TESTIMONY OF DR ELLEN K. SILBERGELD JUNE 15, 2010

"LEAD EXPOSURE IN D.C.: PREVENTION, PROTECTION AND POTENTIAL PRESCRIPTIONS"

SUBCOMMITTEE ON FEDERAL WORKFORCE, POSTAL SERVICE
AND THE DISTRICT OF COLUMBIA

COMMITTEE ON OVERSIGHT AND GOVERNMENT REFORM

STATEMENT OF DR ELLEN K SILBERGELD

Thank you for the invitation to provide testimony on lead in drinking water in the context of preventing lead toxicity and on the value of interventions to improve the outcomes of prior lead exposure. I am Ellen Kovner Silbergeld, Professor of Environmental Health Sciences and Epidemiology at the Johns Hopkins Bloomberg School of Public Health in Baltimore MD. I attach a copy of my professional resume, and I note those positions and activities relevant to the topics of this hearing. I have conducted research on lead toxicity, both epidemiological and mechanistic, for over 30 years as a research fellow at Hopkins, a staff fellow at NIH, and a professor at the University of Maryland Medical School, as well as at Johns Hopkins. At present I am an investigator on a research project on lead funded by NIH, in which we are examining the associations between early life exposures to lead and later risks of cardiovascular disease. I have published over 400 papers, chapters and scientific abstracts, including reviews of childhood and adult lead toxicity. I have served on several US government committees concerned with lead as an environmental health risk, including scientific advisory committees for EPA on lead in air and water and for HUD on lead in paint, and to CDC on guidelines for preventing childhood lead toxicity. I also chaired the Maryland State Advisory Council on Preventing Lead Poisoning in Children. Currently I am a member of a CDC advisory committee on interventions for lead-exposed children.

I will present testimony to you on three topics: our current understanding of the health hazards of lead to young children and others; the contribution of lead in drinking water to exposures and health risks; and the importance of interventions after exposure to mitigate toxicity to children.

1. Health hazards of lead

At present, there is extensive scientific consensus that lead is associated with significant risks to health at blood lead levels well below the guidance level for children set in 1991 (10 ug/dL) (Jusko et al 2008). For adults, there are also significant health impacts of exposures below 10 ug/dL. I note the importance of extending our public health purview to adults in light of the serious health effects of lead exposures that occur after childhood. I agree with the recent conclusion of the Human Biomonitoring Commission of the German government that it is not possible to set a level of lead exposure that is without risk due to the millenia of lead extraction

and use throughout the world. To provide an appropriate context, it has been demonstrated that a blood lead level of 1 ug/dL is more than 100 times the blood lead level experienced by human populations as recently as the 1500's (Silbergeld 1997).

The health risks of lead exposures below 10 ug/dl include impairments in neurodevelopment in children and increased risks of cardiovascular disease (and related mortality) in adults. Moreover, we now recognize that lead-induced impairments in neurodevelopment children that are measured early in life in terms of neurocognitive function are followed by highly significant risks for adolescents and young adults. Early elevations in blood lead are associated with failure to complete high school, attention deficit disorder, learning disabilities and disruptive behavior (Froehlich et al 2009; Braun et al 2006 and 2008), and a range of sociopathic behaviors including delinquency and drug use (Nevin 2009). In a national study of young adults (whose mean blood lead levels were under 2 ug/dL), there was a three-fold increase in major depressive disorders related to increases in blood lead levels (Bouchard et al 2009). Thus the early impacts of lead not only persist throughout later life, but also their severity and social impacts appear to be amplified.

Moreover, with respect to the risks of lead to adults, over the past decade our knowledge has undergone a revolution such that we can no longer ignore the risks and sources of lead exposure for the rest of us. As reviewed in several recent papers by my colleagues and me, as well as others, the scientific literature supports a causal association between very small increments in blood lead – from 0.5 to 3 ug/dL – and highly significant increases in blood pressure, risks of atherosclerosis, and premature death due to cerebrovascular disease and stroke (Navas Acien et al 2007; Navas Acien et al 2004). Cardiovascular disease is the leading cause of death in the US, and the possibility of reducing this burden by reducing both early childhood and adult lead exposures is of very great importance.

2. The contribution of lead in drinking water to elevations in blood lead levels

Lead exemplifies the importance of cumulative risk, that is, the importance of all exposures to lead in evaluating the significance of any specific exposure source. We are not "lead free" – everyone of us carries a burden of lead in our blood and possibly more importantly in our bones, which results from the sum of past and present uses of lead in our environments. Lead in drinking water is important as part of the overall contribution of lead in our environment to lead

in our bodies (Fertmann et al 2004). As reviewed by Maas et al (2005), it was estimated by EPA in 1991 that lead in drinking water at that time contributed between 14-20% of total lead exposure in the US. Additionally, as our understanding of lead toxicity impels us to re-evaluate guidelines and standards, the contribution of this source becomes more important. Maas also calculated that a child drinking 2 L of water per day at the current action level of 15 ppb would exceed a blood lead level of 5 ug/dL within a year under conditions of regular consumption.

The importance of identifying and preventing exposures to lead in drinking water can be seen in the recently published revision by CDC of screening data from DC: 30% of the children from homes with lead service lines had blood lead *levels greater than or equal to 5 ug/dL*, as compared to 15% of children from homes without the suspect lines (MMWR 5/21/2101; vol 59(19) 592).

This information challenges our current strategies for testing children and preventing lead exposures. Under current guidance from CDC (developed in 1991), local health departments are advises to establish programs that are designed to take action when children's blood lead levels exceed 10 or 15 ug/dL. Under this recommendation, the CDC guidance for risk assessment has prioritized housing and housing in poor repair as a strategy for targeted screening and interventions. However, if we accept the conclusions of research since 1991 and reset the health guidance to 5 ug/dL or lower, then the assumption that housing is the main source of elevated lead exposure no longer holds. We undertook an analysis of blood lead screening data in Baltimore City several years ago, in which we confirmed that there was a strong association between housing sources and children with blood lead levels >10 ug/dL (Aloe and Silbergeld, unpublished). However, we found no reliable association with housing for children with blood lead levels less than 10 and greater than 5 ug/dL. This same attenuation of the relationship between housing and elevated blood lead levels, for levels greater than or equal to 5 ug/dL, was observed in a national study (Bernard and McGeehin 2003).

It is noteworthy that EPA has recognized the importance of reconsidering its standards and guidance for environmental concentrations of lead. As you know, the EPA recently proposed a significant lowering of the national ambient air quality standard for lead, to 0.15 ug/m³ and this regulation was recently upheld in court. EPA is currently considering revisiting the drinking water standard for lead, as reported in Inside EPA on June 7th. I was a member of the Science Advisory Board panel that reviewed the justification for the current drinking water lead standard,

and members of that panel recommended adoption of zero as the maximum contaminant level goal, on the basis of our knowledge at that time. The enforceable standard was set at 15 ppb. Given what we know now, the current SDW standard is not acceptable, nor is the strategy for sampling in the current regulations. Congress should consider oversight of EPA's current programmatic evaluations of the drinking water standard for lead and its implementation.

3. Interventions for lead exposed children

Lamentably, many children in the US - particularly but not only in our Nation's capital as well as other major cities including Baltimore -- continue to be exposed to lead. Primary prevention the elimination of exposures – is the most effective way to prevent the individual and societal impacts of lead, but we cannot ignore the importance of interventions that can mitigate the impacts of these unprevented exposures. For that reason, there has been important research examining options for interventions after the fact of poisoning. The first studies were undertaken to determine if there were any health benefits from more aggressive chelation therapy for children with blood lead levels below 30 ug/dL. The results of a multi-site clinical trial, in which Johns Hopkins researchers participated, did not support this strategy. More recently, clinical and experimental researchers have examined the efficacy of educational and behavioral interventions for children expressing the characteristic impairments of lead toxicity such as neurocognitive impairments, impulsivity and attentional deficit disorder, and heightened aggressiveness. Some of this research has also been conducted by my colleagues at Hopkins. The Kennedy Krieger School – which is dedicated to developing and testing interventions for children with untreatable developmental disorders as well as lead poisoning has developed specific curricula and pedagogical approaches that respond to the cognitive and behavioral dysfunctions associated with lead toxicity. Experimental research, led by Prof Tomas Guilarte (now at Columbia University) demonstrated that social enrichment strategies could reverse both learning deficits and actual neurobiological changes in rats exposed to low levels of lead early in development (Toscano and Guilarte 2005).

This is an approach that has been adopted by parents and in school systems and is one of the focal points of the CDC Lead Poisoning Program. I am a member of the advisory committee that the CDC has established to consider this topic. In many respects, we have information on the efficacy of some relevant intervention programs because of the overlap between developmental lead toxicity and other major neurobehavioral problems in children, including

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conduct disorder, learning disability, and attention deficit disorder. In some school districts, a diagnosis of lead toxicity based on elevated blood lead level is one of the elibigility criteria for children to receive educational and behavioral interventions. This is an important response: as indicated above in this testimony, failing to meet the needs of lead-exposed children will increase the risks of school failure, learning disabilities, and sociopathic behaviors in the next generation of young adults.

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To summarize this testimony:

- The health impacts of lead on children are well established to occur at blood lead levels well below 10 ug/dL, and these impacts persist throughout childhood and early adulthood.
- Adults are also at risk at blood lead levels below 10 ug/dL; increased risks of cardiovascular disease and mortality associated with CVD have been demonstrated.
- Drinking water is a significant source of lead exposure for children and adults.
 With concern over lower levels of exposure, the contribution of lead in drinking water is of increasing importance
- Educational and behavioral interventions are important methods to mitigate the impacts of early lead exposure on the child and on society.

Thank you for your invitation and attention, and I would be pleased to respond to your questions and comments on this statement.

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