1967 Leverhulme and Fulbright Fellowships
 1971-72 Rockefeller Foundation Predoctoral Research Fellowship
 1974-75 Joseph P. Kennedy Jr. Fellowship in Neurosciences
 1987 Warner-Lambert Award, Distinguished Women in Science, University of Wisconsin
 1991 Wolman Award, Maryland Public Health Association
 1992 Barsky Award, APHA
 1993 MacArthur Foundation Fellow
 1995 Women Who Make a Difference, Chatham College
 1998 Fellow, International Commission of Occupational Health WHO

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