AMERICAN UNIVERSITY
WASHINGTON, DC

Robert Perciasepe
Administrator (Acting)
U.S. Environmental Protection Agency

DEPARTMENT OF CHEMISTRY

April 22, 2013

Dear Administrator Perciasepe:

On behalf of my co-petitioners, I am submitting on this 43rd anniversary of Earth Day a petition (enclosed) pursuant to 15 United States Code, Chapter 53, Section 2620, also known as section 21 of the Toxic Substances Control Act (TSCA), requesting that you take an action that will save the United States between $1 billion and $6 billion annually. The action will simultaneously prevent hundreds of cases of lung and bladder cancer through the reduction in the amount of arsenic now being delivered to our citizens who drink water that is fluoridated with hydrofluorosilicic acid (HFSA).

Our petition asks that you exercise authority under section 6 of the TSCA , (15 United States Code, Chapter 53, Section 2605), to prohibit the use of HFSA as a water fluoridation agent. A commercially available substitute, pharmaceutical grade sodium fluoride, delivers at least 100-fold lower levels of arsenic than does HFSA when water authorities choose to adjust their water supply to contain about 0.7 milligrams per liter of fluoride.

By prohibiting the use of HFSA the Agency will not be taking any action that would interfere with local decisions about whether or not to artificially fluoridate water supplies. Rather, the Agency would mandate that citizens of the Unites States not be subjected to unnecessarily increased cancer risks – based on the Agency's own analysis of arsenic's carcinogenicity, that as a society we reduce the cost of medical care by a substantial amount, and that the public water systems of the United States no longer be used as extremely profitable hazardous waste disposal sites for the phosphate fertilizer manufacturing industry.

As a former EPA senior scientist during your tenure as Assistant Administrator for the Office of Water, I look forward to working with you once again to help protect the health of our citizenry.

Sincerely,

J. William Hirzy

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April 22, 2013

Citizens Petition in re: Use of Hydrofluosilicic Acid in Drinking Water Systems of the United States

1. Introduction: This petition seeks a determination by the United States Environmental Protection Agency under authority of 15 United States Code, Chapter 53, Section 2605, that, where implemented, artificial fluoridation of public water supplies, which is not here challenged as a public policy measure, be accomplished exclusively by the use of pharmaceutical or food grade materials, and that, for this purpose, hazardous waste by-products of domestic and foreign industries, most particularly hydrofluosilicic acid (HFSA including its derivative sodium salt) be prohibited in the interests of public health and economy.

HFSA meets the criteria for classification as a hazardous waste (toxicity, reactivity and corrosivity) under 42 United States Code, Section 6901 et seq.

Artificial fluoridation of public drinking water supplies in the United States began on an experimental basis in 1945 using sodium fluoride, largely derived from by-products of the aluminum smelting industry. By 1951 drinking water fluoridation had become a national policy goal of the U.S. Public Health Service, and shortly thereafter sodium fluoride began to be replaced by hydrofluorosilicic acid (HFSA), a waste by-product of the phosphate fertilizer manufacturing industry. Presently, about 90 percent of drinking water systems that add fluoride use HFSA including its derivative sodium salt (known together as "silicofluorides") as the fluoridating agent., and well over 150,000,000 people in the United States consume silicofluorides in their drinking water. They are not pharmaceutical grade substances. Collectively, the public water supply systems of the United States are now a hazardous waste disposal system for these products.[1]

With the advent of fluoridated tooth pastes and other such pharmaceutical products in the 1950s and 1960s, pharmaceutical grade sodium fluoride and other fluoride containing compounds came on the market.

Silicofluoride agents used for artificial fluoridation of public water supplies contain arsenic. For example, HFSA is typically reported by suppliers to contain about 30 parts per million (ppm), or 30 milligrams of arsenic per kilogram of HFSA. This amount of arsenic in HFSA delivers about 0.078 micrograms of arsenic per liter of drinking water, based on calculations shown in Reference 1. The United States Environmental Protection Agency (USEPA) has set a health-based standard for arsenic in drinking water, known as the Maximum Contaminant Level Goal, of zero, based on arsenic's ability to cause cancer in humans. Pharmaceutical grade of sodium

[1] This petition seeks a reversal of the policy expressed in a letter from Deputy Administrator for Water, Rebecca Hanmer, to Leslie A. Russell dated March 30, 1980, insofar as it states: "In regard to the use of fluosilicic acid (sic), this Agency regards such use as an ideal environmental solution to a long-standing problem. By recovering by-product fluosilicic acid from fertilizer manufacturing, water and air pollution are minimized, and water utilities have a low-cost source of fluoride available to them."

fluoride is available containing typical levels of arsenic of less than 1 milligram per kilogram of sodium fluoride, which delivers 0.00084 micrograms of arsenic per liter of drinking water, a 99% reduction from the amount delivered by HFSA.

Of the HFSA which is disposed of in the nation's drinking water supplies, over 99 percent is used for flushing toilets, washing clothes, bathing, showering, watering lawns, and the like, and is simply widely redistributed back into the environment, illustrating the use of the nation's drinking water systems as simply a convenient way to deal with this hazardous waste and giving truth to an aphorism unworthy of the United States Environmental Protection Agency, "dilution is the solution to pollution."

Petitioners contend that, for USEPA to allow foreign and domestic fertilizer producers to dispose of their hazardous waste in the drinking water of the United States and to deny this petition on the basis of comparative prices of HFSA and USP grade sodium fluoride, it must abrogate its duty to protect public health and its stated policy on carcinogens, in this case arsenic, by mandating that a higher level of a carcinogen be present in drinking water.[2] Furthermore, as shown in Reference 1, the net cost to the citizens of the United States of using HFSA is, conservatively, at least $1 billion -$6 billion more per year than using the pharmaceutical grade of sodium fluoride. These savings are based on risk and cost of treating cancers data published by USEPA at *Federal Register* 66 (14) 6975 – 7066, January 22, 2001, in its Final Rule establishing its current Maximum Contaminant Level for arsenic.

2. Petitioner Information: Petitioners are citizens of the United States, some residing in the District of Columbia, and at the time of filing are students, alumni and faculty at American University. Other petitioners also are residents of the United States concerned about the adverse health effects attributable to the addition of silicofluorides to their drinking water. Written inquiries to or concerning the petitioners may be addressed to Dr. William Hirzy, 506 E Street, N.E., Washington, D.C. 20002.

3. Description of the Problem: The problem is two-fold: (A) direct contamination of drinking water with heavy metals, particularly arsenic, contained in hydrofluosilicic acid; and (B) subsequent contamination of drinking water flowing through piping systems with lead-brass or other lead fixtures by silicofluorides in conjunction with chloramine.

(A) Many public water authorities in the United States use hydrofluosilicic acid, CASN 16961-83-4, to adjust the fluoride level in finished drinking water to about 0.7 milligrams per liter (mg/L). The HFSA used in this way is a by-product of the manufacture of phosphate fertilizers, and is formed when phosphate containing ores, which also contain fluoride and silicon compounds among other substances, are heated with sulfuric acid. The chemical reaction between sulfuric acid and the ore mixture produces, among other substances, silicon tetrafluoride and hydrogen fluoride, which are gases that must be prevented from escaping into the environment where they would cause significant harm. See footnote 1.

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[2] **Chemical Contaminants -- Carcinogens:** If there is evidence that a chemical may cause cancer, and there is no dose below which the chemical is considered safe, the MCLG is set at zero. The **MCLG** for **arsenic** is zero.

(B) These gases, and other substances, are removed from the off-gas stream from the reactor vessel by passing them through a water spray system called a scrubber. The silicon tetrafluoride and hydrogen fluoride combine in the water to form a solution of HFSA. The concentration of HFSA in water is about 25 percent by weight when the solution is transferred to storage or transportation vessels.

Among the other substances present in the HFSA as ultimately sold to water authorities are so-called heavy metals, which include arsenic and lead. In addition, phosphate ores are known to contain radionuclides, and in fact have been a source of uranium for defense and electric power generation uses. See Reference 2. Under provisions of the Safe Drinking Water Act (SDWA), these heavy metals and radionuclides all have the same Maximum Contaminant level Goal (MCLG) of zero, meaning that the Environmental Protection Agency (EPA) does not recognize any level of these substances to be safe when in drinking water[3].

HFSA as supplied by distributors, as to which see Reference 1, contains arsenic at about 30 mg/kg of HFSA[4]. In addition it has been reported that "90 percent of the arsenic that would be contributed by treatment chemicals is attributable to fluoride addition." See Reference 3.

Some phosphate producers convert HFSA to its sodium salt by reacting the acid with sodium carbonate or sodium hydroxide, and the resulting solid, sodium hexafluorosilicate, is used rather than HFSA by some water authorities for fluoridation[5]. Petitioners assert that conversion of the acid to the sodium salt does little or nothing to reduce the levels of lead, arsenic and radionuclides in the final fluoridation product solution used for fluoridation.

Whether HFSA or its sodium salt is used by a particular water authority, work by Richard Maas et al.(Reference 4), Myron Coplan et al., (Reference 5) and Marc Edwards et al. (Reference 6) shows that chloramine, when used as a disinfectant in water systems, results in leaching of lead from lead-containing water piping systems into water (Edwards), and that when chloramine is used in conjunction with silicofluorides greatly enhanced leaching of lead into water occurs (Maas). Maas et al. have shown that when sodium fluoride is used as the fluoridating agent, rather than a silicofluoride, leaching of lead is greatly reduced or eliminated altogether. The publications by Coplan et al. and Edwards et al. offer an explanation of the elevated blood lead levels observed in children residing in Washington, D.C. in 2001-2004.

While not attributing their findings directly to increased leaching of lead from water delivery systems, Masters and Coplan (Reference 7) and Masters et al. (Reference 8) have shown that

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[3] EPA has delegated authority to set standards for contaminants introduced into drinking water through "water treatment" chemicals to NSF, Inc.. That organization has established Standard 60, which limits the amount of any contaminant having a Maximum Contaminant Level (MCL) Primary Drinking Water Standard under the SDWA that may be introduced through use of a "water treatment" chemical to less than ten percent of the MCL.

[4] While EPA delegated authority to NSF, Inc. to, in effect, assure the public through its Standard 60 that no more than ten percent of any contaminant for which a Primary Drinking Water Standard exists, evidence submitted with this petition (Reference 1) shows that water authorities (or some suppliers) feel under no obligation to provide analytical evidence to consumers that batches of HFSA they use comply with Standard 60.

[5] The solid sodium hexafluorosilicate is dissolved in water in a "day tank" to produce a solution of the hexafluorsilicate ion that is then metered into the finished water.

children drinking water fluoridated with silicofluorides are at increased risk of having elevated blood lead levels.

4. Nature and Severity of Harm Caused by Contaminants in HFSA: EPA's Drinking Water Contaminants website cites increased risk of cancer as a common concern for all radionuclides and arsenic, justifying the SDWA MCLG of zero for these substances. That same website cites delays in physical or mental development and deficits in learning abilities as concerns for exposure to lead. EPA's Integrated Risk Information System provides further justification for the SDWA MCLG of zero for lead with the following quote: "....changes...in aspects of children's neurobehavioral development may occur at blood lead levels so low as to be essentially without a threshold." The well established toxic effects of lead are dealt with by Marcus (Reference 9).

The social costs attributable to increased blood-lead levels and their sequelae associated with use of SiFs as fluoridating agents, have been addressed by Masters et al (References 10a and 10b) and are not directly a part of this analysis. These latter costs to society may well exceed those associated with cancer treatments.

For example, a recent study by Shapiro and Hassett (Reference 11) of the social cost of violent crime shows that direct (e.g., medical treatment, lost salary) and indirect (e.g. pain and suffering) costs in just four cities – Chicago, Boston, Philadelphia and Seattle – which use HFSA to fluoridate total about $10.3 billion/year. If as little as ten percent of those costs, which do not include lower housing values and reduced tax revenue, were avoided, then the social benefit of substituting USP NaF for silicofluorides would increase by about $ 1 billion/year for those four cities alone.

With respect to the excess cancer cases caused by arsenic in HFSA a recent publication by Hirzy et al., (Reference 1), shows that for typical levels of arsenic in HFSA and pharmaceutical grade NaF, the latter produces about 100 fold fewer lung and bladder cancer cases than the former. That study also shows that using typical pharmaceutical grade NaF rather than HFSA delivering an average level of arsenic reported as determined by NSF tests, as to which see the supplemental material in Reference 1, results in over 500 fold fewer lung and bladder cancer cases. On a national scale, these differences result in increased social cost to the United States from using HFSA of about $1 billion to nearly $6 billion annually as shown in Reference 1. Use of HFSA that complies with the NSF/ANSI Standard 60 for arsenic would result in 1200 fold more lung and bladder cancer cases than pharmaceutical grade NaF, resulting in increased social costs for the country of over $14 billion annually, which calls that standard into serious question. See Reference 1

The financial costs above do not account for the additional pain and suffering of those afflicted with these extra cancer cases, nor the anguish of their friends and families.

5. Petitioners' Remedy and Requested Relief: An alternative source of fluoride for water fluoridation exists that would not contribute to drinking water levels of arsenic, lead or radionuclides comparable to those in HFSA. USP Pharmaceutical grade sodium fluoride (NaF) of the quality used in some tooth pastes must contain no more than 0.003% "heavy metals."

Commercially available (Reference 1) USP Grade NaF can be purchased containing no more than 0.00002% arsenic "as As_2O_3" (equivalent to 0.000015% or 1.5 mg As/kg NaF) and no more than 0.003% "heavy metals, as lead."

With respect to lead leaching, NaF does not contribute to leaching of lead from brass water fittings or other lead-containing water delivery hardware (Reference 4). By requiring the replacement of HFSA with commercially available USP Grade NaF in those locations that choose to adjust the fluoride level of their water supply, EPA will take concrete steps to reduce levels of a contaminant (lead) for which the MCLG is zero.

In terms of convenience to water authorities with respect to reduction of leached lead levels in drinking water, those authorities, such as that serving the metropolitan Washington D.C. community, which add orthophosphate for that purpose can cease adding one salt solution (orthophosphate) and begin adding another (NaF) using essentially the same engineering. At the same time those authorities can eliminate the costly maintenance and safety aspects of dealing with highly corrosive HFSA.

Petitioners note that EPA is prohibited from *requiring* (emphasis added) the addition of "any substance" to drinking water for preventive health care purposes, and are using the example of USP grade sodium fluoride as a substitute for HFSA only to illustrate that if local or state water authorities elect to add fluoride to their water supplies, they may do so without the elevated risks of cancer or neurotoxicity to their citizens that come with HFSA.

In summary, by granting petitioners' requested relief, USEPA will reduce the levels of two drinking water contaminants for which its health based standards are zero. It will thereby reduce risks of lung and bladder cancer and of brain damage to children, as to which see References 5 through 10, and will save the nation at least $1 billion in annual health care costs through lower lung and bladder cancer rates. Even greater savings may be realized through lowering blood lead levels in the population. Petitioners reiterate that the risk levels and costs for treating lung and bladder cancer cases used above are derived from the Agency's "National Primary Drinking Water Regulations; Arsenic and Clarifications to Compliance and New Source Contaminants Monitoring; Final Rule," published in 2001 (See Reference 1).

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Comparison of hydrofluorosilicic acid and pharmaceutical sodium fluoride as fluoridating agents—A cost–benefit analysis

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ABSTRACT

Water fluoridation programs in the United States and other countries which have them use either sodium fluoride (NaF), hydrofluorosilicic acid (HFSA) or the sodium salt of that acid (NaSF), all technical grade chemicals to adjust the fluoride level in drinking water to about 0.7–1 mg/L. In this paper we estimate the comparative overall cost for U.S. society between using cheaper industrial grade HFSA as the principal fluoridating agent versus using more costly pharmaceutical grade (U.S. Pharmacopeia – USP) NaF. USP NaF is used in toothpaste. HFSA, a liquid, contains significant amounts of arsenic (As). HFSA and NaSF have been shown to leach lead (Pb) from water delivery plumbing, while NaF has been shown not to do so. The U.S. Environmental Protection Agency's (EPA) health-based drinking water standards for As and Pb are zero. Our focus was on comparing the social costs associated with the difference in numbers of cancer cases arising from As during use of HFSA as fluoridating agent versus substitution of USP grade NaF. We calculated the amount of As delivered to fluoridated water systems using each agent, and used EPA Unit Risk values for As to estimate the number of lung and bladder cancer cases associated with each. We used cost of cancer cases published by EPA to estimate cost of treating lung and bladder cancer cases. Commercial prices of HFSA and USP NaF were used to compare costs of using each to fluoridate. We then compared the total cost to our society for the use of HFSA versus USP NaF as fluoridating agent. The U.S. could save $1 billion to more than $5 billion/year by using USP NaF in place of HFSA while simultaneously mitigating the pain and suffering of citizens that result from use of the technical grade fluoridating agents. Other countries, such as Ireland, New Zealand, Canada and Australia that use technical grade fluoridating agents may realize similar benefits by making this change. Policy makers would have to confront the uneven distribution of costs and benefits across societies if this change were made.

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1. Background

The first recommendation to use fluoride for the purpose of reducing dental caries was made in the United States, but not adopted, in 1939 (Cox, 1939). Cox had studied the effect of fluoride on caries formation in rats, which followed research carried out earlier (e.g., Dean, 1938) on caries incidence in humans. In 1945 the program of adding fluoride to public water supplies for this purpose was begun on an experimental basis. The amount of fluoride in drinking water supplies of three cities, Newburgh, N.Y., Grand Rapids, MI and Brantford, Ontario, Canada was adjusted upward to approximately 1 mg/L. Later, the recommended amount of fluoride to be achieved in public water supplies was set by the U.S. Public Health Service at 0.7–1.2 mg/L, depending on local climate. In January 2011, the U.S. Department of Health and Human Services proposed lowering the recommended optimal target concentration to 0.7 mg/L (DHHS, 2011). At the time fluoridation began, the form of fluoride used was technical grade NaF. Since 1947 when NaSF was introduced (McClure, 1950), the dominant fluoride source has become HFSA itself. NaSF and HFSA together are commonly referred to as silicofluorides (SiFs). Some European countries along with some regions of Canada, New Zealand and Australia, among others countries, followed the American lead in instituting fluoridation programs using these agents. It is beyond the scope of this article to go deeply into the historical development or the economic and health public implications of fluoridation programs on an international scale. Our work is focused on information from the U.S. experience with this practice.

HFSA arises in a by-product stream from production of phosphate chemicals through the presence of various silicates, calcium fluoride and calcium fluorapatite in the phosphate ore (Denzinger et al., 1979). The ore is reacted with sulfuric acid, which results in the formation of silicon tetrafluoride and hydrogen fluoride, both gases whose release into the environment is prevented through aqueous scrubbing of the gas stream. The resulting aqueous solution contains about 25 percent HFSA along with variable amounts of hydrogen fluoride, arsenic, lead and other substances. EPA considers use of this by-product as "an ideal environmental solution to a long-standing problem. By recovering by-product fluosilicic (sic) acid from fertilizer manufacturing, water and air pollution are minimized, and water utilities have a low-cost source of fluoride available to them." (EPA, 1980). Arsenic levels in this HFSA product vary substantially (e.g. Pollock, 2011), but are typically about 30–35 mg/kg (see Supplemental Material Appendix A). It is noteworthy that HFSA arising from phosphate production in China is at least under consideration for use in the U.S., and may be under consideration – or currently being used – in other countries with fluoridation programs.

The most common form of NaF used in tooth paste in the U.S. is made by a different process and contains markedly less arsenic (Phibro, 2009 see Supplemental Material Appendix B). It is produced by reacting high purity calcium fluoride with sulfuric acid to produce hydrogen fluoride, which is then neutralized with high purity sodium carbonate or sodium hydroxide (Pollock, personal communication – email and phone – December 2, 2010).

The EPA (EPA, 2011) has set the health-based, non-enforceable drinking water standards, known as Maximum Contaminant Level Goals (MCLGs), for both As and Pb at zero. The enforceable standards, known as Maximum Contaminant Levels (MCLs) are 10 μg/L and 15 μg/L, respectively. While EPA does not regulate the amount of either contaminant that can be introduced to drinking water by substances added to water supplies, in practical terms the amount of As and Pb entering drinking water supplies in this way is regulated by NSF, Inc. and the American National Standards Institute via the NSF/ANSI Standard 60 (CDC, 2011a,b). This standard sets the maximum amount of any substance that can be added to drinking water for which EPA has established an MCL at less than 10 percent of that MCL, or less than 1 μg/L for As and less than 1.5 μg/L for Pb.

In the Supplemental Material Appendix A provides examples of the variability of the arsenic content of HFSA along with substantiation of our assertion that our use of 30 mg/kg As as an average for typical HFSA is reasonable. We show in Section 3 how this average As level in HFSA leads to an As level in finished water of 0.078 μg/L. Supplementary Material Appendix C contains a statement by the former National Fluoridation Engineer, Thomas Reeves of the U.S. Department of Health and Human Services, Centers for Disease Control and Prevention (CDC) Oral Health Division, that As levels in finished drinking water found by the entity responsible for overseeing compliance with legal requirements governing drinking water treatment chemicals, NSF, Inc., averaged 0.43 μg/L (IFIN, 2001). In our calculation of comparative risks we use both the 0.078 μg/L and 0.43 μg/L As levels as well as the maximum level that could arise from use of HFSA that complies with the NSF/ANSI Standard 60, viz. 0.99 μg/L.

There appears to be little or no concern by officials responsible for implementing water fluoridation, at least in Wellington, Florida., over the health consequences of adding as much as 1.1 μg/L of As to the local water supply via HFSA because that amount is much "less than the MCL of 10 μg/L (ppb) set by the USEPA and deemed safe for human consumption over extended periods of time." (Riebe W, personal communication – email – to Charlene Arcadipane, February 21, 2011. See Supplementary Material Appendix D).

It is noteworthy that if the assertion made by Riebe is correct, then the HFSA used in Wellington, Florida that resulted in adding that much As to the community water supply was in violation of the NSF/ANSI Standard 60 (NSF, 2011), because it added more than 10 percent of the MCL for As to that drinking water supply. The above information about the Wellington, Florida water supply is in the second paragraph of Supplementary Material Appendix D. The City of Wellington officially reported its drinking water in 2009 to have a total As content of 1.6 μg/L (Wellington, 2009).

An independent study of operations at the South Central Connecticut Water Authority published by the American Water Works Association (Weng et al., 2000) showed that HFSA contributed about 90 percent of As found in finished water. The study showed that the "normal" amount of As contributed by HFSA in that community was 0.114 μg/L.

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2. Objective

In comparing the overall costs for U.S. society between using cheaper industrial grade HFSA as the principal fluoridating agent versus using more costly pharmaceutical grade NaF the simplest component of comparison, and the one which is the subject of this paper, involves the cost of treating As-related lung and bladder cancer cases associated with HFSA versus USP NaF. We used information on numbers of cancers associated with exposure to As in drinking water and their treatment costs published by EPA when it established its current MCL for arsenic (EPA, 2001).

Another component of social cost, increased blood-lead levels and their sequelae associated with use of SiFs as fluoridating agents, has been addressed by others (Edwards et al., 2007; Maas et al., 2007; Masters and Coplan, 1999; Masters et al., 2000; Masters, 2003; Shapiro and Hassett, 2012) and is not part of our analysis. These latter costs to society may well exceed those associated with cancer treatments.

3. Method

While a larger number of cost comparisons could be made, we chose to present only four comparisons of costs associated with the As-related cancer risks presented by the two alternative fluoridating agents:

1. Typical levels of As in both agents. Calculations based on this case are shown below in this section.
2. Typical levels of As in HFSA and maximum level in USP NaF (Phibro, 2009).
3. Maximum allowed As level for HFSA permitted under NSF/ANSI Standard 60 and typical levels for USP NaF (Phibro, 2009).
4. Average level of As reported by NSF in finished water and level of As in finished water using typical USP NaF (Phibro, 2009). See Supplementary Material Appendix C.

3.1. *Estimation of comparative fluoridation costs*

We calculated the amount of each agent required to increase the fluoride level in water by 0.5 mg/L to reach 0.7 mg/L, and used sale prices (Boulder, 2007; Pollock, personal communication, 2010) for each agent to determine the cost of artificially fluoridating one liter of water. Then we calculated the size of the U.S. population receiving water fluoridated with HFSA based on available data (See below). Next we calculated the total cost in the U.S. to fluoridate the water used by the population currently receiving fluoridated water using EPA data on daily water use per capita (EPA, 2009), U.S. Department of Commerce data (DOC, 2010) on U.S. population and U.S. Public Health Service data (CDC, 1993, 2008a, 2008b) on the fraction of the U.S. population affected.

3.1.1. *Estimation of 2010 exposed population*

We used available data from CDC to estimate the number of U.S. citizens receiving water containing HFSA, employing a compromise between strict adherence to significant figure usage and maximum use of available population data. In 1992, the last year in which CDC made such data available (CDC, 1993), 10,006,000 U.S. citizens received naturally fluoridated water. We assumed that the number receiving naturally fluoridated water was unlikely to grow faster than the general population, which was 256,500,000 in 1992 (CDC, 1993). In 2010 the U.S. population was estimated at 312,000,000 (DOC, 2010). So the number receiving naturally fluoridated water in 2010 was estimated at 10,006,000 × 312,000,000/256,500,000 = 12,170,000.

We used CDC data (CDC, 2008a) on the U.S. population on public water systems receiving fluoridated water (which includes both natural and adjusted fluoride levels meeting the CDC criteria for fluoridated water) in 2008, the last year such data were made available. These data showed 269,912,000 people on public water systems, of which 72.4 percent received fluoridated water. This number is 195,500,000. We adjusted this number the same way the naturally fluoridated population was adjusted, by multiplying that value by the ratio of the 2010 U.S. population to the 2008 U.S. population (CDC, 2008a), arriving at this value: 195,500,000 × 312,000,000/304,060,000 = 200,600,000 people receiving fluoridated water. Then we subtracted the number receiving naturally fluoridated water from the total fluoridated population: 200,600,000 −12,140,000 = 188,460,000 to estimate the population receiving any fluoridation chemical.

We used data (CDC, 1993) from the last report by CDC on the populations receiving each of the three major fluoridating agents. These data showed 62.6 percent of people receiving adjusted fluoride were exposed to HFSA. Thus we estimate that currently 188,460,000 × 0.626 = 118,000,000 people are exposed to HFSA. We think this number may be lower than the actual number exposed. For instance, in Bexar County, TX prior to the 1992 fluoridation census there were 70,000 people exposed to HFSA (CDC, 2008b). Data from the 2008 census show that 1,474,000 additional people in Bexar County were receiving water fluoridated with HFSA (CDC, 2008b).

3.2. *Estimation of per capita water use*

EPA (2002) provides information on a variety of public water systems and their efforts at water conservation. The following 1998 per capita water uses for a sample set of systems – Albuqueque, NM 200 gal/day; Ashland, OR 150 gal/day; New York City 167 gal/day; Massachusetts Resources Board 136 gal/day; Seattle, WA 115 gal/day.

EPA (2009) cites household per capita water use as approximately 87 gal/day.

Considering the data from these two EPA sources we settled on the per capita value of 100 gal/day for use in our analysis.

3.3. *Risk estimation method*

We used Unit Risk values computed from EPA's Arsenic in drinking water final rule (EPA, 2001) to calculate the anticipated number of cancers attributable to use of each fluoridating agent along with estimated benefit values for

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cancer cases avoided (EPA, 2001, 7013–7019) to estimate the total cost of treating those cancers. Rather than use the unit risk as published (EPA, 2011), we instead used a lower risk factor derived from EPA's analysis presented in its Final Rule (EPA, 2001, 7008) for Arsenic in Drinking Water. This lower risk factor employs the central tendency risk rather than the upper 95 percent confidence limit risk and leads to a lower number of cancer cases attributable to As in drinking water. A further measure to avoid over estimating cases of cancer was that we did not consider As transferred to foods or beverages made or processed with fluoridated water. We followed EPA's example (EPA, 2001, 7004) by rejecting a sub-linear dose-response hypothesis in calculating the number of cancer cases that could be attributed to the use of each agent from the amount of As in each. We then compared the costs of fluoridating and treating cancers attributable to each fluoridating agent.

3.3.1. *Modified unit risk calculation (data from table III D-2[a], EPA, 2001, 7008)*

As level (μg/L)	Mean exposed population risk	Unit risk [pop. risk/(μg/L)]
3	$9.3–12.5 \times 10^{-5}$	$3–4 \times 10^{-5}$
5	$16.3–20.2 \times 10^{-5}$	$3–4 \times 10^{-5}$
10	$24.1–29.9 \times 10^{-5}$	$2–3 \times 10^{-5}$

From the above a Unit Risk value of 3.5×10^{-5}/(μg/L) was derived and used below

3.4. *Sample comparative costs calculation – case 1*

3.4.1. *Typical HFSA and typical NaF As Levels in drinking water*

HFSA mixture: HFSA has a typical level of 30×10^{-6} g As per g HFSA (as delivered as 24% assay H_2SiF_6) (see Supplementary Material Appendix A). The product's density is 1.24 g/mL, and H_2SiF_6 is 79.2% (w/w) fluoride. Therefore 1.24 g/mL × 0.24 × 0.792 = 0.235 g fluoride/mL HFSA (235 mg fluoride/mL HFSA). And 1.24 g HFSA/mL × 30×10^{-6} g As/g HFSA = 37.2×10^{-6} g As/mL HFSA

One needs about 0.5 mg fluoride/L of water to reach target fluoride level from typical surface water fluoride levels of about. 0.2 mg/L. So one needs:

0.5 mg fluoride/235 mg fluoride/mL of HFSA = 2.1×10^{-3} mL HFSA.

That much HFSA delivers 2.1×10^{-3} mL × 37.2×10^{-6} g As/mL = 7.8×10^{-8} g As = 7.8×10^{-2} μg As/L water.

NaF: One needs about 1.1 mg NaF to yield 0.5 mg fluoride/L of drinking water.

1.1 mg USP NaF × 0.76×10^{-6} mg As/mg NaF = 0.84×10^{-6} mg (0.84×10^{-3} μg) As/L water

3.4.2. *Population cancer risks: As concentration × unit risk*

HFSA: 7.8×10^{-2} μg As/L × 3.5×10^{-5}/μg As/L = 2.7×10^{-6}

NaF: 8.4×10^{-4} μg As/L × 3.5×10^{-5}/μg As/L = 2.9×10^{-8}

3.4.3. *Cancer cases: population risk[3.4.2] × exposed population[3.1.3]*

HFSA: $2.7 \times 10^{-6} \times 118 \times 10^{6} = 320$/yr

NaF: $2.9 \times 10^{-8} \times 118 \times 10^{6} = 3.4$/yr

3.4.4. *Cancer treatment costs (in $Millions)*

HFSA: 320 cases/yr × $3,500,000/case (EPA, 2001, 7013–7019) = $1120/yr

NaF: 3.4 cases/yr × $3,500,000/case = $12/yr

NaF advantage: $1120 – $12/yr = $1,108/yr

3.4.5. *Annual fluoridation costs*

Per capita water use: 100 gal. = 380 L [3.2]

Total daily water use by exposed population 380 L/day - × $1.18 \times 10^{8} = 4.5 \times 10^{10}$ L

HFSA: Sales price $500/ton; $0.25/lb (Boulder, 2007)

Need 2.1×10^{-3} mL/L H_2O × 1.24 g/mL × 1 lb/454 g × $0.25/lb = 1.4×10^{-6}/L

1.4×10^{-6}/L × 4.5×10^{10} L/day × 365 day/yr = $23 × 10^{6}/yr

NaF: Sales price $3/lb (Pollock, 2010, personal communication)

Need 1.1×10^{-3}g/L × 1 lb/454 g × $3/lb = 7.3×10^{-6}/L

7.3×10^{-6}/L × 4.5×10^{10} L/day × 365 day/yr = $120 × 10^{6}/yr

HFSA advantage ($Millions): $120 – $23/yr = $97/yr

3.4.6. *Annual total social costs ($Millions)*

HFSA Cancer treatment cost [3.4.4] plus fluoridation cost [3.4.5]

$1120 + $ 23 = *$1143/year*

NaF: Cancer treatment cost [3.4.4] plus fluoridation cost [3.4.5]

$12 + $120 = *$132/year*

NaF advantage: $1143 – $132 = *$1011/year*

4. Results

The additional cost of purchasing and using pharmaceutical grade NaF over technical grade HFSA is about $97 million annually. Cancer risk reduction factors, based on the ratio of the As level in HFSA fluoridated water to the As level in NaF fluoridated water are in Table 1. The corresponding numbers of cancers along with the estimated treatment costs associated with each fluoridating agent are in Table 2. In Table 3 the realistic net annual social cost savings by using NaF is shown to range from about $1 billion (Case 1) to about $6 billion (Case 4) as shown in Table 3.

We show in the Supplementary Material that with As levels that would pass the NFS/ANSI Standard 60, i.e. 380 ppm As, the savings could be as great as $14 billion/year, and that based on the reported actual amount of As delivered to the Wellington, FL water system (Riebe, 2011), the savings would be about $16 billion/year.

Additional social cost savings would be realized through avoidance of the consequences of elevated blood lead levels. We did not attempt to assess these additional social cost savings because of the complexities and uncertainties associated with the resulting effects, especially on lowering IQ of children exposed to higher lead levels. Nevertheless, based on one very limited example that has been analyzed (Masters, 2003), we recognize that there are potentially greater social cost savings attributable to avoiding this effect than from lowering cancer rates associated with As exposure.

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Table 1 – Comparison of expected cancer cases HFSA vs. U.S.P. NaF.

Case	Fluoride source	mg As/kg source	μg As/L H_2O	Cancer risk per million	Lung/bladder Cancers
1	Typical HFSA	30	0.078	2.7	320
	Typical NaF	0.76	0.00084	0.029	3.4
2	Typical HFSA	30	0.078	2.7	320
	Max. NaF	1.5	0.0017	0.06	7
3	Max. HFSA[a]	380	0.99	35	4100
	Typical NaF	0.76	0.00084	0.029	3.4
4	NSF HFSA	nd[b]	0.43	15	1800
	Typical NaF	0.76	0.00084	0.029	3.4

[a] Maximum As level permitted under NFS/ANSI Std 60.
[b] As reported by NSF, Inc in terms of average As level in treated water. As level in the HFSA used was not reported (Reeves in IFIN, 2001; see Supplementary Material Appendix C).

Table 2 – Comparison of cancer treatment costs.

Case	Fluoride source	Lung/bladder cancer cases	Treatment cost ($Millions)	Cost savings ($Millions)
1	Typical HFSA	320	1120	
	Typical NaF	3.4	12	1108
2	Typical HFSA	320	1120	
	Max. NaF	8	24	1096
3	Max. HFSA[a]	4100	14,350	
	Typical NaF	3.4	12	14,338
4	NSF HFSA[b]	1800	6,300	
	Typical NaF	4	12	6388

[a] Maximum As level permitted under NFS/ANSI Std 60.
[b] As reported by NSF, Inc in terms of average As level in treated water. As level in the HFSA used was not reported (Reeves in IFIN, 2001; see Supplementary Material Appendix C).

Table 3 – Comparison of total social costs.

Case No.	Fluoride source	Chemical cost ($ Millions)	Cancer treatment cost ($ Millions)	Total social cost ($Millions)	Social cost saving ($ Millions)
1	Typical HFSA	23	1120	1143	
	Typical NaF	120	12	132	1011
2	Typical HFSA	23	1120	1143	
	Max. NaF	120	24	144	999
3	Max. HFSA[a]	23	14,350	14,373	
	Typical NaF	120	12	132	14,241
4	NSF HFSA[b]	23	6300	6323	
	Typical NaF	120	12	132	6191

[a] Maximum As level permitted under NFS/ANSI Std 60.
[b] As reported by NSF, Inc in terms of average As level in treated water. As level in the HFSA used was not reported. (Reeves in IFIN, 2001; see Supplementary Material Appendix C)

5. Conclusions

Our analysis shows that if local governments that currently add HFSA to their drinking water wish to continue delivering fluoride to their citizens and at the same time reduce the number of lung and bladder cancers among their citizens, they could do so with a significant net benefit to society by switching to USP NaF for fluoridation. We note that with respect to As added to water supplies by fluoridation additives, NSF/ANSI Standard 60 allows for significant cancer treatment costs, up to $14 billion annually, to be incurred by society.

It is obvious that the benefits and cost associated with a switch from HFSA to the grade of fluoride used in tooth paste are distributed unevenly across a society organized as is the U.S. That is, local governments that would purchase the more expensive fluoridating agent are unlikely to be the direct beneficiary of reduced cancer treatment costs. We cannot go further in this paper than to recommend that in the U.S. our results stimulate a study, perhaps by the Congressional Research Service, of how our society would deal with the cost/benefit distribution question.

The last Congressional review of the national program of water fluoridation in the U.S. took place over thirty years ago, and a great deal of new knowledge has been developed during that time. What we have presented here is but a small, but we think important, portion of that new knowledge.

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.envsci.2013.01.007.

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Supplemental Material

Comparison of Hydrofluorosilicic Acid and Pharmaceutical Sodium Fluoride as Fluoridating Agents – a Cost-Benefit Analysis

J. William Hirzy, Robert J. Carton, Christina D. Bonanni, Carly M. Montanero, Michael F. Nagle

Table of Contents

Appendix A: Exemplary Arsenic Content of Hydrofluorosilicic Acid

The following arsenic levels reported in sample Certificates of Analysis (COAs) and analytical results obtained from the City of Denver (Pollock 2011) for HFSA show that our assumption of 30 ppm As HSFA is reasonable.

City of Denver Analyses of HFSA:		COA Source/Date	As Content
Date:	ppm As	Mosaic Co. 10/04/2010	40.75 ppm
12/1/2003	20	LCI Ltd. 10/1990	"Typical..0.0035%" (35 ppm)
6/18/2004	73	Aurora Co. 5/16/2010	5.18 ppm
11/22/2004	21		
3/1/2005	32		
8/10/2004	26		
8/15/2004	<2		
12/17/2007	44		
1/14/2009	42		
7/7/2010	37		
8/16/2010	61		
10/11/2010	39		

The email message that conveyed the above excerpted Denver results is below (Pollock 2011).

"From: Pollock, Kenneth A. <Kenneth.Pollock@denverwater.org>
Date: Wed, Apr 27, 2011 at 4:53 PM
Subject: Fluoride Information
To: "mn1350a@student.american.edu" <mn1350a@student.american.edu>
Cc: "Sloan, Joseph A." <Joseph.Sloan@denverwater.org>, "Bond, Matt" <Matt.Bond@denverwater.org>

"Mike,

"Here's the information you've requested on the analysis of the fluoride products that Denver Water uses. The spreadsheet includes data from samples analyzed by a Denver Water contract lab. Sorry for the delay in getting you this information.

"Regards,

"Ken Pollock
Denver Water 303-628-6632"

Appendix B: Technical Data. Sodium Fluoride. USP/FCC Grade

Phibro
PhibroChem Division

TECHNICAL DATA

SODIUM FLUORIDE
USP / FCC Grade

TYPICAL ANALYSIS

Assay, as NaF	%	98.0 – 102.0
Fluoride, as F	%	44.0 - 46.0
Chloride, as Cl	%	0.008
Heavy Metals, as Pb	%	0.003 Max.
Iron, as Fe	%	0.002
Arsenic, as As_2O_3	%	0.0002 Max
Loss on Drying	%	1.0
Fluosilicate	-	Passes test
Alkalinity	-	Passes test
Identification tests A & B	-	Passes test
Solubility test, 2% (W/V) water solution	-	Passes test
Texture	-	Passes test

This material conforms to requirements of the current USP monograph in all respects.
No organic volatile materials are used in its manufacture.

PHYSICAL PROPERTIES

White, odorless, fine powder.

PACKAGING

50 & 100 lb fiber drums; 500 kg supersacs

C.A.S. No.: 7681-49-4

TD041.01 / 13 – 05/08/09

A Division of Phibro Animal Health Corporation
65 Challenger Road, Third Floor, Ridgefield Park, NJ 07660
1-800-223-0434 • 201-329-7300 • Fax: 201-329-7094

Appendix C: Manufacture of Fluoride Chemicals

Letter from Thomas Reeves, CDC Fluoridation Engineeer to Paul Connett, Director Fluoride Action Network. (IFIN 2001)

"The Manufacture of Fluoride Chemicals

"A number of questions have been raised about the fluoride chemicals used in water fluoridation.

"This communication will attempt to respond to those concerns.

"All of the fluoride chemicals used in the U.S. for water fluoridation, sodium fluoride, sodium fluorosilicate, and fluorosilicic acid, are byproducts of the phosphate fertilizer industry. The manufacturing process produces two byproducts: (1) a solid, calcium sulfate (sheetrock, CaSo4); and (2) the gases, hydrofluoric acid (HF) and silicon terafluoride (SiF4). A simplified explanation of this manufacturing process follows: Apatite rock, a calcium mineral found in central Florida, is ground up and treated with sulfuric acid, producing phosphoric acid and the two byproducts, calcium sulfate and the two gas emissions. Those gases are captured by product recovery units (scrubbers) and condensed into 23% fluorosilicic acid. Sodium fluoride and sodium fluorosilicate are made from this acid.

"The question of toxicity, purity, and risk to humans from the addition of fluoride chemicals to the drinking water sometimes arises. Almost all of over 40 water treatment chemicals that may be used at the water plant are toxic to humans in their concentrated form, e.g., chlorine gas and the fluoride chemicals are no exception. Added to the drinking water in very small amounts, the fluoride chemicals dissociate virtually 100% into their various components (ions) and are very stable, safe, and non-toxic.

"Opponents of water fluoridation have argued that the silicofluorides do not completely dissociate under conditions of normal water treatment and thus may cause health problems. To counter these claims, the basic chemistry of this dissociation has been carefully reviewed. Scientists at the U.S.Environmental Protection Agency (EPA) and CDC epidemiologists have examined the research that opponents of water fluoridation cite. Both groups have concluded that these charges are not credible.

"The claim is sometimes made that no health studies exist on the silicofluoride chemicals used in water fluoridation. That is correct. We, the scientific community, do not study health effects of concentrated chemicals as put into water, we study the health effects of the treated water, i.e., what those chemicals become: fluoride ion, silicates and the hydrogen ion. The health effects of fluoride have been analyzed by literally thousands of studies over 50 years and have been found to be safe and effective in reducing tooth decay. The EPA has not set any Maximum Contaminant Level (MCL) for the silicates as there is no know health concerns for them at the low concentrations found in drinking water. Of course, the hydrogen ion is merely a measurement of the pH of the water.

"Concern has been raised about the impurities in the fluoride chemicals. The American Water Works Association (AWWA), a well-respected water supply industry association, sets standards for all chemicals used in the water treatment plant, including fluoride chemicals. The AWWA standards are ANSI/AWWA B701-99 (sodium fluoride), (ANSI/AWWA B702-99 (sodium fluorosilicate) and ANSI/AWWA B703-00 (fluorosilicic acid). Also, the National Sanitation Foundation (NSF) sets standards and does product certification for products used in the water industry, including fluoride chemicals. ANSI/NSF Standard 60 sets standards for purity and provides testing and certification for the fluoride chemicals. Standard 60 was developed by NSF and a consortium of associations, including AWWA and the American National Standards Institute (ANSI),. Standard 60 provides for product quality and safety assurance that aims to prevent the addition of harmful levels of contaminants from water treatment chemicals. More than 40 states have laws or regulations requiring product compliance with Standard 60. NSF tests the fluoride chemicals for the 11 regulated metal compounds that have an EPA MCL. In order for a product [for example, fluorosilicic acid] to meet certification standards, regulated metal contaminants must be present at the tap [in the home] at a concentration of less than the percent of the MCL when added to drinking water at the recommended maximum use level. EPA has not set any MCL for the silicates as there is no know health concerns, but Standard 60 has a Maximum Allowable Level (MAL) of 16 mg/L [for sodium silicates as corrosion control agents] primarily for turbidity reasons. NSF tests have shown the silicates in the water samples to be well below these levels.

"Arsenic, according to NSF tests, had an average of 0.43 ug/L (parts per billion) in the drinking water attributable to the fluoride chemical. Opflow, a monthly magazine from the AWWA, has found the arsenic levels in the finished water from the fluorosilicic acid to be 0.245ug/L [Opflow, Vol 26, No. 10, October, 2000]. The NSF Standard 60 has a Maximum Allowable Level (MAL) of 2.5 ug/L and EPA has a MCL of 50 ug/L, although they have proposed to lower their MCL to 5 ug/L. As you can see arsenic is less than 1/10th of even the proposed EPA MCL. Finally, tests by NSF and other independent testing laboratories have shown no detectable levels of radionuclides in product samples of fluoride chemicals. There is no evidence that any of the known impurities in the fluoride chemicals have failed to meet any of these standards.

"Opponents of water fluoridation have sometimes charged that "industrial grade fluoride" chemicals are used at the water plant instead of pharmaceutical grade chemicals. All the standards of AWWA, ANSI, and NSF apply to these industrial grade fluoride chemicals to ensure they are safe. Pharmaceutical grade fluoride compounds are not appropriate for water fluoridation, they are used in the formulation of prescription drugs.

"Finally, it is sometimes alleged that the fluoride from natural sources, like calcium fluoride, is better than fluorides added "artificially", such as from the fluoride chemicals presently used. There is no difference.

"There is no reason to change the opinion of CDC that water fluoridation is safe and effective.

"DOH"

(Written at bottom) Reference - Tom Reeves, water engineer, CDC Jan-2001

Appendix D: re: Wellington, FL City Water Arsenic Content (Riebe 2011)

"-----Original Message-----
From: Bill Riebe <wriebe@wellingtonfl.gov>
To: chararcadipane@aol.com <chararcadipane@aol.com>
Cc: Darell Bowen <dbowen@wellingtonfl.gov>; Matthew Willhite <mwillhite@wellingtonfl.gov>; Dr. Carmine Priore <CPriore@wellingtonfl.gov>; hchcoates@wellingtonfl.gov <hchcoates@wellingtonfl.gov>; Anne Gerwig <AGerwig@wellingtonfl.gov>; PaulSchofield <pauls@wellingtonfl.gov>; John Bonde <jbonde@wellingtonfl.gov>; JimBarnes <jbarnes@wellingtonfl.gov>; Francine Ramaglia <FrancineR@wellingtonfl.gov>
Sent: Mon, Feb 21, 2011 5:20 pm
Subject: RE: Fluoride

"Ms. Arcadipane,

"A copy of Wellington's Annual Drinking Water Quality Report (for calendar year 2009) is attached for your use. Wellington publishes this report and mails it to each of its customers each year to let them know about the quality of Wellington's drinking water. As noted in the Report, the level of fluoride detected in Wellington's drinking water ranged from 0.56 mg/l (ppm) to 1.17 mg/l (ppm) – with an average concentration of 0.8 mg/l. Please note that approximately 0.2 mg/l (ppm) of the fluoride in Wellington's drinking is from the ground water – naturally occurs. The maximum contaminant level (MCL) for fluoride is 4 mg/l (ppm). The levels of fluoride in Wellington's drinking water are well below the USEPA MCL of 4 mg/l.

"As shown in the report, the level of arsenic detected was 1.6 micrograms/liter (ppb). Please note that 0.5 micrograms per liter (ppb) of the arsenic in Wellington's drinking water (1/3 of the total) is from the ground water – naturally occurs. This means that arsenic is added to the drinking water (approximately 1.1 micrograms per liter) as part of the fluoridation process (2/3 of the total). The maximum contaminant level (MCL) for arsenic is 10 micrograms/liter (ppb). Nonetheless, the total level of arsenic in Wellington's drinking water is nearly 6 times less than the MCL of 10 micrograms per liter (ppb) set by the USEPA and deemed safe for human consumption over extended periods of time.

"Wellington purchases its hydrofluosilicic acid from Harcros Chemicals, Inc. of Tampa, Florida. Please go to the following link to verify Harcros's NSF60 certification for fluoride - http://www.nsf.org/Certified/PwsChemicals/. A copy of the certification also is attached for your use. Please note that fluoride produced in accordance with the NSF60 certification means that it meets the purity requirements to be used in drinking water at prescribed doses. The fact that Wellington only has 1.6 ppb of arsenic in its drinking water is evidence of the quality of the hydrofluosilicic acid that Wellington uses.

"As noted in previous correspondence, Wellington continually monitors developments for all water quality parameters, including fluoride. In response to the US Department of Health and Human Services (USHHS) notice for public input concerning fluoride levels (1/7/11), Wellington voluntarily reduced its average fluoride levels on 1/17/11 from 0.8 mg/l to 0.7 mg/l. The 0.7 mg/l dosage conforms to the recommendations by the USHHS. Please note that the USHHS is not recommending the discontinuation of drinking water fluoridation. In fact, the USHHS specifically recognizes the benefits of drinking water fluoridation and recommends the continuation of the same using new guidelines.

"In regard to reports/studies, Wellington receives regular updates/information from several trade organizations and from the Florida Department of Environmental Protection and other agencies. Some of the most pertinent reports regarding fluoride are provided below along with references.

"On August 17, 2001, the US Center for Disease Control issued a report recommending fluoridation of drinking water. Below are listing of the references cited in the report.

"2001 CDC REPORT References (*The original message then contains citations to 270 literature references, and other attachments giving rise to a message size of 4.5 MB, and then ends with...)*

"In December, 2010, the USEPA published a report entitled "Fluoride: Dose Response Analysis for Non-Cancer Effects" in response to the 2006 NRC Report (above) recommending further study of fluoride. The link to the report is http://water.epa.gov/action/advisories/drinking/upload/Fluoride_dose_response.pdf. The report noted a Point of Departure (POD) of 1.87 mg/l of fluoride ingestion per day - above which the effects of fluorosis become noticeable. This report does not recommend discontinuation of drinking water fluoridation and recognizes the public health benefits of the same. Numerous references are provided at the end of the report.

"Also in response to the 2006 NRC Study, the USEPA published another report entitled 'Fluoride: Exposure and Relative Source Contribution Analysis'. The link to this report is http://water.epa.gov/action/advisories/drinking/upload/Fluoridereport.pdf. The purpose of this report was to: 1) identify sources of fluoride for children and adults; 2) Quantify fluoride exposures; 3) Quantify fluoride intake by source (drinking water, food, toothpaste, etc.) and 4) Provide information to reduce risks associated with fluoride in drinking water. This report does not recommend discontinuation of drinking water fluoridation and recognizes the public health benefits of the same.

"Based upon the information contained in the 2010 USEPA Reports, the US Department of Health and Human Services issued a revised dosage level for communities practicing drinking water fluoridation. The new level is 0.7 mg/l. The old level ranged from 0.7 mg/l to 1.2 mg/l depending on the climate of the community. The thought was that higher doses of fluoride were needed in colder climates since people drank less water. The corollary was used for warm climates. The reports above indicated that this assumption no longer is valid...in other words, water consumption (and associated fluoride consumption) is not related to climate. As noted in the recent reports, the USEPA intends to conduct additional studies to evaluate and possibly reset the MCL for fluoride. As noted above, Wellington immediately adjusted fluoride levels to match the USHHS recommendations.

"The USEPA, CDC and NRC websites have tons of information regarding fluoride and drinking water fluoridation practices. A fact sheet for fluoride can be found at the USEPA at http://water.epa.gov/action/advisories/drinking/upload/fluoridefactsheet.pdf.

"I hope this information answers your questions and provides some helpful information.

Best Regards,

Bill Riebe, P.E., CGC
City Engineer
561-753-2454"

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United States Patent [19]

Demarthe et al.

[11] **4,311,676**

[45] **Jan. 19, 1982**

[54] **PROCESS FOR THE RECOVERY OF URANIUM CONTAINED IN PHOSPHATED COMPOUNDS**

[75] Inventors: **Jean-Michel Demarthe**, Viroflay; **Serge Solar**, Rambouillet, both of France

[73] Assignee: **Minemet Recherche**, Trappes, France

[21] Appl. No.: **17,164**

[22] Filed: **Mar. 2, 1979**

[30] **Foreign Application Priority Data**

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Feb. 23, 1979 [FR] France 79 4761

[51] **Int. Cl.**[3] **B01D 11/04**

[52] **U.S. Cl.** **423/10**; 423/15; 423/21.5; 423/139; 423/143

[58] **Field of Search** 423/10, 15, 21.5, 139, 423/140, 143; 210/21

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Primary Examiner—Richard E. Schafer
Attorney, Agent, or Firm—Fleit & Jacobson

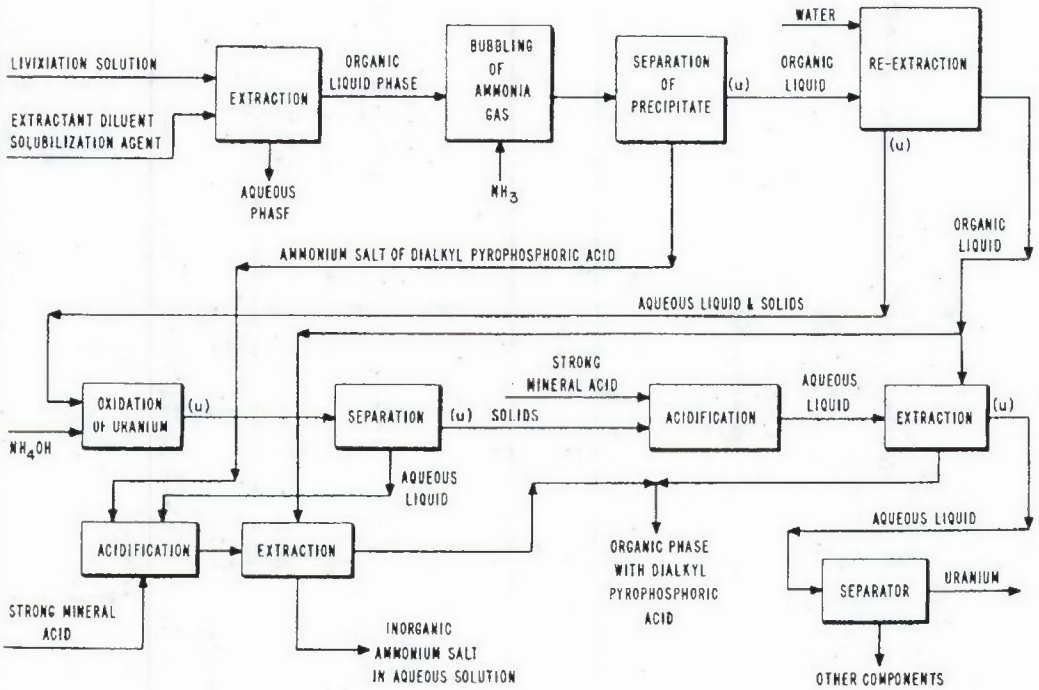
[57] **ABSTRACT**

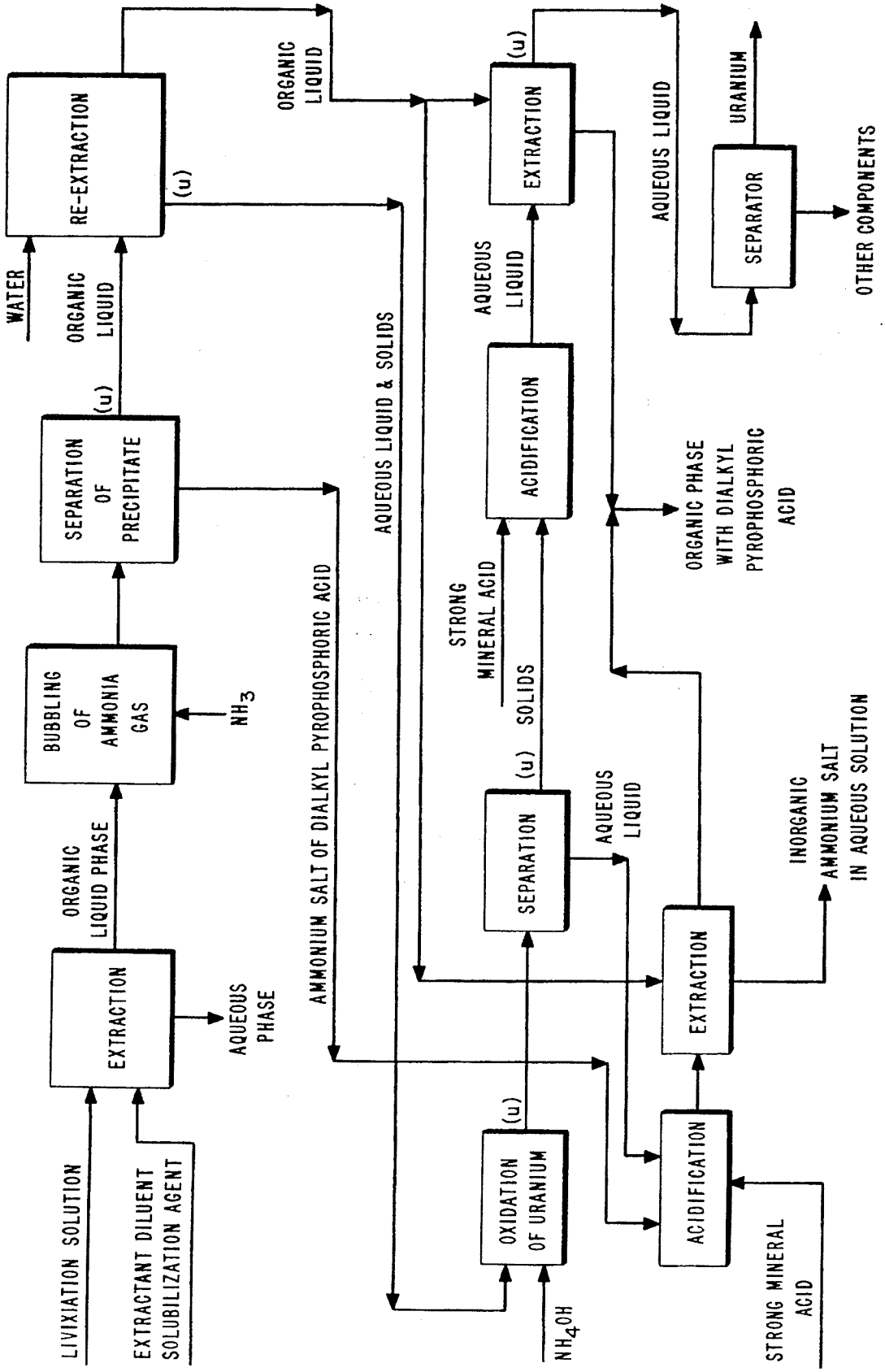
This present invention provides a process of recovering uranium from a uranium-containing acid lixiviation solution comprising extracting the lixiviation solution with an organic extractant comprising an organic diluent, a dialkyl pyrophosphoric acid, and a stabilization agent, and treating the mixture of solutions as follows:

(a) bubbling anhydrous ammonia gas into the solution mixture to precipitate the ammonium salt of excess dialkyl pyrophosphoric acid; and

(b) separating the precipitate from the solution and recovering the uranium from the solution.

20 Claims, 1 Drawing Figure




LIVIXIATION SOLUTION
EXTRACTANT DILUENT SOLUBILIZATION AGENT
EXTRACTION
ORGANIC LIQUID PHASE
AQUEOUS PHASE
BUBBLING OF AMMONIA GAS
NH_3
SEPARATION OF PRECIPITATE
(u)
WATER
ORGANIC LIQUID
RE-EXTRACTION
(u)
ORGANIC LIQUID
AMMONIUM SALT OF DIALKYL PYROPHOSPHORIC ACID
AQUEOUS LIQUID & SOLIDS
OXIDATION OF URANIUM
(u)
NH_4OH
SEPARATION
(u)
SOLIDS
AQUEOUS LIQUID
STRONG MINERAL ACID
ACIDIFICATION
AQUEOUS LIQUID
EXTRACTION
(u)
ORGANIC PHASE WITH DIALKYL PYROPHOSPHORIC ACID
AQUEOUS LIQUID
SEPARATOR
URANIUM
OTHER COMPONENTS
EXTRACTION
INORGANIC AMMONIUM SALT IN AQUEOUS SOLUTION
ACIDIFICATION
STRONG MINERAL ACID

PROCESS FOR THE RECOVERY OF URANIUM CONTAINED IN PHOSPHATED COMPOUNDS

BACKGROUND OF THE INVENTION

1. Field of Invention

The present invention relates to a process for the recovery of the uranium contained in phosphated compounds, solid or in solution. More specifically, the present invention is directed to a process for the recovery of the uranium present in an organic phase in the form of a salt of a pyrophosphoric diester.

2. Description of the Prior Art

It is known that the natural phosphates are, essentially, compounds of tricalcic phosphates which absolutely cannot be assimilated by plants, so that they cannot be used as fertilizer. This is why transforming tricalcic natural phosphates into phosphates assimilable by plants and, especially into superphosphates which constitute the widely used fertilizers, represents the heart of the activity of the phosphate industry.

The first stage of this transformation is lixiviation with an acid, usually sulfuric, which converts the tricalcic phosphate into phosphoric acid and into insoluble calcium sulfate. In addition, this lixiviation liberates the different metals such as uranium, thorium, vanadium and the rare earth metals which are trapped in phosphate deposits over the ages.

The concentration of these metals in phosphated ores is very low. Thus, for uranium, it is generally between 0.005 and 0.02%, by weight. Although these concentrations are low, because of the high market value of uranium and because of its strategic characteristics, extensive research has been conducted in the last 25 years to recover the uranium contained in acid lixiviation solutions of phosphated ores.

Among the various recovery processes which have been proposed, a particular one has found important industrial use. This process consists of submitting the lixiviation solution to a liquid-liquid extraction by means of an organic phase containing a diester of pyrophosphoric acid, which will also be called herein dialkyl pyrophosphoric acid.

This technique which is described in detail in U.S. Pat. No. 2,866,680 permits very rapid and very complete removal of the uranium present, even in very low concentrations, in different types of solutions.

However, in spite of the advantages, there are serious drawbacks in this process. The pyrophosphates hydrolyze very rapidly, which necessitates frequent regeneration of the organic phase, thus raising the cost in using this process. Also, the pyrophosphate-uranium complex is so stable that it requires a very powerful reagent to reextract the uranium.

Thus, until now experiments of elution in an alkaline medium have not permitted the regeneration of the pyrophosphoric ester and have generally led to the precipitation of an organic pyrophosphate of uranium which had to be calcined in order to eliminate all organic traces.

Until recently, only the use of hydrofluoric acid gave appreciable results for this reextraction. However, the use of this acid on one hand accelerates considerably the hydrolysis of the pyrophosphates, with all the drawbacks that entail and which have been mentioned above, and on the other hand, requires the use of very costly materials because of the toxic and corrosive characteristics of hydrofluoric acid.

An additional drawback of the above-mentioned process lies in the fact that the diesters of pyrophosphoric acid are not very selective, so that they extract, in addition to uranium, an important part of the other metals trapped in the phosphates. Upon reextraction, these metals are also precipitated by the hydrofluoric acid and they make the uranium fluoride so obtained very impure.

An important step forward in the recovery of the uranium contained in phosphated solutions is described in French patent application No. 77-25899 submitted by the present assignee. Nevertheless, this process requires the application of material which is relatively expensive because of the colloidal nature of the precipitates and the relative difficulty of separating the liquid, aqueous, or organic phases from the solid phases. The colloidal nature of the precipitates is essentially due to their impregnation by compounds of dialkyl pyrophosphoric acid.

SUMMARY OF THE INVENTION

Accordingly, it is one of the objectives of the present invention to provide a new process for the recovery of the uranium present in phosphated compounds by means of an organic phase containing a dialkyl pyrophosphoric acid, a process in which the consumption of this latter material is strongly decreased compared with presently known processes.

Another objective of this invention is to provide a process for the reextraction of the uranium present in an organic phase which contains a diester of pyrophosphoric acid, and this process avoids the use of hydrofluoric acid.

An additional objective of this invention is to provide a new process for the recovery of the uranium contained in phosphated solutions which permits easy operation for liquid-solid and liquid-liquid separation of the salts obtained and which reduces the impregnation of the latter by the compounds of dialkyl pyrophosphoric acid.

DETAILED DESCRIPTION OF THE INVENTION

These objectives, as well as others which will appear in due course, are attained by extracting the uranium-containing acid lixiviation solution with an organic phase containing a diluent, a dialkyl pyrophosphoric acid, and at least 0.5 mole of a solubilization agent for the double dialkyl pyrophosphate of uranium and ammonium per mole of dialkyl pyrophosphoric acid, and applying the following steps:

(a) bubbling anhydrous ammonia gas into the organic phase, which has been previously charged with uranium, to precipitate the ammonium salt of excess dialkyl pyrophosphoric acid;

(b) carrying out a liquid-solid separation to separate from the organic phase the ammonium salt of the dialkyl pyrophosphoric acid precipitated in step (a).

The alcoholic chains (i.e. alkyl) of the dialkyl pyrophosphoric acid specified above may be linear or branched. They may also include functions other than the alcohol function and, specifically, they may have one or several other functions. As alcohols susceptible of being esterified to give dialkyl pyrophosphates having one or several ether functions, one may cite the

alcohols obtained by the condensation of a heavy alcohol such as that defined below, with an alkylene oxide, such as ethylene or propylene oxide. By the expression "heavy alcohol" used above, it is meant an alcohol whose alkyl radical includes from 4 to 20 carbon atoms and preferably from 6 to 12 carbon atoms.

As a diester of pyrophosphoric acid, one may, in particular, use dicapryl pyrophosphoric acid, or dioctyl pyrophosphate which will sometimes be referred to by the abbreviation: "OPPA" (octyl pyrophosphoric acid).

As to the condensation of the above-defined alcohols with phosphoric anhydride P_2O_5, a condensation which leads to the pyrophosphates used in the process according to the present invention, it may be carried out according to the method described in U.S. Pat. No. 2,947,774.

On the other hand, research which has lead to the present invention has shown that one could choose, as solubilization agent, liposoluble compounds which have electronic doublets susceptible of giving hydrogen bonds with the diesters of pyrophosphoric acid. Among these compounds, one may mention such trialkyl phosphates as, for example, tributyl phosphate, which will sometimes be referred to by the abbreviation: "TBP" (tributyl phosphate), the oxides of trialkylphosphine as, for example, trioctylphosphine oxide, and the heavy alcohols defined above.

These compounds also increase the pyrophosphates' resistance to hydrolysis.

Preferably, the organic phase contains from about one to five moles, more preferably from about 2 to 4 moles, of a solubilization agent per mole of dialkyl pyrophosphoric acid. Examples of the preferred solubilization agents include the primary and secondary alcohols which follow: pentanol, hexanol, heptanol, octanol, nonanol, decanol, undecanol, dodecanol, with heptanol 1 and 2, octanal 1 and 2, nonanol 1 and 2 and decanol 1 and 2 being preferred.

It may also be mentionned that it is more convenient to use as solubilization agent the same alcohol as the one condensed with phosphoric anhydride to form the dialkyl pyrophosphoric acid.

The pressure in which the invention may be carried out is preferably atmospheric pressure for reasons of simplicity.

The temperature must not be higher than about 50° C.; otherwise, the dialkyl pyrophosphoric acid hydrolyzes very rapidly. Thus, the present process is preferrably carried out within a temperature range of from about 15° to about 30° C.

The concentration of dialkyl pyrophosphoric acid in the organic phase is preferably between 1 and 10% in weight, the rest of this phase being composed of the solubilization agent and organic diluent which is chosen from those well known in the art, such as kerosene and dodecane.

Uranium is charged into the organic phase mentioned above either by contacting the organic phase with a uranium-bearing aqueous acid solution of phosphate whose pH is lower than or equal to about 2 and preferably lower than or equal to about 1, or by solid-liquid extraction of a uranium-bearing phosphated compound, such as a phosphate ore, for example. The volume ratio of acid solution to the organic extractant solution is from about 1 to about 200, preferably from about 1 to about 50.

Before the application of step (a), it is advantageous to check to see if the concentration of water in the organic phase is sufficiently low, that is, if it is lower than one part per thousand by weight, preferably from about 92 to about 0.5 per thousand by weight. If this concentration is too high, it is desirable to dry the organic phase so as to lower the water concentration to below the concentration limit, either by centrifugation or any physical means for eliminating water suspended in the organic phase or by contacting the organic phase with one of the following dehydration agents: (no exhaustive list)

anhydrous calcium sulfate;
calcium chloride;
alumina;
silica gel;
molecular sieve.

The gaseous ammonia added in the aqueous phase in step (a) must be in a quantity at least equal to the quantity necessary for transforming the dialkyl pyrophosphoric acid in excess into its double salt of ammonium. The excess ammonia does not remain in the organic phase and is released into the atmosphere. It is preferred, therefore, to economize on this costly reagent, by limiting the quantity of ammonia introduced. A satisfactory application consists in choosing, as the quantity of ammonia added in the course of step (a), two to five moles of ammonia per mole of dialkyl pyrophosphoric acid.

The time duration of step (a) is advantageously set between about one half hour and about two hours, and preferably about one hour.

Step (b) may be carried out by filtration, cycloning, or centrifugation.

The present invention rests in part on the discovery that the colloidal and/or viscous nature of the precipitates obtained in the course of the reextraction of the uranium was due to the presence of excess dialkyl pyrophospheric acid, that is, not linked to a uranium atom in the organic phase.

The purpose for steps (a) and (b) is to eliminate the excess dialkyl pyrophosphoric acid in the organic phase and to recover it in a solid form. This dialkyl pyrophosphoric acid, in the form of an ammonium salt, is not subjected to the reextraction stage and may be directly recycled to the stage of treatment of the phosphated compounds to extract the uranium therefrom. Thus, an important part of the dialkyl pyrophosphoric acid is not subjected to conditions which would facilitate its hydrolysis.

Thus steps (a) and (b) permit substantial savings of reagent and this is the case even if the following stages of reextraction of the uranium facilitate the hydrolysis of the dialkyl pyrophosphoric acid.

The introduction of steps (a) and (b) into the above-described process of reextraction with hydrofluoric acid permits therefore a substantial saving of reagents.

The process for the recovery of uranium contained in phosphated solutions described in the above-mentioned French patent application includes the steps of: (1) contacting the uranium-bearing phosphated solution of the beginning step with an organic phase containing one diester of pyrophosphoric acid, and (2) contacting the uranium-charged organic phase obtained in (1) with an alkaline solution. Between these two steps, one may easily insert process steps (a) and (b) of the present invention. Applied to the earlier process, the present steps decrease notably the consumption of dialkyl pyrophosphoric acid and greatly facilitate the subsequent operations of recovery of the uranium, the actinides and

the rare earths, as well as the dialkyl pyrophosphoric acid entailed by the uranium.

Nevertheless, according to a preferred application of the present invention, the organic phase coming from step (b) is treated according to the following:

(c) contacting the liquid phase from step (b) with an aqueous phase (water) whose volume is between about 0.5 and about 10% of that of the organic phase, and preferably between about 1 and about 2%, then separating the organic phase from the aqueous and solid phases.

Taking into account the duration of the contact (from about 15 minutes to about an hour) and of the fact that no precaution is taken to avoid the oxidation of uranium IV into uranium VI, it has not been necessary until now to foresee the presence of an oxidation agent in the aqueous phase of step (c). Nevertheless, it is possible to add to this aqueous phase such an oxidation agent.

As the oxidation agent, one may use chlorine, hypochlorites, chlorates and, in a more general manner, all the oxidation agents whose normal apparent potential is higher than 300 millivolts compared to the hydrogen electrode. It is appropriate to point out however that it is preferable to use hydrogen peroxide, persulfates, as well as materials which produce hydrogen peroxide upon coming in contact with water as, for example, oxylith. The amount of the oxidizing agent in water ranges from about 1.5 QS to about 4 QS, preferably from about 1.5 QS to about 3 QS (QS stands for stochiometrical quantity for oxidizing uranium IV present in the organic phase in uranium VI).

According to a preferred embodiment of the invention, the process includes the following additional step:

(d) treating the aqueous and solid phases obtained in step (e) with alkalin solition, preferably ammonia solution.

Advantageously, this is carried out so that the pH obtained after the addition of ammonia is between about 8 and 10, and preferably between 9 and 10.

To attain these pH values and to obtain good solubilization of the dialkyl pyrophosphoric acid, it is preferable that the amount of ammonia added in step (d) be equal to a value between about one and four moles per gram of phosphorus contained initially in the aqueous and solid phases obtained in step (c), with the concentration of the ammonia used being advantageously between about 1 and 4 N, and, preferably, between about 1.5 and 2.5 N.

The treatment in step (d) lasts preferably from about 30 minutes to up to about two hours. During this treatment, the oxidation of the uranium IV into uranium VI continues if it was not complete after step (c). An oxidation agent may be added in the course of this step. Useful oxidizing agents include those mentionned above. The amount of the oxidizing agent added ranges from about 1.5 QS to about 4 QS, preferably from about 1.5 QS to about 3 QS. based on the total weight of the solid and liquid phases from step (c).

According to another preferred application of the invention, the process includes the following additional step:

(e) separating the liquid and solid phases present in the reaction mixture obtained in step (d).

Step (e) may be carried out by filtration, cycloning, or preferably centrifugation.

The liquid phase so obtained may be advantageously treated in the following manner:

(f) combining the liquid phase obtained in step (e) and the solid phase obtained in step (b), and thereafter acidifying the mixture thus obtained with a sufficient amount of a strong mineral acid so that the pH of the mixture is from about 2 to about-1, preferably from about 1 to about 0;

(g) extracting the reaction mixture from step (f) with at least a part of the organic phase coming from step (c) to obtain, on one hand, an organic phase charged with dialkyl pyrophosphoric acid and, on the other hand, an ammonium salt solution of the strong acid used in step (f).

In step (f), the strong mineral acid used may be one of the known strong mineral acids with the exception of those whose redox potential is sufficient to react with and oxidize the dialkyl pyrophosphoric acid. It is, therefore, preferred to avoid the use of nitric acid.

Because of the high commercial value of ammonium phosphates, the preferred strong mineral acid is phosphoric acid.

The solid phase obtained in step (d) and separated in step (e) is preferably treated in accordance with the following steps:

(h) redissolving the solid phase of step (e) with a strong mineral acid;

(i) contacting the reaction mixture of step (h) with an organic solution to reextract the dialkyl pyrophosphoric acid, the volume ratio of the mixture to the organic solution being from about 0.5 to about 4, preferably from about 0.5 to about 1.5;

(j) treating the residual aqueous phase to recover therefrom the iron as well as uranium, the actinides and the rare earths.

Preferably the organic solution applied in step (i) has a composition identical to that issuing from step (c) or may even be a fraction thereof. The fraction ranges from about 2 to about 10.

After re-extraction of the dialkyl pyrophosphoric acid in step (i) the organic phase is advantageously recycled to the extraction step at the beginning of the process.

The drawing provides a flow chart showing various stages in uranium separation, and combines many of the embodiments of the invention. The drawing shows all of the operations presented in the claims which follow. Further, "(U)" indicates the stream which contains the bulk of the uranium after certain of the operations.

The following examples, which are non-limiting, are provided to further illustrate the present invention.

The following examples are divided into two parts. The first part is for purposes of comparison whereas the second part exemplifies the present invention.

EXAMPLE 1

Reextraction of the uranium and iron from a uranium charged organic phase not containing octanol (3% OPPA+97% kerosene)

Kinetic study of the precipitation of the dialkyl pyrophosphoric acid, of uranium and of iron. Operative method:

Into an organic phase charged with uranium, ammonia is bubbled at a flow rate of 25 liters per hour. Specimens of the organic phase are removed for analysis after 5 minutes, 15 minutes, 30 minutes, and 60 minutes of reaction. At the time when the last specimen is taken, the reaction is stopped and the reaction mixture centri-

fuged for a period of 60 minutes at 2100 rpm. The centrifugation apparatus RUNNE is used
Results:

Composition of the organic phase			
Time (minutes)	U mg/l	Fe mg/l	P mg/l
0	830	193	3500
5	32	6	<200
15	29	7	<200
30	34	7	<200
60	50	9	<200
60 + centrifugation	140	26	<200

It is seen that after 5 minutes of bubbling, the majority of the uranium, iron, and phosphorous is in the precipitate. By increasing the duration of the reaction, the uranium and the iron already precipitated are partially redissolved. The phenomenon is even more marked after one hour of centrifugation.

EXAMPLE 2

Reextraction of uranium and iron from a uranium charged organic phase containing octanol (3% OPPA + 3% octanol + 94% kerosene)

Kinetic study of the selective precipitation of excess dialkyl pyrophosphoric acid.
Operative method:
Into an organic phase charged with uranium, ammonia is bubbled at a flow of 25 liters per hour. Specimens are removed from the organic phase for anlaysis after 5 minutes, 10 minutes, 15 minutes, 30 minutes, 60 minutes, and 90 minutes. At the same time as the last specimen is collected, the reaction is stopped and the reaction mixture centrifuged for a period of 60 minutes at 2100 rpm.
Results:

Composition of the organic phase			
Time (minutes)	U g/l	Fe mg/l	P g/l
0	1.12	220	3.50
5	1.13	215	2.32
10	1.13	205	1.80
15	1.13	205	1.68
30	1.17	215	1.66
60	1.13	210	1.64
90	1.08	215	1.61
90 + centrifugation	1.15	215	1.58

In contrast to the results of Example 1, the uranium and iron remain in solution while the phosphorus is precipitated in part. The concentration of the phosphorus in the organic phase initially decreases rapidly from 3.5 to 1.7 g/l after 15 minutes of reaction, and then, very slowly.

EXAMPLE 3

Behavior of the precipitate of Example 2

After 90 minutes of reextraction and 60 minutes of centrifugation, the following are collected:
organic phase: 740 ml
precipitate: 49 g
The precipitate is divided into two parts, one part being treated with water, and the other with 2 N ammonia solution.
3.1. Water and precipitate.
Operative conditions:
Precipitate: 24.5 g
H_2O: 100 ml
duration of agitation: 30 minutes at 30° C.
duration of centrifugation: 60 minutes
Results:

Composition of the precipitate							
Precipitate	Moist Weight g	Dry Weight g	Humidity %	U	Fe	P	Observations
Interface	1.70	0.10	94	ND	ND	ND	All the precipitate remains in the interface

ND: not determined.

Composition of the aqueous phase					
	Volume ml	U mg/l	Fe mg/l	P g/l	Observations
aqueous phase	105	253	16	7.70	aqueous phase, whitish pH = 8.5 (20° C.)

3.2 2N Ammonia solution and precipitate.

The operative conditions are identical to those of Example 3.1, but the water is replaced by 100 ml of ammonia solution.
Results:

Composition of the precipitate							
Precipitate	Moist Weight g	Dry Weight g	Humidity %	U	Fe	P	Observations
Interface	1.04	0.06	94	ND	ND	ND	All the precipitate remains in the interface

ND: not determined

Composition of the aqueous phase					
	Volume ml	U mg/l	Fe mg/l	P g/l	Observations
aqueous phase	105	125	13	7.15	aqueous phase, whitish pH = 10.3 (20° C.)

The precipitate is composed primarily of ammonium salts of OPPA which are soluble in water and in diluted ammonia solution.

EXAMPLE 4

Elution and precipitation of uranium contained in the organic phase after the treatment of Example 2

4.1. Influence of amounts of H_2O_2 and of H_2O on reextraction.
The organic phase is divided into four equal volumes which are submitted to a second reextraction under different conditions. Common operative conditions:
Organic phase: 185 ml
Flow rate: 5 l/h
duration: 1 hour
The amounts of hydrogen peroxide and of water used vary according to the experiments.

Composition of the organic phase		
Experiments	H_2O_2 introduced ml/l	H_2O introduced ml/l
1	1	0

-continued

2	3	0
3	1	5
4	1	10

Composition of the organic phase

Experiments	Volume Organic phase ml	U g/l	Fe mg/l	P g/l	Weight moist precipitate (g)
1	174	1.5	200	1.07	1.25
2	174	0.875	165	1.02	2.65
3	180	0.510	115	0.99	1.51
4	172	<0.005	<10	<0.2	5.01

In experiments 3 and 4, the precipitation takes place approximately 2 minutes after the beginning of the bubbling of the ammonia. But in experiment 3, redissolution of a large portion of the precipitates already formed is noticed as the reaction time is prolonged. The organic phase changes from yellow to colorless and then yellow again after one hour of reaction.

Conclusion:

These results show that hydrogen peroxide alone does not permit a complete reextraction of the uranium and iron contained in the organic phase. It is the amount of water introduced and the duration of the injection of ammonia that permits the uranium and iron to be totally precipitated. It is nevertheless necessary to determine the minimal amounts of water and ammonia needed for total reextraction. The use of an excess of one of these amounts could diminish the rate of reextraction. It also appears that the use of hydrogen peroxide to oxidize uranium IV into uranium VI at the time of the second reextraction is not necessary in view of the intense yellow color of the organic phase which is characteristic of uranium.

4.2. Behavior of various precipitates obtained at the time of treatment with water.

Operative conditions: precipitate of variable weight

H_2O: 37 ml (200 ml/l organic phase)

Duration of agitation: 30 minutes at 30° C.

Duration of centrifugation: 1 hour.

Results:

Composition of the aqueous phase

Experiment	Volume ml	U mg/l	Fe mg/l	P g/l
1	37	25	<5	0.17
2	37	32	<5	0.66
3	35	19	<5	0.41
4	33	1600	148	3.60

Composition of the precipitate

Experiment	Moist Weight g	Dry Weight g	Humidity %	U %	Fe %	P %
1	<0.1					
2	1.43	0.11	92			
3	1.10	0.08	93			
4	4.12	0.49	88	22.2	3.89	9.99

Observations:

All the aqueous phases obtained are cloudy, particularly that of experiment 4, which is milky. In experiments 1, 2, and 3, all the precipitates remain at the interface of the organic and aqueous phases. In experiment 4, the precipitate is found in the centrifugation residue but in all of the experiments traces of precipitate are found trapped in a gelatin bed at the interface of the organic and aqueous phases.

Conclusion:

It is observed that there is partial redissolution of uranium, iron and phosphorus after treating the precipitate with water. But the precipitates obtained are much richer in uranium (22.2% in experiment 4) and are soluble in normal sulfuric acid.

EXAMPLE 5

Continuous experiment

A complete continuous experiment applying steps (a) through (j) of the process according to the present invention is conducted with extraction of a uranium-bearing phosphated solution. The organic phase charged with dialkyl pyrophosphoric acid of step (g) is recycled to extraction of the uranium-bearing phosphated solution.

The summary shown in the table is based on a flow rate of 1 m^3/hr of the uranium charged organic phase.

The initial organic phase has the same composition as that of Example 2 (3% of OPPA, 3% octanol, 94% kerosene).

	Flow rate m^3/hr	Composition Uranium	Fe	Phosphorus	Concentration
Phosphated uranium-bearing solution	10 m^3				
Organic phase charged in uranium before step (a)	1 m^3	1.14 g/l	0.22 g/l	3.60 g/l	
Ammonia used in step (a) (gas)	5 m^3				
Organic phase recycled in step (i) to first extraction step	0.04m^3	6.5 g/l	1.5 g/l	4.2 g/l	
Precipitate coming from (b)	65 kg	0.12% wt	0.02% wt	3% wt	
Organic phase coming from (b)	0.95 m^3	1.12 g/l	0.22 g/l	0.61 g/l	
Organic phase coming from (c)	0.95 m^3	1.02 g/l	0.005 g/l	0.02 g/l	
Organic phase coming from (c) recycled to (g)	0.91 m^3	0.02 g/l	0.005 g/l	0.02 g/l	
Organic phase coming from (c) recycled to (i)	0.04 m^3	0.02 g/l	0.005 g/l	0.02 g/l	
Water added to step (c)	0.01 m^3				
Precipitate obtained at step (c)	0.01 m^3	1.04 kg	0.21 kg	1.59 kg	
Ammonia added in (d)	0.4 m^3				
Aqueous phase obtained at step (c)	0.4 m^3	0.160 g/l	0.02 g/l	3 g/l	
Strong acid used in step (f) (H_3PO_4)	0.1 m^3				
OPPA supply to compensate losses before recycl. toward uranium-bearing phos-					

-continued

	Flow rate m^3/hr	Composition Uranium	Fe	Phosphorus	Concentration
phated solution to be extracted	2.9% vol				
Solid phase obtained in (e):					
humid	30 kg				
dry	3 kg	35.2% wt	6% wt	8.9% wt	
Acid introduced in (h) (sulfuric acid)	0.06 m^3				1N
Aqueous phase coming from (i)	0.085m^3	9.3 g/l	1.6 g/l	1.2 g/l	

Conclusions:

This example shows that the percentage of OPPA which is precipitated selectively in the form of ammonium salt by the injection of gaseous ammonia is 54.2%.

The addition of water in step (c) causes the precipitation of 98.1 wt% of the uranium contained in the initial organic phase.

According to this experiment, the uranium is recovered in the form of "yellow-cake" with a return on the order of 94%.

It is also observed that at the time of the sulfuric acid dissolution, about 75% of the uranium is found again in the organic phase and about 25% in the aqueous phase.

URANIUM EXTRACTION FROM PHOSPHATE SOLUTION

It has been shown above that it was advisable to stabilize and dissolve the diester of the pyrophosphoric acid, or dialkyl pyrophosphoric acid, using dissolving and stabilising agents. These agents must be liposoluble components showing electronic doublets liable to produce hydrogen liaisons with the diesters of the pyrophosphoric acid. Among thoses components, one may quote the trialkyl phosphates as, for instance, the tributyl phosphate (hereafter often referred to as "TBP"), the trialkylphosphine oxides such as the trioctylphosphine oxide, and the heavy alcohols.

In the course of further investigations, it has been shown that the best suited among those agents were the heavy alcohols as defined above, i.e. alcohols with an alkyl radical that comprises 4 to 20 and preferably 6 to 12 atoms of carbon.

It has also been shown that these agents should preferably figure in a ratio of at least 0.5 mole per mole of dialkyl pyro phosphoric acid.

Nevertheless, the use of such agents generally entails an antisynergetic effect towards the extraction power of dialkyl pyrophosphoric acids, either directly or in so far as the stabilising agents tend to favour the extraction of the iron present in the phosphate solutions as compared with that of uranium. This adverse effect is a nuisance when extracting uranium out of phosphate solutions with an iron concentration of 1 to 30 grams per liter and particularly when the iron concentration exceeds 5 grams per liter.

This is why one of the further objectives of the invention is to provide a process that favours the extraction of uranium with regard to that of iron.

Another aim of the invention is to provide a process that wholly exhausts the uranium contained in the phosphate solutions.

These aims are achieved by means of the improvement characterized by an organic phase concentration in dialkyl pyrophosphoric acid comprised between 0.1 and 0.4, preferably 0.15–0.25 M.

It should be noted that, contrary to what had been disclosed earlier, the use of dialkyl pyrophosphoric acid at such concentration levels does not give rise to troubles at extraction stage, when bringing the phosphate solution and the organic phase into contact, and that if the contact is made with a ratio of the organic to the aqueous phase (O/A) ranging from 1 to 1/50, no emulsion problem occurs. If an O/A ratio lesser than 1/50 or greater than 1/200 is wanted, emulsion problems may be avoided by extracting at a temperature above 30° C., preferably between 35° and 45° C. Besides, emulsion problems are only to be encountered whenever the reduction of the phosphate solution was not complete.

The improvement is based on the surprising fact that the ratio of extracted uranium to extracted iron notably increases with the concentration of dialkyl pyrophosphoric acid in the organic phase, and that contrary to previous theoretical investigations, the ratio of extracted uranium to dioctyl pyrophosphoric acid increases with the latter. This inconsistency is hard to understand but could be ascribed to the fact that iron was largely present in the phosphate solutions treated by the applicant, whereas there was none in the earlier theoretical studies.

It should also be noted that the presence of dissolving and stabilising agents enhances the dissolution of dialkyl pyrophosphoric acid.

One of the advantages that may also be underlined when using a high concentration of dialkyl pyrophosphoric acid is that either the constraints pertaining to the reduction of the solution are eased or the uraniferous solution of 30% phosphoric acid % is completely exhaused with relatively few extraction stages.

One may state for example that using an organic phase that contained about 0.1 mole per liter octylpyrophosphoric acid and 0.1 mole of octylic alcohol, it was possible, in four stages to extract 99% of the uranium contained in a phophate solution at 30% phosphoric acid with 10 grams per liter of iron and a ratio of iron III to total iron not exceeding 2%, 100 milligrams per liter of uranium as well as the impurities mentioned on page 5 of the main patent. It is also possible to exhaust the uranium contained in an acid whereof ratio Iron III to total Iron is high (about 90%), but it necessitate to use concentrated organic phase, e.g. organic phase with about 10% OPPA.

The non-restrictive examples quoted hereunder should facilitate the specialist's assessment of the operating conditions required for each particular case. Operating directions for the examples 6 to 10:

The trials are carried out on a batch of industrial phosphoric acid with the following mean analysis:

Fluorine: 13.7 g/l (the fluor is mainly present as SiF_4).

Sulphates: 33.1 g/l

Calcium: 0.98 g/l

Aluminium: 3.2 g/l

P_2O_5: 329 g/l.

The uranium and iron content of the solution is indicated for each example.

The contacts are carried out as follows: the aqueous and organic phase are introduced in a setting ampule, the two phases are strenuously shaked for a minute. Then the phases are left to settle for 15 minutes after which they are separated and aliquots of each phase are titrated.

In the examples, the table "Exhaustion of aqueous phase" concerns the extraction of an aqueous phase brought into contact with several fresh organic phases successively, whereas the table "Upgrading of organic phase" refers to the upgrading of an organic phase by putting it into contact with a succession of fresh aqueous phases.

EXAMPLE 6

Organic phase composition:
di-octyl pyrophosphoric acid (OPPA): 3% in weight (0,074 M)
octanol-2:3% in weight
kerosen: 94%
Temperature: 40° C.

Exhaustion of the aqueous phase

	Organic phase		Aqueous phase		
O/A	Uranium mg/l	Iron mg/l	Uranium mg/l	Iron g/l	Fe^{2+} Fet
			125	12,70	0,96
1/50	1230	325	100	12,69	0,95
1/10	620	535	38	12,64	0,94
1/10	220	660	16	12,57	0,91
1/10	75	720	9	12,50	0,92
1/10	25	765	6	12,43	0,92
1/10	<5	810	6	12,34	0,91

Up-grading of the organic phase

	Organic phase		Aqueous phase		
O/A	Uranium mg/l	Iron mg/l	Uranium mg/l	Iron g/l	Fe^{2+} Fet
1/2	215	655	17	12,37	0,95
1/10	865	440	60	12,72	0,95
1/10	1240	355	87	12,71	0,95
1/10	1290	335	120	12,70	0,95
1/10	1340	295	120	12,70	0,95
1/10	1240	310	135	12,70	0,95

EXAMPLE 7

Organic phase composition:
di-octyl pyrophosphoric acid (OPPA): 3% in weight
octanol-2:3% in weight
kerosen: 94%
Temperature: 30° C.

Exhausting of the aqueous phase

	Organic phase		Aqueous phase		
O/A	Uranium mg/l	Fer mg/l	Uranium mg/l	Fer g/l	Fe^{2+} Fet
			127	13,00	0,98
1/50	1310	370	101	12,99	0,97
1/10	660	505	35	12,94	0,97
1/10	230	640	12	12,88	0,96
1/10	65	720	5	12,80	0,96
1/10	20	760	3	12,73	0,96
1/10	<5	810	3	12,65	0,96

Up-grading of the organic phase

	Organic phase		Organic phase		
O/A	Uranium mg/l	Iron mg/l	Uranium mg/l	Iron g/l	Fe^{2+} Fet
1/2	220	625	17	12,69	0,98
1/10	540	395	65	13,02	0,97
1/10	985	320	112	13,01	0,97
1/10	1170	275	109	13,00	0,97
1/10	1150	270	129	13,00	0,97
1/10	1140	260	128	13,00	0,97

EXAMPLE 8

Organic phase composition:
di-octyl pyrophosphoric acid (OPPA): 3% in weight
octanol-2: 0%
kerosen: 97%
Temperature: 30° C.

Exhaustion of the aqueous phase

	Organic phase		Aqueous phase		
O/A	Uranium mg/l	Iron mg/l	Uranium mg/l	Iron g/l	Fe^{2+} Fet
			126	13,60	0,97
1/50	1670	205	93	13,60	0,97
1/10	720	525	21	13,54	0,95
1/10	130	780	8	13,47	0,95
1/10	20	860	6	13,38	0,96
1/10	<5	915	5	13,29	0,96
1/10	<5	940	5	13,19	0,96

Up-grading of the organic phase

	Organic phase		Aqueous phase		
O/A	Uranium mg/l	Iron mg/l	Uranium mg/l	Iron g/l	Fe^{2+} Fet
1/2	235	775	8	13,21	0.97
1/10	1070	430	42	13,63	0,97
1/10	1540	315	79	13,61	0.96
1/10	1670	260	113	13,61	0,96
1/10	1770	240	116	13,60	0.96
1/10	1680	235	135	13,60	0,96

EXAMPLE 9

Organic phase composition:
di-octyl pyrophosphoric acid (OPPA): 6% in weight
octanol-2: 3% in weight
kerosen: 91%
Temperature: 30° C.

Exhaustion of the aqueous phase

	Organic phase		Aqueous phase		
O/A	Uranium mg/l	Iron mg/l	Uranium mg/l	Iron g/l	Fe^{2+} %
			112	10,20	1
1/50	3010	425	52	10,19	1
1/10	465	1070	5	10,08	1
1/10	40	1380	1	9,94	1
1/10	<5	1420	0	9,80	1
1/10	<5	1470	0	9,70	1
1/10	<5	1490	0	9,50	1

Up-grading of the organic phase

	Organic phase		Aqueous phase		
O/A	Uranium mg/l	Iron mg/l	Uranium mg/l	Iron g/l	Fe^{2+} %
1/2	205	1170	9	9,61	1
1/10	1100	905	22	10,23	0,98
1/10	1950	705	27	10,22	0,97
1/10	2580	565	49	10,21	0,96
1/10	3010	495	69	10,21	0.95
1/10	3380	445	75	10,21	0,95

EXAMPLE 10

Extraction tests were carried out with extracting organic phase of various composition and with various ratios Iron II/Total Iron in the aqueous phase. The 30% phosphoric acid used presents the same composition as for examples 6 to 9. With:

Uranium: 120 mg/l
Iron: 12,8 g/l
The ratio O/A is always 1/50.
The diluant is kerogen.

Composition of the loaded organic phase			Iron	
OPPA WE %	Octanol-2 WE %	Uranium g/l	Iron Total %	Temperature
6	0	1,32	10	30° C.
6	3	1,12	10	30° C.
12	0	2,44 10	30° C.	
12	3	1,88	10	30° C.
12	3	1,76	10	45° C.
12	3	2,41	50	30° C.
12	3	2,15	50	45° C.
6	0	2,7	98	30° C.
6	3	2,3	98	30° C.
12	0	3,97	98	30° C.
12	3	3,40	98	30° C.
12	3	3,11	98	45° C.

In conclusion, this example shows clearly that it is possible to completely extract the uranium present in 30% phosphoric acid even not reduced when using organic phase with high concentration in dialkyl pyrophosphoric acid.

It is clear from this example that the extraction rate decreases when the Iron III concentration increases and that it is possible to overcome this drawback by increasing the dialkyl pyrophosphoric acid concentration.

The high concentration of the pyrophosphoric acid may entail difficulties for reextraction. These difficulties may easily be overcomed by diluting organic phase before stripping.

What is claimed is:

1. A process for recovering uranium from a uranium-containing acid lixiviation solution comprising: extracting the lixiviation solution with an organic extractant solution comprising a dialkyl pyrophosphoric acid, a diluent, and at least 0.5 mole of a solubilization agent for the double dialkyl pyrophosphate of uranium and ammonium which is present at any point during the process per mole of dialkyl pyrophosphoric acid, and treating the resulting mixture of solutions as follows:

(a) bubbling anhydrous ammonia gas into the loaded organic solution to precipitate the ammonium salt of the excess dialkyl pyrophosphoric acid, and

(b) separating the precipitate of step (a) from the uranium-containing liquid phase and recovering uranium from the liquid phase.

2. The process of claim **1** wherein the organic solution comprises from about 2 to about 5 moles of the solubilization agent per mole of dialkyl pyrophosphoric acid.

3. The process of claim **1** wherein the organic solution comprises from about 1 to about 10% by weight of dialkyl pyrophosphoric acid, from about 2 to about 5 moles of the solubilization agent per mole of dialkyl phosphoric acid, and the balance being the diluent.

4. The process of claim **3** wherein the dialkyl pyrophsophoric acid is dioctyl pyrophosphoric acid, the stabiliation agent is octanol-2, and the diluent is kerosene.

5. The process of claim **1** wherein prior to the application of ammonia in step (a), the mixture of solution is dried by contacting the mixture with a dehydration agent selected from the group consisting of anhydrous calcium sulfate, calcium chloride, alumina, silica gel, and molecular sieve, so that the water content of the mixture is less than 1 part per thousand by weight.

6. The process of claim **1** wherein the amount of ammonia added in step (a) is at least equal to the quantity necessary for transforming the excess dialkyl pyrophosphoric acid into its double ammonium salt.

7. The process according to claims **1, 2, 3, 4,** or **5** wherein the amount of ammonia added is from about 2 to about 5 moles per mole of excess dialkyl pyrophosphoric acid.

8. The process of claim **1** wherein the extraction and steps (a) and (b) are conducted at a pressure of about 1 atmosphere and a temperature of not higher than about 50° C.

9. The process of claim **1** wherein step (b) is performed by filtration, cycloning, or centrifugation.

10. A process of recovering uranium from an aqueous uranium-containing acid lixiviation solution comprising: extracting the solution with an organic extractant solution comprising a diluent, a dialkyl pyrophosphoric acid, and at least one mole of a solubilization agent for both of the dialkyl pyrophosphorate of uranium and ammonium which is present at any point during the process per mole of dialkyl pyrophosphoric acid, and treating the resulting mixture of solutions as follows:

(a) bubbling into the mixture of solutions anhydrous ammonia gas to precipitate the ammonium salt of the excess dialkyl pyrophosphoric acid;

(b) separating the precipitate of step (a) from the uranium-containing liquid phase;

(c) contacting the liquid phase of step (b) with water, the volume of which is from about 0.5 to about 10% of that of the liquid phase from step (b) for from about 15 minutes to about 1 hour, and separating the organic phase from the aqueous and solid phases;

(d) treating the aqueous and solid phase from step (c) with ammonia until the pH of the solid-liquid phases is from about 8 to about 10;

(e) separating the aqueous liquid and solid phases obtained in step (d);

(f) combining the liquid phase from step (e) and the solid phase obtained in step (b) and acidifying with a strong mineral acid selected from the group consisting of sulfuric acid and phosphoric acid so that the pH of the resulting mixture is from about −1 to about 2;

(g) extracting the mixture of step (f) with a portion of the organic phase obtained in step (c) to form an organic phase charged with dialkyl pyrophosphoric acid and an ammonium salt solution of the acid used in step (f);

(h) redissolving the precipitate obtained in step (e) with a strong mineral acid selected from the group consisting of sulfuric acid and phosphoric acid;

(i) contacting the resulting mixture of (h) with an organic solution to reextract the dialkyl pyrophosphoric acid, the organic solution having a composition identical to that obtained in step (c) and the volume ratio of the mixture to the organic solution being from about 0.5 to about 4;

(j) treating the aqueous phase from step (i) to recover therefrom uranium, iron, the actinides and rare earth metals.

11. The process of claim **10** wherein prior to step (a), the mixture of solution is contacted with a dehydration agent selected from the group consisting of anhydrous calcium sulfate, calcium chloride, alumina, silica gel, and molecular sieve, so that the water content in the mixture is lower than 1 per 1000 by weight.

12. The process of claim **10** wherein in step (a), the amount of ammonia bubbled into the mixture of solution is at least equal to the quantity necessary for transforming the excess dialkyl pyrophosphoric acid into its double ammonium salt.

13. The process of claim **12** wherein in step (a), the amount of ammonia added is from about 2 moles to about 5 moles per mole of dialkyl pyrophosphoric acid.

14. The process of claim **10** wherein the organic extractant solution comprises from about 2 to about 5 moles of the solubilization agent per mole of dialkyl pyrophosphoric acid.

15. The process of claim **10** wherein the organic solution comprises from about 1 to about 10% by weight of dialkyl pyrophosphoric acid, from about 2 to about 5 moles of the solubilization agent per mole of dialkyl phosphoric acid, and the balance being the diluent.

16. The process of claim **15** wherein the dialkyl pyrophosphoric acid is dioctyl pyrophosphoric acid, the stabilization agent is octanol-2, and the diluent is kerosene.

17. The process of claim **10** wherein in step (c) the liquid phase of step (b) is contacted with water and an oxidizing agent, the amount of oxidizing agent in the water ranging from about 1.5 QS to about 4 QS.

18. The process of claim **17** wherein in step (d), an oxidizing agent is added to the solid and liquid phases, the amount of oxidizing agent added being from about 1.5 QS to about 4 QS.

19. The process of claim **10** wherein a fraction of the organic phase from step (c) is fed to step (i).

20. The process of claim **10** wherein the organic phase containing dialkyl pyrophosphoric acid obtained in step (i) is fed to the organic extractant solution for extracting the lixiviation solution.

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Behaviour of Uranium During Phosphate Ore Calcination

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During industrial calcination of phosphate rock, uranium is mobilised with changes both in location and oxidation state. Fission track micromapping of uranium under the microscope has been employed to study the precise distribution of uranium within the samples. Oxidation state ratios of U(IV) to U(VI) were determined by chemical separation followed by delayed neutron activation. The calcination process was studied both in the laboratory and in a full-scale production kiln of the Oron plant (Israel). The phosphate ore contains about 100 parts of 10^{-6} of uranium, and U(IV) comprises 35–40% of the total U present. The uranium which was not extractable in weak acids, was evenly distributed in the apatite rock components. The study revealed that the following changes occur during the calcination process: (a) carbonate-fluorapatite loses CO_2, and recrystallises to fluorapatite. The recrystallisation process intensifies with increasing temperature. (b) Around 600°C all the uranium is oxidised to the hexavalent state. (c) Migration of uranium in the apatite fragments initiates at 800°C. On increase of temperature to around 900°C, it forms uranium-rich phases in which it reaches 1–2%. The uranium-rich phases exhibit different solubilities in dilute acids, in some cases enabling its preferential leaching from the rock. Other U-rich phases are extremely insoluble (possibly fluorite, Ca-silicates) even in phosphoric acid, lowering extraction efficiency. Extraction yields can be improved by addition of salts (e.g. Na_2CO_3) which prevent the formation of insoluble U-rich phases.

1. Introduction

The rapid increase in uranium oxide prices together with the declining life span of known high-grade uranium reserves is again stimulating interest in alternative sources of uranium. One such source is sedimentary phosphorite (or phosphate rock) which typically contains 30–260 parts 10^{-6} U (average of 120).[1] Extraction of uranium from phosphate rock during its conversion into a soluble phosphate fertiliser is desirable. With present technologies uranium can, and is being recovered during wet-process phosphoric acid production. Six such recovery plants were operational in 1980 in the US alone, and the projected production capacity of these plus three additional plants which were in final stages of construction was 4 385 000 lb of U_3O_8 per year in 1981.[2]

So far, no process has been developed to selectively extract uranium from phosphate rock without completely dissolving the rock. Attempts at such selective extraction showed[3] that the U/P_2O_5 ratio in the leached phase does not differ significantly from the same ratio in the rock. It was, however, shown by several studies[4,5] that preferential leaching of U is feasible from calcined phosphate rock. Such a process might be very attractive in view of the fact that in several phosphate rock plants benefication is achieved by calcination.[6] Among these are plants in Poland, India, Egypt, Jordan and Israel.

The present work aimed at studying the behaviour of uranium during calcination, with the Oron Plant in the Israeli northern Negev serving as a case study.

The major method used was a combination of fission track mapping, determination of uranium oxidation states and leaching experiments of calcined phosphorites. This combination established

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if, and how, uranium was mobilised during the rock calcination, as well as measured the amount of uranium which could be leached from the rock either directly after calcination, or by adding some additives (NaCl, Na_2CO_3) before calcination. The determination of uranium oxidation states is crucial in this respect in view of the generally much higher solubility of all U(VI) compounds compared with U(IV), and the sharp difference in recovery strategies for tetravalent and hexavalent uranium.

The Oron phosphate field is located 30 km SE of Beer Sheva. The main phosphorite is of Campanian age, and has an average P_2O_5 content of 24%. It is enriched according to the flow sheet represented in Figure 1, to produce an end product with about 32% P_2O_5. The annual production of the plant in 1979 was 500 000 t of calcined calcium phosphate. This amount contains about 50 t of U_3O_8.

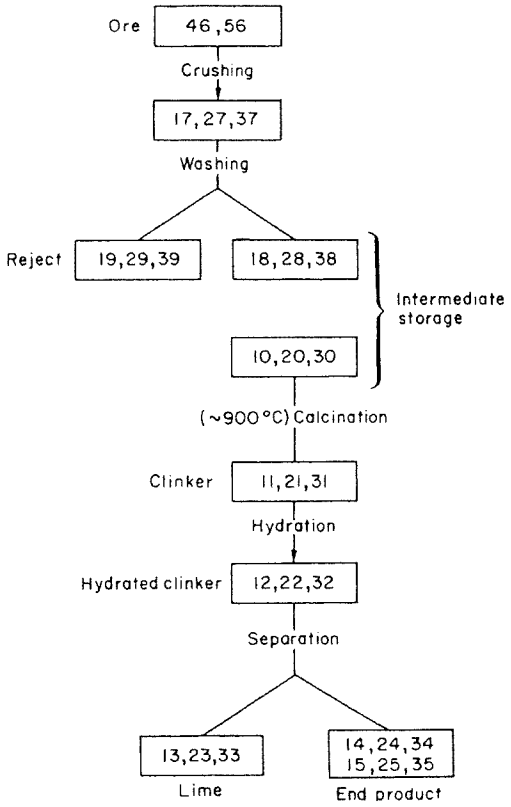

Figure 1. Flow sheet of phosphate rock calcination in Oron Plant. Numbers in rectangles designate sample numbers (see Tables). Note that 'intermediate storage' disrupts sampling continuity.

2. Experimental

Samples were taken from unprocessed Oron phosphorites and from different stages of the Oron benefication plant. Three series were sampled on different days from the various plant stages.

All samples (2 kg originally) were homogenised by splitting, a 1 kg subsample was removed and five subsamples were taken for (a) measuring total uranium, (b) the U(IV) percentage, (c) U fission track mapping, (d) X-ray fluorescence chemical analysis of major components (XRF), (e) X-ray diffraction mineralogical analysis (XRD) and (f) thermal analyses.

2.1. Uranium concentration

Uranium was determined at the Soreq Nuclear Centre by delayed-neutron measurement after irradiation of powdered samples in the reactor.[7]

2.2. U(IV) determination

Tetravalent uranium was precipitated as cupferrate as described by Clarke and Altschuler.[8] The rock was dissolved in cold phosphoric acid or a cold hydroxylamine hydrochloride solution,[8] a titanium carrier was added to the solution, the U(IV) cupferrate precipitate filtered on a Millipore (0.45) filter, and introduced into a small polyethylene bag which was inserted into a polyethylene vial. Thus U in the cupferrate was again determined by delayed neutrons identically with the total U determination; U(VI) was then determined by difference. To check that no insoluble uranium phases were lost, filter papers, through which the original phosphoric acid solutions were passed, were also treated as samples for delayed-neutron determinations. It was found that samples which were heated to more than 800°C could not be completely dissolved in cold phosphoric acid and up to 80% of the total uranium was left as an insoluble residue on the filter paper. Complete sample dissolution was achieved by dissolving the same samples in 1.2M-$NH_2OH.HCl$. Hydroxylamine hydrochloride does not oxidise uranium as proven by analysing standard rocks.[8] To determine the oxidation state of the phosphoric acid insoluble uranium, U(IV) was precipitated as cupferrate from both the phosphoric acid, and the $NH_2OH.HCl$ solutions (Table 1). Since the U(IV) value does not depend on the dissolution method, we conclude that the uranium which was detected on the filter paper is hexavalent. (Throughout this paper reference is made to the concentration of U in the rock which was found in the fraction insoluble in phosphoric acid as U_x.)

Table 1. U(IV) concentration as determined by two dissolution procedures

	U (parts 10^{-6})	
Sample	$NH_2OH.HCl$	H_3PO_4
NBL-1	168 (162)[a]	161
KP-65	137 (149)	143
30	37	44
31	10	5
32	5	10
34	4	7

[a] Values in parentheses are from Kolodny[9] and Burnett.[10]

Accuracy and precision in uranium analysis were checked by repeatedly processing two standards through our analytical procedures. These were 'Phosphate Rock No. 1' supplied by the New Brunswick Laboratories (marked NBL 1) which was analysed previously[9,10] and sample KP65.[11] The average analyses obtained here are within 7% of the 'best accepted value' for both standards. Analyses of duplicate samples and their comparison with analyses by Y. Avital, A. Starinsky and Y. Kolodny (unpublished work) indicate a lower precision than that determined from standards: it is ±10% for total U determination and ±20% for U(IV) measurements. This is probably due to inhomogeneity of samples in comparison with standards.

2.3. U-fission track mapping (f.t.m.)

Micro-maps of uranium distribution were prepared by inducing ^{235}U fission by thermal neutrons and recording fission particles on a solid.[12] The procedure included impregnation of a sample in polystyrene and preparation of a petrographic thin section from the hardened sample. This thin section served also for textural analysis of the sample. A mica (muscovite) sheet was then attached to the sample and the assemblage irradiated by a thermal neutron dose of about 1.5×10^{14} neutrons

cm^{-2}. The dose was monitored with gold monitors. After irradiation the mica was separated from the thin section, etched for 30 mins in 48% HF at room temperature and dried. The thin section and its f.t.m. were then examined in parallel under two microscopes. To help identify location on the f.t.m. thin masking-tape markers were attached to the thin sections, which could be recognised as blank markers on the map.

U concentration was estimated from f.t.m. by comparison with a standard U-bearing glass [NBS standard containing 461.5 parts 10^{-6} U, with 0.2376% ^{235}U (instead of the natural 0.7256%)]. Concentration was estimated from

$$Cx(U) = \left(\frac{Is}{Ix}\right)\left(\frac{Px}{Ps}\right) Cs(U) \tag{1}$$

where $Cx(U)$ and $Cs(U)$ are uranium concentrations in the unknown and in the standard correspondingly, Is and Ix the ratio $^{235}U/^{238}U$ in standard and sample and Px, Ps the number of fission tracks per unit area in sample and standard.

Substituting the known values in (1)

$$Cx(U) = 0.3274\left(\frac{Px}{Ps}\right) 461.5 = 151\,\frac{Px}{Ps} \tag{2}$$

Thus U concentration in a morphological-petrographic species (e.g. ovulite) is determined by the density of fission tracks in it. To correct for geometrical differences between standard and samples, uranium concentrations were determined on rocks by both delayed neutrons and fission track counting. The factor F was determined as

$$F = \frac{C_{dn}}{C_{ft}} \tag{3}$$

where C_{dn} is the concentration of U as determined by delayed neutrons and C_{ft} the same concentration by f.t. counting. C_{ft} estimation included thus both f.t. counting and an estimate of the abundance of petrographic species. The value $C_{ft}' = C_{ft}.F$ was then comparable with values obtained by delayed neutrons.

2.4. XRF analyses

Analyses of P_2O_5 were carried out on an ARL 72000 by D. Isaskhari at Oron Phosphate Works.

2.5. XRD

Mineralogical changes were checked by X-ray diffractograms. Specifically CO_2 content in apatite was measured by recording the 20 differences between the (410) and the (004) reflections.[13] The CO_2 concentrations measured by this method are in error by less than $\pm 0.5\%$ CO_2.

2.6. Thermal analysis

Differential thermal analysis (DTA) and thermo-gravimetric analysis (TGA) were performed on selected samples heated to different temperatures.

2.7. Laboratory experiments

A simulation of calcination processes was carried out by: (1) heating samples for 1 h from 200 to 1000°C at steps of every 100°C; (2) Na_2CO_3 and NaCl were added as calcination additives to check their influence on U behaviour. These samples were then heated to 800 and 900°C.

Samples were leached by dilute acetic acid to check U mobilisation as a result of calcination. Selected samples were examined under the scanning electron microscope.

3. Results and discussion

3.1. Mineralogical and textural changes during calcination

The Oron phosphorite has been described before.[14–16] It consists of apatitic fragments and mainly calcitic matrix; accompanying minerals are dolomite, gypsum, quartz and clays. Apatite is car-

bonate-fluorapatite (francolite), and appears mainly as ovulites (0.15–0.35 mm in diameter, Figure 2A); these are brownish oval bodies, isotropic under crossed polarisers. Larger particles (0.5–2 mm) are referred to as coprolites. Bone fragments constitute only a small fraction (<1%) of the Oron rock. The rock also contains some carbonate clasts. The matrix is calcitic apatitic and foraminifera are often found in it.

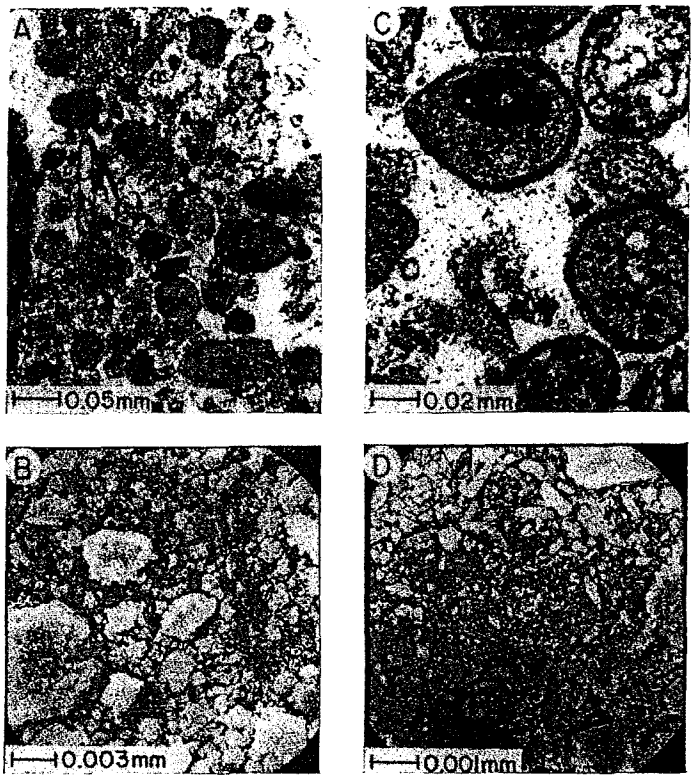

Figure 2. (A) A mineralogical thin section of the Oron phosphorite ore. The matrix is mainly calcite. (B) Scanning electron microscope (SEM) photograph of ovulites before calcination ('cauliflower' texture). (C) A mineralogical thin section of the Oron clinker. The intergranular material is an impregnating hardener. Note sintering envelope and collapsed ovulites. (D) SEM photograph of ovulites after calcination. Note prismatic apatite crystallites.

As observed previously,[6,17] the major changes which occur during phosphorite calcination are both mineralogical–chemical and textural. These include the decomposition of organic matter, water removal, decomposition of carbonates, evolution of a part of the fluorine as well as recrystallisation of apatite and some crystallisation of calcium silicate phases.

A major observable change is that of colour: samples turn greenish instead of the pale brown raw material. In the clinker, ovulites become more concentrated, the rock attains a more homogeneous appearance, which is preserved in the final marketable product (Figure 2C). Calcination in the laboratory produced identical textural changes with those in the industrial kiln. In samples heated above 700°C the isotropic ovulites began to show first-order low birefringence under the microscope; probably reflecting incipient grain growth. The apatite recrystallisation can be well documented by following it under the scanning electron microscope. Whereas the raw material ovulites have a characteristic 'cauliflower' texture under the microscope (Figure 2B), growth of small (<4 μm) prismatic apatite crystals is recorded after heating to 800°C. These crystals increase both in number and size when samples are consecutively heated to 900°C (Figure 2D). These are probably changes parallel to those described before.[18–20] During calcination the recrystallisation of apatite is accompanied by the decomposition of francolite and by the formation of fluorapatite, probably following the reaction[20,21]

$$Ca_{5-0.5x}(PO_4)_{3-x}CO_{3_x}F_yF_2 \rightleftharpoons Ca_5(PO_4)_3F_2 + xCaO + xCO_2 + yF$$

0.15–0.35 mm in diameter, Figure
isers. Larger particles (0.5–2 mm)
mall fraction (<1%) of the Oron
; calcitic apatitic and foraminifera

Figure 2. (A) A mineralogical thin section of the Oron phosphorite ore. The matrix is mainly calcite. (B) Scanning electron microscope (SEM) photograph of ovulites before calcination ('cauliflower' texture). (C) A mineralogical thin section of the Oron clinker. The intergranular material is an impregnating hardener. Note sintering envelope and collapsed ovulites. (D) SEM photograph of ovulites after calcination. Note prismatic apatite crystallites.

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This process is demonstrated by the sharpening of apatite peaks (improved crystallinity) on diffractograms with increased heating and by the gradual decarbonation of apatite. Whereas the ore contains 4.6% CO_2, 60–70% of that quantity left the lattice after heating to 700°C for 1 h, and another 20–30% after heating for the same duration to 900°C or after Oron kiln calcination (Figure 3, Table 2). The details of the calcination process were followed by XRD and DTA. The major CO_2 loss occurs above 600°C. Most of the calcite is being decomposed around 800°C, whereas the bulk of the organic matter if present in the rock is removed at 300–400°C.

At temperatures around 750°C, calcium silicate phases begin to form. The major chemical change during calcination is that which is the process's purpose: an increase in P_2O_5 content from 24–25% in the ore, to 27–28% after washing to 32% in the end product.

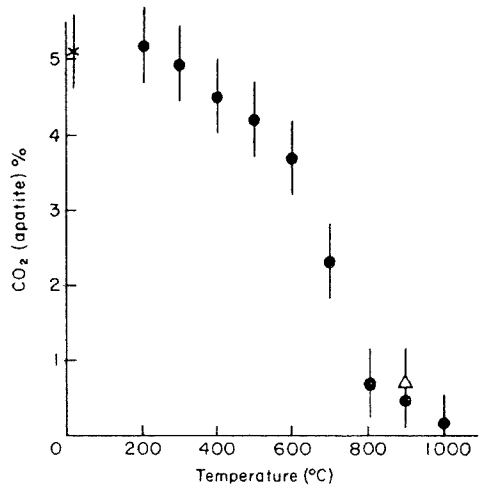

Figure 3. Relation between CO_2 content in apatite and temperature of heating (duration of heating 1 h). ●, Experimental; △, clinker; ×, ore.

Table 2. CO_2 content in consecutively heated apatites

Temperature (°C)	Sample no.	2θ	%CO_2
Ore	38	1.26	5.1
200	331	1.25	5.2
300	332	1.27	4.9
400	333	1.30	4.5
500	334	1.32	4.2
600	335	1.35	3.7
700	302	1.45	2.3
800	303	1.56	0.8
900	304	1.57	0.5
1000	305	1.59	0.2
Clinker	31	1.56	0.7

3.2. Uranium behaviour during calcination

Whereas untreated phosphate ore contains 80–120 parts 10^{-6} uranium (Table 3) the Oron Plant end products contain on average 100–140 parts 10^{-6} U. This increase probably reflects the effective decarbonation during calcination (both $CaCO_3$ decomposition and francolite decarbonation). The constancy of the U/P_2O_5 ratio throughout the calcination process at about $3–4 \times 10^{-4}$ (Table 3, Figure 4) seems to support the above conclusion.

Table 3. P_2O_5, U and U(IV) concentrations

Run	Sample no.	Description	U[a] (parts 10^{-6})	U(IV) (parts 10^{-6})	P_2O_5 (%)
	46	Ore	109 (2)	33 (2)	26.5
	56	Ore	113 (2)	34 (2)	27.1
1	17	Crushed	82 (2)	38 (2)	25.7
	18	Washed	89 (2)	44 (2)	28.9
	19	Washed	91 (2)	26	n.d.
	10	Washed	92 (2)	54 (2)	28.3
	11	Clinker	111 (4)	5.9 (3)	30.8
	12	Hydrated clinker	100 (3)	4.1 (3)	30.0
	14	End product	109 (3)	4.9 (3)	30.1
	15	End product	104 (3)	7.2 (3)	30.9
2	27	Crushed	89 (2)	29 (2)	24.7
	28	Washed	89	40	29.0
	29	Washed	72 (2)	—	—
	20	Washed	106 (3)	40 (2)	23.4
	22	Hydrated clinker	123 (3)	15 (2)	31.8
	23	Lime	99 (2)	4	25.3
	24	End product	125 (3)	7.6 (3)	32.2
	25	End product	129 (2)	7.5 (2)	32.8
3	37	Crushed	110 (2)	33 (2)	25.9
	38	Washed	111	38	29.8
	39	Washed	109 (2)	36 (2)	31.4
	30	Washed	122 (2)	44 (2)	24.2
	31	Clinker	138 (3)	4.5 (2)	31.5
	32	Hydrated clinker	130 (2)	9.8 (2)	31.4
	33	Lime	129 (3)	4.5 (2)	29.4
	34	End product	126 (2)	7.4 (2)	32.7
	35	End product	132 (2)	—	32.2

[a] Values in parentheses indicate the number of replicates.

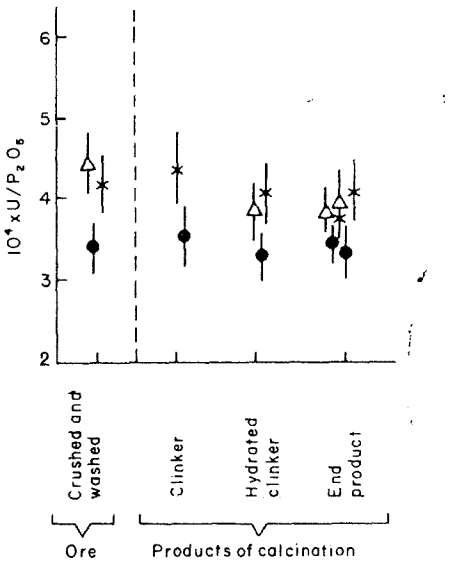

Figure 4. Variation of the U/P_2O_5 ratio during the calcination process. Runs: ●, 1; △, 2; ×, 3.

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U(IV) (parts 10^{-6})	P_2O_5 (%)
33 (2)	26.5
34 (2)	27.1
38 (2)	25.7
44 (2)	28.9
26	n.d.
54 (2)	28.3
5.9 (3)	30.8
4.1 (3)	30.0
4.9 (3)	30.1
7.2 (3)	30.9
29 (2)	24.7
40	29.0
—	—
40 (2)	23.4
15 (2)	31.8
4	25.3
7.6 (3)	32.2
7.5 (2)	32.8
33 (2)	25.9
38	29.8
36 (2)	31.4
44 (2)	24.2
4.5 (2)	31.5
9.8 (2)	31.4
4.5 (2)	29.4
7.4 (2)	32.7
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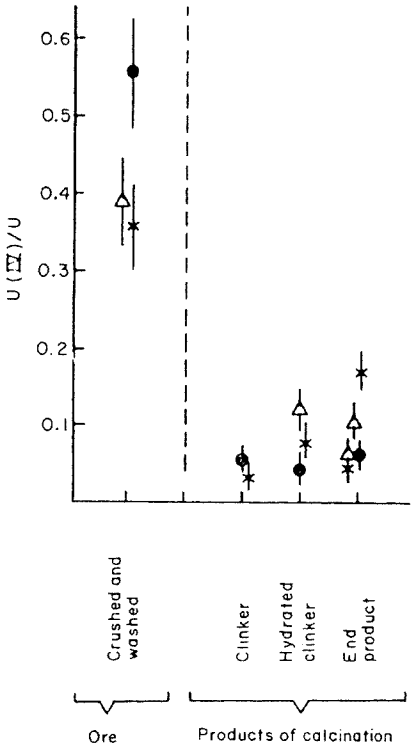


Figure 5. Change in U oxidation state along the production process. Runs: ●, 1; △, 2; ×, 3.

Figure 5 represents the change in U oxidation state along the production process in the Oron Plant. Whereas the untreated ore contains about 60% hexavalent uranium, the remaining part being tetravalent, practically all the uranium has been oxidised in the end product. Hence, the kiln is an oxidising, not a reducing, environment. A 'reducing kiln' has previously been suggested (Y. Folkman, personal communication).

To follow more closely the uranium oxidation process, a set of laboratory heating experiments were carried out (Table 4). Figure 6 summarises these experiments and shows that the major oxidation step occurs around 600°C, a range close to that where the major CO_2 loss from apatite occurred.

As mentioned before, during calcination some uranium enters a phase which is insoluble in phosphoric acid but is well dissolved in hydrochloric acid. The amount of tetravalent uranium U(IV) remained the same irrespective on the method of rock dissolution. Hence the insoluble uranium-bearing phase formed at high temperatures contains hexavalent, not tetravalent uranium. This process too was followed in laboratory heating experiments.

Figure 7 shows the change of the U_x (uranium-insoluble in phosphoric acid) with temperature. It indicates the appearance of an insoluble uranium-bearing phase above 800°C. A similar phenomenon was observed in the Oron kiln operating at 900°C.

3.3. Localisation of uranium

Migration of uranium within the phosphorite during calcination can be traced by means of sets of consecutive f.t. maps. In untreated phosphorite uranium is homogeneously distributed within each apatitic rock component (Figure 8A).

Differences exist between apatitic components of varying nature (ovulites versus bone fragments). The ovulites have about twice the U concentration as bones. With progressive heating uranium

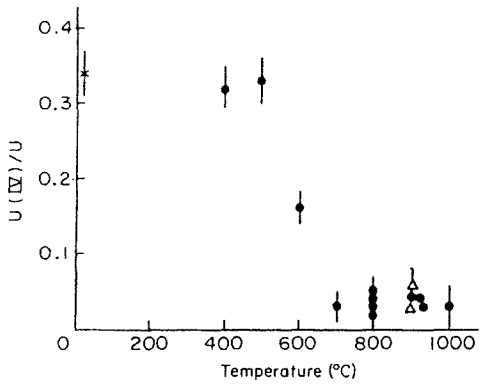


Figure 6. Change in U oxidation state during laboratory heating experiments. ●, Experimental; △, clinker; × ore.

Table 4. U, U(IV) and U/P_2O_5 in the samples before and after extraction

Temp. (°C)	Sample no.	Before extraction (parts 10^{-6}) U	U(IV)	U_x	U/P_2O_5	After extraction (parts 10^{-6}) U[a]	U(IV)[a]	U/P_2O_5	Extracted U%
Room	38	111	38	3		105	40		4
200	331	117				108			7
300	332	116				108			7
400	333	113	36			106			6
500	334	115	38			110			4
600	335	121	19	14		118			3
	78	157	9	6.8		145			8
700	372	111	3	4.5		38			66
	302	126	4	8.7		44	2		65
800	373	121	4	4.6	4.3×10^{-4}	44	5	1.2×10^{-4}	68
	303	137	5	5.5		48	2		65
	303A[b]	128	6	3.6		34	3		74
	383B[b]	116	2	3.5		25			78
900	374	123	6	40.4		76	2		38
	304	130	5	43.3	4.1×10^{-4}	83	3	2.3×10^{-4}	41
	304A[b]	135	5	12.5		21			85
	384B[b]	119	3	27.7		28			76
1000	375	114	3	47.0		103	10		11
	305	118	3	87.8		102	9		14
Kiln	31	138	4	72.1		94	4		34
	34	126	7	62					
~900	35	132	8	61		89	4		32

[a] Normalised to the original untreated sample weight.
[b] A and B designated additives of Na_2CO_3 and NaCl respectively.

begins to migrate and forms first, above 700°C, 'dense track concentrations' (Figure 8B) which become sharper at about 800°C. Between 800 and 900°C a radical change occurs in the U distribution leading to the formation of 'track-stars', which become even more impressive at about 900°C (Figures 8C, D). The relocation of uranium is probably correlated with the strong recrystallisation occurring in the temperature range of 700–1000°C, during which new apatite crystals were observed under the scanning electron microscope. Repeated examination and microprobe analysis showed, however, that the 'track star' uranium concentrations do not form on the new prismatic apatite crystals which were described above (Figure 8D) but on the 'cauliflower' crystallites (Figure 2B). It seems thus that the newly formed prismatic apatite crystals reject uranium from their lattices.

Figure 6. Change in U oxidation state during ›oratory heating experiments. ●, Experi-:ntal; △, clinker; × ore.

ore and after extraction

After extraction (parts 10^{-6})			
U[a]	U(IV)[a]	U/P_2O_5	Extracted U%
105	40		4
108			7
108			7
106			6
110			4
118			3
145			8
38			66
44	2		65
44	5	1.2×10^{-4}	68
48	2		65
34	3		74
25			78
76	2		38
83	3	2.3×10^{-4}	41
21			85
28			76
03	10		11
02	9		14
94	4		34
89	4		32

‹ concentrations' (Figure 8B) which lical change occurs in the U distribu-:ven more impressive at about 900°C ated with the strong recrystallisation :h new apatite crystals were observed ›n and microprobe analysis showed, form on the new prismatic apatite 'cauliflower' crystallites (Figure 2B). s reject uranium from their lattices.

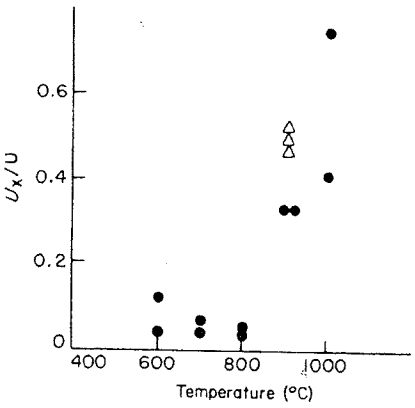


Figure 7. Variation of U_x (the uranium insoluble in phosphoric acid) with temperature. ●, Experimental; △, kiln products.

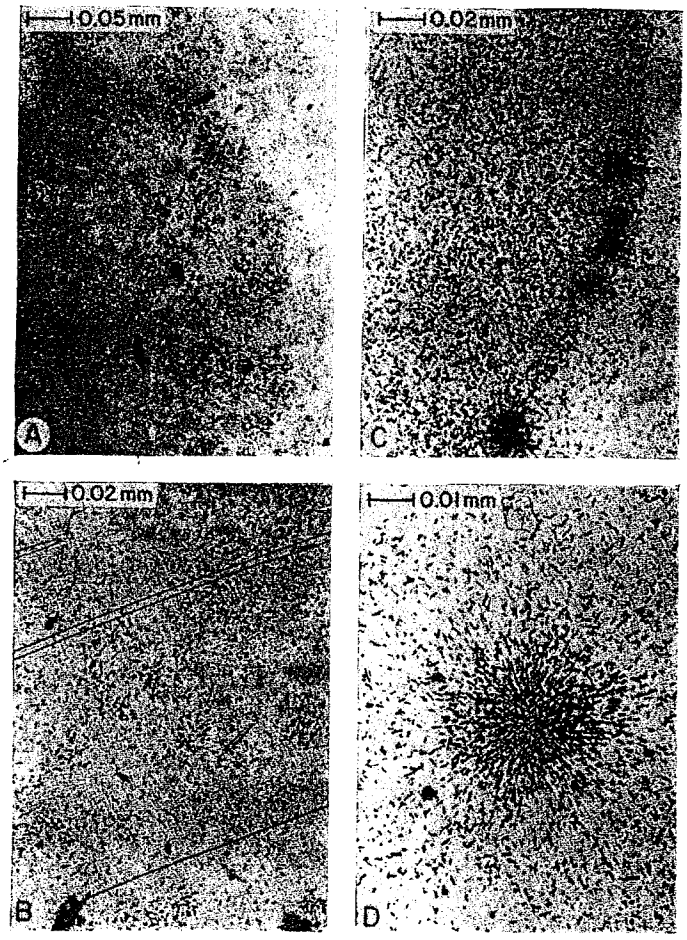


Figure 8. Fission track maps of ore and calcined phosphorite samples. (A) Ore-homogeneous distribution. (B) Sample heated to 800°C: radial track pattern. (C) Calcined sample from Oron kiln (~900°C): 'track stars' in a track-rich area. (D) 'Track stars' from a sample heated in the laboratory at 900°C.

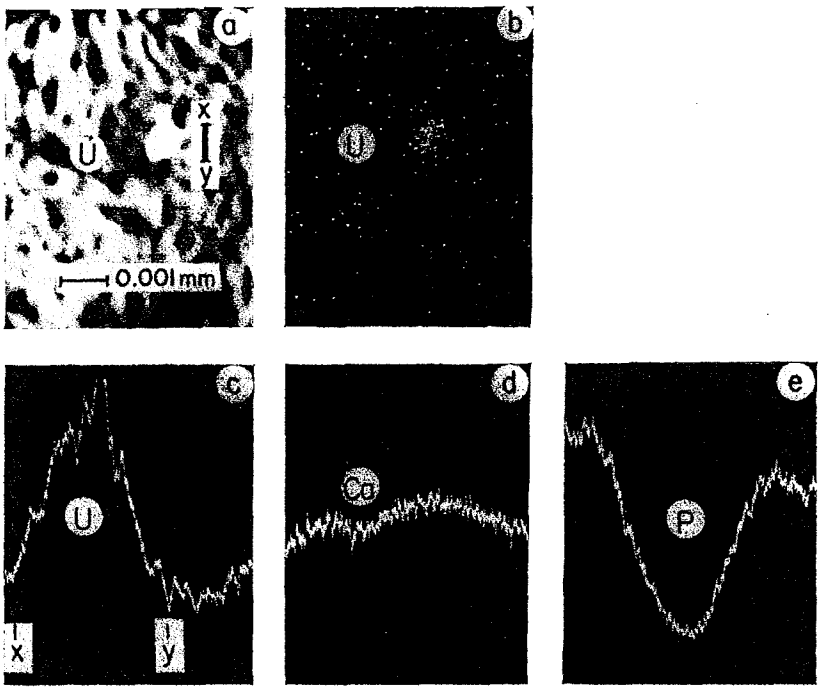
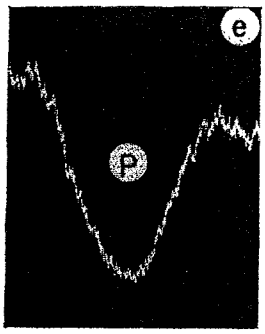


Figure 9. Electron microprobe traverse for U, Ca, P across a 'track star'.

The number of f.t. in 'track stars' was too large to be counted. Analysis of the corresponding areas on the electron microprobe (D. Shafranek, Analyst) yields a uranium concentration of about 1–2%. The 'track star' areas are slightly higher in Ca and low in P (Figure 9). The insoluble residue of a calcined rock (sample 34, Table 4) dissolved in phosphoric acid was also analysed. This is the fraction which contains U_x (Table 4). When calculated per rock weight, sample 34 contains 62 parts 10^{-6} uranium-insoluble in phosphoric acid (U_x). The residue itself contained 600 parts 10^{-6} U, and when an f.t.m. of it was made, 'track-stars' were abundant. An XRD analysis of this residue indicated the presence of CaF_2 (fluorite), and Ca silicates(?). Mair[22] found that after calcination to 1000°C a large portion of the uranium is immobilised by uptake in CaF_2. It is therefore suggested that at about 900°C fluorite is formed from the interaction of the released fluorine and calcium, which incorporates a part of the uranium in the rock. Since this formation occurs at a higher temperature than the temperature of effective uranium oxidation (600°C, Figure 6), the uranium which is assumed as contained in fluorite is hexavalent.

The above outlined localisation pattern of uranium during phosphorite calcination is confirmed by observations on the Oron kiln products. 'Dense track concentrations' and 'track stars' are the predominant f.t. patterns in Oron clinker and in the end product phosphate. In several cases the mobilisation of uranium during calcination was so severe that the f.t. distribution of the calcined rock no more follows boundaries of petrographic species, neither ovulites nor bone fragments; rather f.t. concentrations cut through fragment boundaries and ovulites forming a pattern not related to the rock texture (Figure 10).

It has been noted that a considerable difference exists between U f.t. maps of samples from different production runs. Whereas in two such runs 'track stars' were observed, in the third run only a pattern of dense track accumulations was observed. We could not decide whether this difference stems from differences in the chemistry of each batch, or from a variability in kiln conditions. Fisher and Bar[23] reported the formation of interstitial melt at high calcination temperatures. Such



across a 'track star'.

Analysis of the corresponding areas
a uranium concentration of about
1 P (Figure 9). The insoluble residue
: acid was also analysed. This is the
ock weight, sample 34 contains 62
ue itself contained 600 parts 10^{-6} U,
nt. An XRD analysis of this residue
air[22] found that after calcination to
ce in CaF_2. It is therefore suggested
the released fluorine and calcium,
this formation occurs at a higher
ion (600°C, Figure 6), the uranium

hosphorite calcination is confirmed
entrations' and 'track stars' are the
uct phosphate. In several cases the
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ither ovulites nor bone fragments;
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observed, in the third run only a
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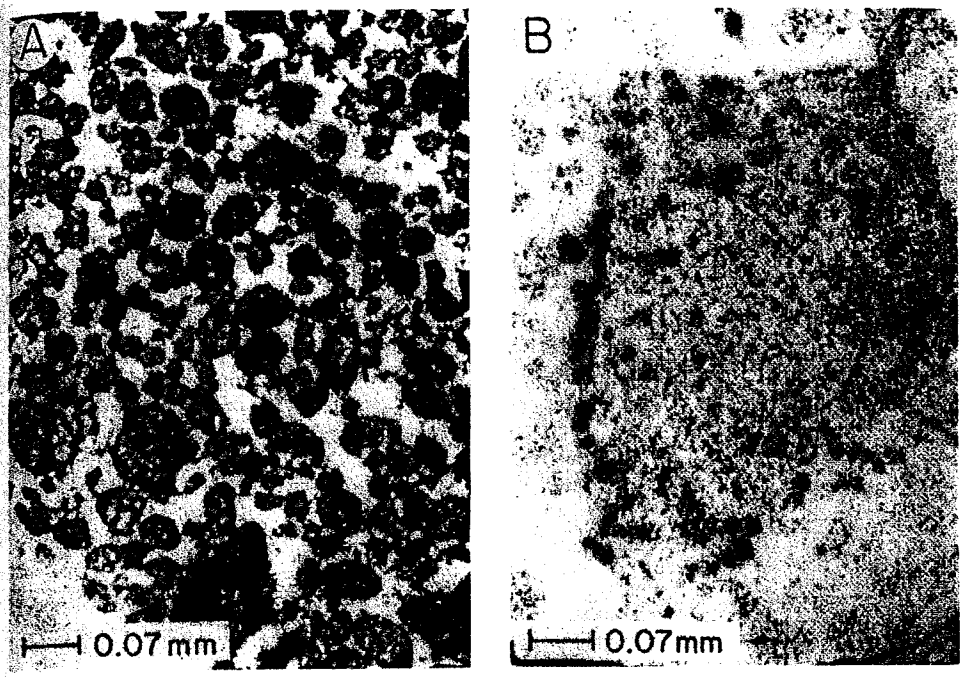

Figure 10. Fission track map of Oron clinker. (A) Thin section of a clinker sample; crossed nikols. (B) Fission track map of the same area showing a pattern unrelated to rock texture.

a melt could facilitate the migration of U through it and might explain the occurrence of the f.t. patterns which cut through grains and fragments (Figure 10).

3.4. Uranium extraction

Extraction experiments are based on extraction methods developed mainly by the Israeli Atomic Energy Group,[5] and summarised recently.[24] Following their experience, acetic acid was used as the leaching agent. It should be noted that in our extraction procedure only hexavalent uranium is being extracted, and that the extracting solutions are not causing uranium oxidation (Y.Folkman, personal communication). No attempt was made here to define optimum conditions for U extraction, but rather to determine the nature and localisation of uranium in the extraction products.

Table 4 and Figure 11 summarise the attempts to extract uranium preferentially without dissolving apatite. In two cases the U/P_2O_5 ratio is compared before leaching to the same ratio in the solid residue after leaching (the 'green cake').

Leaching of uncalcined raw material results in practically no preferential extraction of uranium. This is in good agreement with previous studies.[3,5] In all cases the difference between U in the ore and in the 'green cake' is within experimental error. Such remains the case also for heated samples up to 600°C. A dramatic change occurs when phosphate rock is being heated to 700–800°C. Here as much as 65% of the total U may be leached away. It is interesting to trace the source of such an impressive increase. Obviously one part of the uranium reservoir in the rock which may become leachable is that fraction which was tetravalent in the ore and became oxidised during calcination, thus being rendered soluble as well. It is reasonable to assume that the uranium which changed its oxidation state also became structurally less stable. In any attempt at uranium mass balance one must note, however, that since the fraction of hexavalent uranium in the uncalcined rock is 55–66% of the total uranium, there must be a portion of uranium which, although hexavalent in the ore, does not become leachable unless heated to 700–800°C. The observation that part of U(VI) which

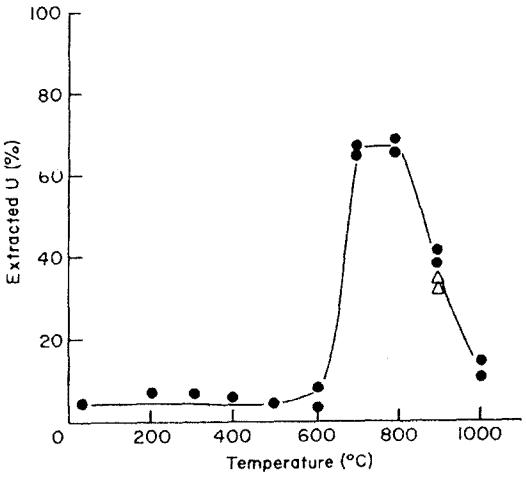

Figure 11. Variation of the extracted U with temperature. ●, Experimental; △, kiln products.

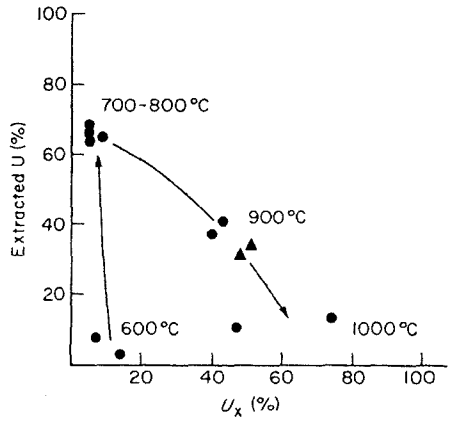

Figure 12. Relation between the extracted U and U_x at various temperatures. ●, Experimental; ▲, kiln products.

could not be extracted from the ore, becomes extractable at 700–800°C may be explained by the fact that it is in this temperature range that the following changes were noted: (a) recrystallisation of apatite was observed under the scanning electron microscope, clearly at 800°C, but possibly incipient already at 700°C; (b) a change in the slope of the ratio 'temperature-lattice/dimension' (which may be related to recrystallisation); (c) the beginning of U migration, probably to grain and crystal boundaries (as demonstrated by changing f.t. patterns). From such new sites U should be more leachable. The fact that only drastic recrystallisation makes U in phosphorites leachable seems to support previous views[25] that uranium is part of the apatite lattice.

The f.t. distribution in the 'green cake' is uniformly lower than in the calcined samples before extraction.

Progressive heating to 900°C and above, reduces the percentage of extractable U from 35–40% at 900°C to as low as 11% of the U content at 1000°C. This efficiency was achieved both in laboratory and kiln experiments. Fission track maps of the 'green cake' of samples calcined to above 900°C show that 'track stars' which were present in the calcined rocks, did not dissolve during leaching, whereas the dispersed uranium was leached out.

It is in this range of above 900°C that the appearance of a phosphoric acid-insoluble uranium fraction was observed (U_x). This was interpreted here, as reflecting the formation of non-apatitic phases (possibly fluorite, silicates) which include uranium. A similar conclusion was reached in other

experiments.[22] The relation between the percentages of the leachable uranium and the percentage of the total U which enters phosphoric acid-insoluble phases is shown on Figure 12. In the range above 700°C the higher the amount of U insoluble in phosphoric acid (U_x in non-phosphatic phases) the lower the amount of leachable uranium.

One way of preventing the formation of non-phosphatic-insoluble phases is the addition of other salts to the calcination process.[22] When Na_2CO_3 was added in our experiments, no phosphoric acid-insoluble residue was formed and, in parallel, about 85% of the uranium was extracted by acetic acid (Table 4). Presumably the additive prevents formation of CaF_2 and silicates by forming acid-soluble salts such as NaF and Na-silicates. The effect of Na_2CO_3 addition is also evident on f.t. maps. Na_2CO_3-bearing calcined samples do not show any 'track stars'. After U has been extracted from these samples only scarce f.t. remained. Progressive heating from 800 to 900°C with a Na_2CO_3 additive promotes recrystallisation and greater U migration to grain boundaries. Whereas at 800°C 74% of the U is extractable, this percentage increases to 85% at 900°C (Table 4). Hence, during U calcination two opposing processes are acting: (a) apatite recrystallisation which results in U(VI) migration to leachable sites; (b) formation of new phases which are acid-insoluble. These phases incorporate uranium and prevent its extraction. To maximise U extraction from calcined phosphorites one should aim at an optimum point where process (a) is most advanced and (b) is minimal; the latter can be achieved either by selecting the appropriate calcination temperature or by salt addition.

Figure 11. Variation of the extracted U with temperature. ●, Experimental; △, kiln products.

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Acknowledgments

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Treatment Chemicals Contribute to Arsenic Levels

By Cheng-nan Weng, Darrell B. Smith, And Gary M. Huntley

Arsenic is an issue that water utilities no longer can avoid. The US Environmental Protection Agency is expected to propose a reduction in the federal drinking water standard on arsenic from 50 µg/L to 5 µg/L later this year, although USEPA is also considering setting the maximum contaminant level at 3 µg/L, 10 µg/L, and 20 µg/L The final arsenic rule is due by Jan. 1, 2001.

Utilities should test their sources of water for arsenic and compare them with the proposed levels of 3, 5, and 10 µg/L. However, testing source water alone may not be sufficient to determine the arsenic load in finished water. Some treatment chemicals may also contain trace amounts of arsenic. Utilities should review and estimate the maximum possible arsenic concentrations contributed by the chemicals they use in drinking water treatment. Even trace amounts add up and may contribute a substantial portion—possibly up to 10 percent—of a 3 or 5 µg/L maximum contaminant level.

Connecticut Experience

The South Central Connecticut Regional Water Authority has three surface water treatment plants (SWTPs) and five wellfields. Recently, SCCRWA calculated the arsenic burden derived from chemicals routinely used to treat surface and groundwater at these facilities. Those chemicals are listed in Table 1.

To estimate the trace arsenic levels in the bulk treatment chemicals, data from the suppliers' analysis report or product specifications were used. The resulting trace arsenic concentrations in the finished water that were contributed by the treatment chemicals were computed by one of the following two methods:

1. For those chemicals with dosages expressed as mg/L of product chemicals (such as polymer, sulfuric acid, bimetallic zinc metaphosphate, and potassium permanganate), the resulting trace arsenic concentration in the finished water was computed by multiplying the chemical dosage by the trace arsenic level in the bulk treatment chemical.

2. For other chemicals (such as alum, ferric chloride, caustic soda, and fluorosilicic acid), a dilution factor was determined by dividing the chemical concentration by the chemical dosage. The resulting trace arsenic concentration in the finished water was computed by dividing the trace arsenic level in the bulk treatment chemical by the dilution factor.

Information produced by several calculations is tabulated as follows:

- Table 2 shows the maximum possible arsenic concentrations contributed by treatment chemicals for one surface water treatment plant that uses alum (0.279 µg/L arsenic contributed).
- Table 3 shows the maximum possible arsenic concentrations contributed by treatment chemicals for the wellfield, which uses sodium hypochlorite for disinfection (0.249 µg/L arsenic contributed).

Treatment Chemical	# Surface Water Treatment Plants (3 total)	# Groundwater Treatment Facilities (5 total)
Sodium hydroxide	3	Not used
Sulfuric acid	1	Not used
Alum	2	Not used
Potassium permanganate	2	Not used
Ferric chloride	1	Not used
Synthetic polymer A	1	Not used
Synthetic polymer B	1	Not used
Chlorine	3	4
Sodium hypochlorite	Not used	1
Bimetallic zinc metaphosphate	3	5
Fluorosilicic acid	3	5

Table 1. Chemicals routinely used by the South Central Connecticut Regional Water Authority, and the number of facilities where they are used.

- Table 4 shows the range of maximum arsenic contribution by treatment chemicals for the SCCRWA (range of all compounds, 0.0002-0.245 µg/L).
- Table 5 compares in finished water the calculated amount of arsenic that is contributed by treatment chemicals with the analytical result (overall calculated range, 0.248—0.306 µg/L; analytical result <1µg/L in all cases).

These data show that in finished water the theoretical arsenic concentrations attributable to normal dosages of water treatment chemicals are extremely low (Tables 2, 3, and 4). This conclusion is supported by the analytical data (Table 5), which show arsenic concentrations to be below 1.0 µg/L in all of the SCCRWA's surface and groundwater treatment facility finished waters.

Conclusion

If the standard were set at 3 µg/L, about 10 percent of the MCL would come from the treatment chemicals, hardly a minimal amount. It is also interesting to note that about 90 percent of the arsenic that would be contributed by treatment chemicals is attributable to fluoride addition.

If your processes include the addition of chemicals, ask your manufacturer for the amount of arsenic in each. If necessary, obtain conversion charts for diluted products, as well. Then calculate how much arsenic those chemicals will add to your finished water. If the total is close to the MCLs proposed by USEPA, you have reason for concern.

To find out more about the proposed arsenic rule, go to the agency's Web site, <www.epa.gov/safewater/arsenic.html>, or call the Safe Drinking Water Hotline at (800) 426-2791.

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Treatment Chemical	Amount of Arsenic in Product	Dosage	Calculation of Contribution	Arsenic Contribution
50% alum	0.25 mg/L	10 mg/L*	Chemical concentration of 50% alum = 650 mg/mL Dilution factor = 650 x 1,000 ÷10 = 65,000 Arsenic contribution = 0.25 ÷ 65,000 mg/L	0.00385 µg/L
Polymer A	< 0.5 mg/L	2.0 mg/L	Arsenic contribution = 0.5 mg/L x 2 mg/L	0.001µg/L
50% Sodium hydroxide (NaOH)	1.5 mg/L (maximum)	12.5 mg/L* (maximum)	Chemical concentration of 50% NaOH = 770 mg/mL Dilution factor = (770 x 1,000)÷12.5 = 61,600 Arsenic contribution = 1.5 ÷ 61,600 mg/L	0.024 mg/L
Fluorosilicic acid (H_2SiF_6)	Maximum = 60 mg/L Normal = 28 mg/L	1.0 mg/L* as F	H_2SiF_6 solution contains 20% F or 244.8 mg/mL of F F dosage = 1.0 mg/L as F Dilution factor = 244.8 x 1,000 ÷1.0 = 244,800 Maximum arsenic contribution = 60 / 244,800 mg/L = 0.245 µg/L Normal arsenic contribution = 28 ÷ 244,800 mg/L=0.114 µg/L	0.114 µg/L (normal) 0.245 µg/L (maximum)
Bimetallic zinc metaphosphate	<2 mg/L	1.7 mg/L	Arsenic contribution = 2 mg/L x 1.7 mg/L	0.0034 µg/L
Potassium permanganate ($KMnO_4$)	4.8 mg/L	0.35 mg/L	Arsenic contribution = 4.8 mg/L x 0.35 mg/L	0.00168 µg/L
Chlorine	All manufacturer reports indicate that arsenic is not present in gaseous chlorine.			0
Total arsenic contributed by treatment chemicals				0.279 µg/L (maximum)

*Based on dry equivalents.

Table 2. Arsenic contributed by chemicals used to treat surface water at Lake Gaillard Water Treatment Plant

Table 3. Arsenic contributed by chemicals used to treat groundwater at North Cheshire Wellfield

Treatment Chemical	Amount of Arsenic in Product	Dosage	Calculation of Contribution	Arsenic Contribution
Sodium hypochlorite (NaOCl)	0.8 mg/L (maximum)	1.2 mg/L	1 lb of chlorine reacts with 1.128 lb of caustic soda to produce 1.05 lb of NaOCl. An excess of caustic soda is used as a stabilizer. Based on the arsenic concentration in the 50% caustic soda, the maximum arsenic concentration in the NaOCl is estimated to be 0.8 mg/L. Arsenic contribution = 0.8 mg/L x 1.2 mg/L	0.00096 µg/L
Fluorosilicic acid (H_2SiF_6)	60 mg/L (maximum)	1.0 mg/L as F	Dilution factor = 244.8 x 1,000 ÷1.0 = 244,800 Maximum arsenic contribution = 60 ÷ 244,800 mg/L	0.245 µg/L
Bimetallic zinc metaphosphate	<2 mg/L	1.7 mg/L	Arsenic contribution = 2 mg/L x 1.7 mg/L	0.0034 µg/L
Total arsenic contributed by treatment chemicals				0.249 µg/L (maximum)

Treatment Chemical	Range of Chemical Dosage (mg/L)	Range of Maximum Arsenic Contribution (µg/L in finished water)
Sodium hydroxide	8.0–12.5	0.0156–0.024
Sulfuric acid	20	0.0002
Alum	10–80	0.00385–0.0308
Potassium permanganate	0.30–0.35	0.0014–0.00168
Ferric chloride	7	0.037
Synthetic polymer A	2.0	0.001
Synthetic polymer B	4.0	0.004
Chlorine	1.2–2.8	0.000
Sodium hypochlorite	1.2	0.00096
Bimetallic zinc metaphosphate	1.5–1.7	0.0030–0.0034
Fluorosilicic acid	1.0	0.245

Table 4. Maximum finished water arsenic concentrations based on chemical dosages applied in the treatment facilities

Table 5. Maximum finished water arsenic concentrations based on chemical dosages applied in the treatment facilities

Treatment Facility	Trace Arsenic Concentration (µg/L) Calculated Maximum	Analytical Result
Lake Gaillard WTP*	0.279	<1
Lake Saltonstall WTP	0.299	<1
West River WTP	0.306	<1
North Cheshire Wellfield	0.249	<1
All other wellfields (N=4)	0.248	<1

*Water treatment plant

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Effects of fluoridation and disinfection agent combinations on lead leaching from leaded-brass parts

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Abstract

This study concerns effects on water-borne lead from combinations of chlorine (CL) or chloramines (CA) with fluosilicic acid (FSA) or sodium fluoride (NaF). CL is known to corrode brass, releasing lead from plumbing devices. It is known that CA and CL in different ratios with ammonia (NH) mobilize copper from brass, which we have found also enhances elution of lead from leaded brass alloys. Phase I involved leaded-brass 1/4 in. elbows pre-conditioned in DI water and soaked in static solutions containing various combinations of CL, CA, FSA, NaF, and ammonium fluosilicate. In Phase II 20 leaded-brass alloy water meters were installed in pipe loops. After pre-conditioning the meters with 200 flushings with 1.0 ppm CL water, seven different solutions were pumped for a period of 6 weeks. Water samples were taken for lead analysis three times per week after a 16-h stagnation period. In the static testing with brass elbows, exposure to the waters with CA + 50% excess NH_3 + FSA, with CA and ammonium fluosilicate, and with CA + FSA resulted in the highest estimated lead concentrations. In the flow-through brass meter tests, waters with CL + FSA, with CL + NaF, and with CL alone produced the highest average lead concentration for the first 3-week period. Over the last 3 weeks the highest lead concentrations were produced by CL + NaF, followed by CL alone and CA + NH_3 + FSA. Over the first test week (after CL flushing concentrations were increased from 1.0 to 2.0 ppm) lead concentrations nearly doubled (from about 100 to nearly 200 ppb), but when FSA was also included, lead concentrations spiked to over 900 ppb. Lead concentrations from the CL-based waters appeared to be decreasing over the study period, while for the CA + NH_3 + FSA combination, lead concentrations seemed to be increasing with time.

Keywords: Water disinfection; Chlorine; Chloramines; Fluoridation; Lead leaching

1. Introduction

1.1. Motivation for this study

The continuing problem of ingested lead from lead-bearing water was highlighted at a US House of Representatives sub-committee hearing convened in March 2004 to investigate issues concerning "First Draw" water lead levels as high as 1000 ppb in Washington DC water circa 2001–2004. An expert witness (Edwards, 2004) testified that this was found in homes without lead service lines or lead soldered copper piping. The only possible lead source had to be leaded-brass plumbing and/or brass faucets. The expert also suggested that a recent switch in disinfectant from chlorine to chloramine caused the problem. The study reported here, conducted by the Environmental Quality Institute of the University of North Carolina (EQI), focused on brass corrosion by combinations of disinfectant and fluoridating agents in two laboratory phases. In the first, small leaded-brass plumbing elbows (2% lead) were exposed under static conditions to DI water with chlorine and chloramines, either alone or in combination with municipal water fluoridating agents. Stagnant water lead data from that phase guided selection of combinations of disinfectant and fluoridating agent for a second phase in which brass water meters (8% lead) were exposed to seven water formulations under flow-through conditions. It was expected that Phase II results would be used to guide field tests under "real world" conditions in cooperating water plants, but the untimely demise of EQI Director Richard P. Maas prevented that follow-on step. Nevertheless, Phase I and Phase II results presented here provide at least heuristic insight

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into the "DC experience" and, more generally, shed new light on lead elution from brass by combinations of chlorine-based disinfectants and fluoridation chemicals.

1.2. Applicable terminology

Herein "CL" means a chlorine species used for potable water disinfection that may be injected as chlorine gas (Cl_2), or hypochlorite solutions carrying chloride ion, hypochlorous acid (HOCl), and/or hypochlorite ion (OCl^-) that may exist together in equilibrium. CL concentration may be expressed in parts per million (ppm) of "free chlorine" where 1 ppm is the stoichiometric equivalent of 29×10^{-6} mol of free chloride ion.

"NH" means ammonia added to CL treated water to produce mono-chloramine. NH may be injected as ammonia gas (NH_3), ammonium hydroxide solution, or as an ammonium salt solution. The desired proportion of CL/NH is 1:1 on a molar basis which is little less than 5:1 in ppm units.

"CA" means "chloramine" produced by adding NH to CL treated water; the desired mono-chloramine is actually part of a mixture with small amounts of di- and tri-chloramine. Actual amounts of NH and CL vary from time to time, yielding either undesired di- or tri-chloramine or excess NH. Chlorine in CA is also known as "combined chlorine," a term also applied to products created when CL reacts with water contaminants (USEPA, 2004).

"SiF" applies to the silicon/fluorine complex (fluosilicate), a class of water fluoridating agents (aka fluorosilicates, silicofluoride, silicofluoric, hexafluorosilicate, and other names) from which fluoride ion (F^-) is released upon dissociation of $[SiF_6]^{2-}$ when diluted in water. The principal SiF agents are fluosilicic acid (H_2SiF_6), herein "FSA", and its sodium salt (Na_2SiF_6). Concentrated (20–30%) FSA is injected as such into water plant water. Sodium fluosilicate (NaFSA) is added as a saturated solution. The term SiF covers $[SiF_6]^{2-}$ and its dissociation derivatives.

2. Relevant background

2.1. Continuing problem of drinking water lead

Lead contaminated drinking water remains a significant public health issue in the United States, even though water-borne lead has steadily declined along with other lead sources such as lead-based paint, roadside soils, food, and other products. In 1991, the EPA estimated that drinking water was responsible for 14–20% of total lead uptake of all ages in the U.S. (USEPA, 1991). EPA's Lead and Copper Rule (LCR) for potable water was expected to reduce drinking water lead levels by 50%. That did occur (Maas et al., 2005) after leaded-solders were banned under the Safe Drinking Water Act Amendments of 1986 (USEPA, 1986); water suppliers were required to reduce corrosivity of their finished waters (Maas et al., 1994; Ramaley, 1993; USEPA, 1991); with better control of alkalinity, pH, and additives (Cardew, 2003; Edwards et al., 1996; Lytle and Schock, 2000). On its own initiative, the California legislature set limits on lead content of leaded-brass plumbing devices and faucets (Patch et al., 1998; State of California, 1995). Much research focused on preventing lead extraction from installed lead service lines by treating water with phosphatic agents (phosphoric acid, combinations of orthophosphoric acid and zinc orthophosphate, polyphosphates, or blends of orthophosphoric acid with polyphosphate) that produce inert barrier coatings inside lead pipes. Along with successes in this area there have also been conflicting results. Orthophosphate treatments can reduce soluble lead levels by 70%, but polyphosphate can actually increase lead and copper in drinking water (Edwards and McNeill, 2002), often manifested as particulates (McNeill and Edwards, 2004).

2.2. Complicating factors

2.2.1. Switch to chloramines for disinfection

Water lead problems have been exacerbated by EPA's Stage I Disinfection By-products Rule (USEPA, 2002) requiring reduction of disinfection by-products (DBPs) such as trihalomethanes (THM's) and haloacetic acids (HAA's) created by CL disinfection (ChemScan, 1997). A switch from CL to CA was recommended and adopted in some systems since it was less expensive than other disinfection methods and easy to add NH to already-chlorinated water. One explanation for the DC experience was that the switch from CL lowered the oxidizing potential of DC water, destroying the normally protective lead dioxide (PbO_2) scale inside lead pipes (Renner, 2004).

Although CA corrosivity has received a lot of attention, no studies have included fluoridating agents (Edwards and Dudi, 2004; Eisnor and Gagnon, 2004; Lin et al., 1997; Reiber, 1993; Sung et al., 2005). A microscopy study revealed how CA alone is a good solvent for lead (Switzer et al., 2006). Whatever these studies may have found under laboratory conditions, it should be noted that CA in the water plant is not added as a commercial product with consistent properties. It is formed by adding NH to CL treated water. Ideally, mono-chloramine is the principal product formed at pH 8 and the proper 1:1 NH/CL molar ratio. Maintaining exactly the ideal NH/CL proportion at all times is not very likely.

2.2.2. The role of fluoridating agents

Fluoridating agents can only complicate matters. Sodium fluoride (NaF), used to treat less than 10% of US fluoridated water, raises pH a little with negligible effect, but the same cannot be said for possible interference by the fluoride ion in the reaction of NH with CL. The effect of SiFs is another matter. The fluosilicate anion $[SiF_6]^{2-}$ of FSA and NaFSA provides the fluoride ion (F^-) in over 90% of fluoridated water. $[SiF_6]^{2-}$ releases F^- in a complicated, poorly understood, sequence of time-, temperature- and pH-dependent steps. Under water plant operating conditions, incompletely dissociated $[SiF_6]^{2-}$ residues may survive and react with other chemicals in the water. Under some conditions, NH and FSA, as such, react to produce silica and ammonium fluoride (Mollere, 1990). How that affects corrosion is not known, but whatever its reaction with NH may be, FSA does not leach lead simply because it is an acid.

The fluosilicate anion $[SiF_6]^{2-}$ and/or partially dissociated derivatives have a unique affinity for lead. Lead fluosilicate is

one of the most water soluble lead species known, a property recognized and exploited for many years (Stauter, 1976). FSA has been used as a solvent for lead and other heavy metals in extractive metallurgy (Cole et al., 1981; Kerby, 1979) and to remove surface lead from leaded-brass brass machined parts (Bonomi et al., 2001; Giusti, 2001, 2002). With or without CA, FSA would extract lead from brass. Besides, in the water plant situation it is reasonable to expect FSA to combine with NH as ammonium fluosilicate, an excellent solvent for copper alloys (Hara et al., 2002) and other metals (Silva et al., 1995).

It has been argued that FSA dissociates almost completely at the levels typically added to drinking water, and therefore cannot be more corrosive than sodium fluoride (NaF) (Urbansky and Schock, 2000). However, in a comprehensive follow-up review of the literature, Urbansky states that FSA may not dissociate completely in drinking water (Urbansky, 2002). Evidence for that is not new (Colton, 1958; Kolthoff and Stenger, 1947; Lenfesty et al., 1952; Munter et al., 1947; Thomsen, 1951). Titration of FSA to a pH 7 end-point only neutralizes the two hydronium ions produced by ordinary hydrolysis of H_2SiF_6, leaving the fluosilicate anion $[SiF_6]^{2-}$ intact. In addition to that, $[SiF_6]^{2-}$ dissociation in cold water could take 20 min to reach 90% completion (Hudleston and Bassett, 1921; Rees and Hudleston, 1936) and may never get to that condition below pH 9 (AWWA, 1994).

Consequently, incompletely dissociated $[SiF_6]^{2-}$ residues may remain in water plant water that is not above pH 8 or some commonly occurring low temperature. Apart from problems with incompletely dissociated $[SiF_6]^{2-}$ residues, injection of concentrated of FSA simultaneously and in close proximity with NH almost guarantees unanticipated side reactions.

2.2.3. Distribution of lead in brass

The varied, occasionally conflicting, reports on elution of lead from brass may have a common explanation. Lead alloyed with copper is not molecularly distributed, as in a solid solution. Discrete lead nodules are embedded in a copper matrix. Agents that attack copper are likely to foster lead mobility, adding significantly to lead (probably particulate) in drinking water. CL, CA, or excess NH are all capable of doing that, either by copper stress cracking (Flom, 2002) or mobilization in an ammonia/copper complex (Clark, 2003), thereby exposing lead nodules in brass for easier transport into water.

This may help to explain the DC experience that homes with only brass as a possible source of lead, not only had high water lead, but were also experiencing serious pitting of copper pipe. In many cases, particulate lead may predominate over soluble lead eluted from brass, as well as other lead sources (McNeill and Edwards, 2004).

3. Materials/methods and statistical analyses

3.1. Phase I (static tests of 2% leaded-brass elbows)

Sixty 2% leaded-brass 1/2 in. barb 90° elbows were purchased locally. Three elbows were assayed for lead in a small piece sliced from one end. Measured lead concentrations ranged from 1.70 to 1.82%. Elbows were labeled, thoroughly rinsed, and placed in a tray of deionized (DI) water for conditioning. The water was changed twice and agitated three times a day for 18 days. After conditioning, two sets of static bottle tests were conducted as follows: individual elbows were removed from the trays of DI water, rinsed with DI water and placed in their own labeled bottle. Exactly 100 mL of the appropriate test water was added to each bottle which was capped and set aside to sit undisturbed overnight. After 16-h stagnation exposure, each elbow was removed from its bottle of test water with plastic tongs, rinsed with DI water, and placed back into its tray of DI water. Test waters were analyzed for lead using the EPA 200.8 method for graphite furnace atomic absorption spectrophotometry.

In the first set of bottle tests, elbows were exposed to waters at pH 7 and pH 8, comprising 2 ppm each of: (1) CL only; (2) FSA only; (3) CA only; (4) CA + FSA. CL was adjusted by adding the appropriate amount of dilute sodium hypochlorite (NaOCl) solution. For the FSA waters, enough FSA was added to produce 2.0 ppm F^- which represents above average, but not unusual conditions within the highest permissible level (MCL) for drinking water fluoride. FSA was added as pre-diluted 26% FSA.

Although the CDC nominal "optimum" adjusted F^- concentration is 1.0 ppm, it is only a mid-range figure (CDC, 2001). The CDC recommends adjusting F^- according to mean annual local temperature. In colder areas (50–54 °F annual mean) such as Great Lakes States, the optimum is 1.1–1.7 ppm and in warmer areas (71–79 °F) it is 0.7–1.3 ppm. An allowance is also made for deviation from these boundaries by 0.1 ppm on the low side and 0.5 ppm on the high side (CDC, 1999). Hence, a water plant taking water in December through April from Lake Ontario or the Northern reaches of the Mississippi River could comply with CDC's optimum F^- at 2.0 ppm. Also, the recommended optimum F^- for school water systems is 4.1–5.0 ppm for the middle temperature range (CDC, 1999). Therefore, the 2.0 ppm F^- concentrations in this study were in the range experienced by much of the U.S. public.

CA was prepared as a stock solution comprising ammonium hydroxide and sodium hypochlorite in stoichiometric equivalent concentrations. For test water exposure, appropriate amounts of stock solution were pH adjusted upward by adding sodium bicarbonate ($NaHCO_3$) or downward using hydrochloric acid (HCl). Elbows were randomly assigned to test waters so that each water composition had five elbows assigned to it.

The conditionings using pH 8 were dropped in the second set of bottle tests to allow testing of more types of water additives. The same procedures used in the first set of bottle tests were carried out in the second set at pH 7 and 2.0 ppm of each constituent: (1) DI water only; (2) CL only; (3) CA only; (4) CA + 50% excess NH; (5) CA + FSA; (6) CA + 50% excess NH + FSA; (7) CA solution into which 26% FSA was added to produce 2 ppm of fluoride without pre-dilution; (8) FSA + NH; (9) CA + ammonium fluosilicate.

CL, CA, FSA, and pH were adjusted the same way as the first bottle tests. NH was adjusted by adding ammonium hydroxide

in appropriate 1:1 molarity with CL alone as well as with 50% excess NH to represent water plant control deviations described above regarding excess ammonia. The 50% excess NH is consistent with 50% greater than nominal 1 ppm F^- optimum, therefore a reasonable condition to occur in a water plant with the risk of side reaction between SiF and NH. The difference between conditions 5 and 7 was based on that premise. In condition 5, FSA was diluted before adding it to the mixture, as was done for other treatments using FSA. In condition 7, it was added at 26% concentration and the resulting mixture diluted to 2 ppm F^-.

For reasons described earlier, mixing concentrated FSA with NH with ample time to react should approximate water plant conditions, producing a species with corrosion potential differing from that when pre-diluted dissociated FSA meets ammonia. It should be noted here that concentrated FSA and NH are frequently injected into water plant water in close proximity to each other (District of Columbia Water and Sewer Authority, 2002). The rationale for including conditions 8 and 9 was that NH is known to react with copper, forming the soluble copper/ammonia complex, thus possibly exposing additional lead surface in the brass.

3.2. Phase I statistical analyses

For both sets of bottle tests, ANOVA analyses conducted on the natural-log-transformed lead concentrations found no evidence of significant non-normality (Kolmogorov–Smirnov p-values = 0.124 and 0.100) or heterogeneity of variances (Levene p-values = 0.191 and 0.979). Tukey's least significant difference (LSD) procedure was used to perform a multiple comparison of log lead concentrations between each combination of water and pH. Confidence intervals for the median lead concentrations of all elbows that might be exposed to those conditions were calculated for each water-pH combination under the assumptions that the log lead concentrations were approximately normally distributed with common variance. Least square means and corresponding individual confidence intervals were calculated from the ANOVA analysis for these log lead concentrations for each combination, and then the inverse transform was conducted on the least square means of the logged data to obtain the estimated medians and 95% confidence intervals for the median lead concentrations.

3.3. Phase II (flow-through tests 8% leaded-brass meters)

Twenty leaded-brass Hersey Model 430 water meters were purchased locally in Asheville, NC. Three meters were selected randomly and assayed for lead on a small slice from the meter exterior with results ranging from 7.59 to 8.44% lead. Meters were randomly assigned to one of seven types of water and hooked up to a plumbing manifold consisting of three meters for each of six waters and two meters for one water. Connected plumbing included a Flojet Model 2100-953-115 plastic vacuum pump and a 100 L Nalgene laboratory carboy (see Fig. 1).

Each manifold system was conditioned by flushing 350 mL of a 1.0 ppm CL/DI water solution through all the meters about

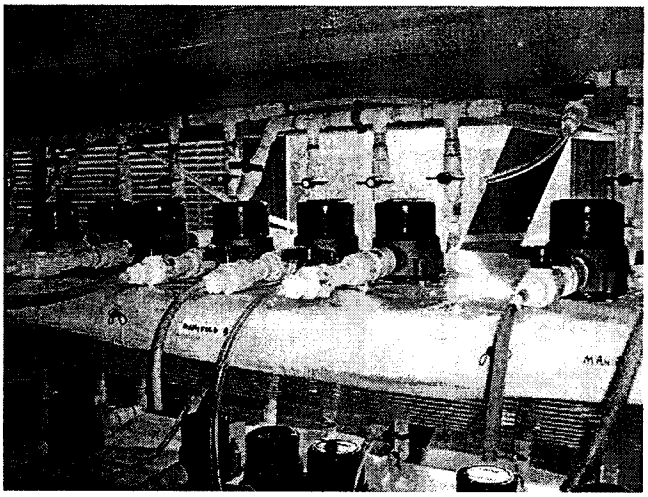

Fig. 1. Picture of Phase II setup.

15 times each weekday for 2.5 weeks for a total of approximately 190 times. One additional week of conditioning was completed by flushing 350 mL of the CL/DI water through the meters another 63 times. For this final week, samples were taken on Wednesday, Thursday, and Friday mornings at approximately 8:30 a.m. The late afternoon before each sample was taken a 2-L flush was completed at 4:30 p.m. and the meters sat undisturbed overnight 16 h until the morning sample at 8:30 a.m. Conditioning provided stagnation water lead data for 1 ppm CL prior to switching to test waters. After conditioning, the following waters with 2.0 ppm of each constituent were studied: (1) CA + FSA; (2) CA + 100% excess NH + FSA; (3) CA + 100% excess NH; (4) CA + 100% excess NH + NaF; (5) CL + FSA; (6) CL + NaF; and (7) CL alone. The target pH for all waters was 7.5 with an acceptable range of 7.3–7.7. The 100% excess NH was used as a potential worst-case scenario simulating a situation that might reasonably occur from time to time in a water plant. The CL, CA, FSA, NH, and pH were adjusted using the same methodology as that of Phase I.

Three meters were tested for each water composition, except the CA + FSA combination which was tested with two meters. For 6 weeks the plumbing manifold systems were flushed four times each weekday. Each meter had 1 L of its respective test water flushed through it three times, and the final flush of each day was 2 L. Sampling occurred each week for the 6-week sampling period on Tuesday, Wednesday, and Thursday mornings following a 16-h stagnation period.

3.4. Phase II statistical analyses

To assure that no meter was used that might be particularly susceptible to corrosion, an ANOVA analysis was performed on the log-transformed lead concentrations for the samples taken during conditioning. Median values of stagnation water lead concentration were found for each set of meters and combination of day and water composition. As with the elbow data, the natural logarithm was taken for each lead concentration. Lead concentrations for each meter were averaged over

the first 3 and last 3 weeks of the study. These arithmetic means were log transformed to give a single value to each meter for each of the two periods.

ANOVA analyses were performed on these log-transformed means and individual 95% confidence intervals for the log-transformed means calculated. An inverse transform was applied to these values to estimate the typical mean stagnation water lead concentration produced by the given water over the given time periods. ANOVA data for each of the pre-treatment period, first 3 treatment weeks, and last 3 weeks provided no evidence of significant non-normality (Kolmogorov–Smirnov p-values = 0.079, >0.15, >0.15, respectively) or heterogeneity of variances (Levene p-values = 0.962, 0.475, 0.218, respectively).

4. Results

4.1. Phase I results (2% lead elbows)

For the first set of bottle tests pH, water treatment, and their interactive effect were all significantly related to log lead concentration (p-values = <0.001, <0.001, 0.006, respectively). Fig. 2 displays the 95% confidence intervals for medians of the first set of static bottle tests. As seen from the results of the Tukey's LSD (Table 1) and individual confidence intervals (Fig. 2), lead concentrations are significantly higher at pH 7 for the CL and the CA waters, but not for the other two. The highest lead concentration was produced by CA + FSA under both pHs. CA at pH 7 produced the next highest lead concentration, but not significantly less than the highest. Absent a factorial design it was not possible to test formally for an interactive effect of CA + FSA. FSA alone gave results very similar to CL. Although CA + FSA produced the highest lead concentrations at both pHs, the combination was significantly higher than CA alone only at pH 8. Thus, bottle test 1 provides evidence, albeit not compelling, for a positive interactive effect of CA + FSA on leaded-brass corrosion.

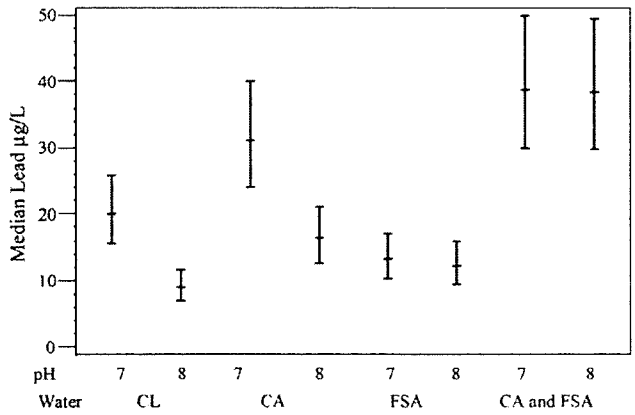

Fig. 2. Median 95% confidence intervals for bottle test 1 at pH 7 and pH 8.

Table 1
Estimated median lead concentrations for bottle test 1

	Water							
	Cl	FSA	FSA	CA	Cl	CA	CA + FSA	CA + FSA
PH	8	8	7	8	7	7	8	7
Estimated median	9.0	12.3	13.3	16.3	20.0	31.1	38.4	38.7

Combinations covered by the same line are not significantly different using Tukey's LSD statistic with a significance level of $\alpha = 0.05$.

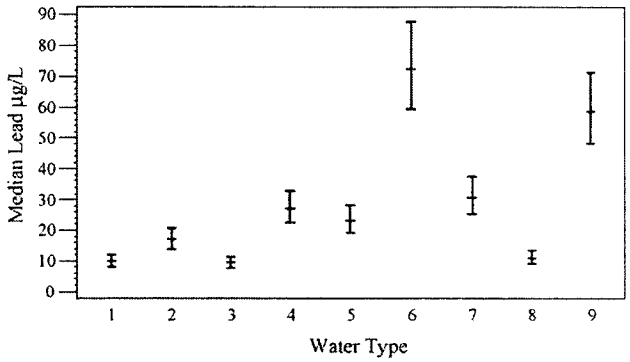

Fig. 3. Median 95% confidence intervals for bottle test 2. 1, Deionized Water; 2, CL Only; 3, CA Only; 4, CA and 50% excess NH; 5, CA and FSA 6, CA,50% excess NH, and FSA; 7, CA with 26% FSA Added; 8, FSA NH; 9, CA with concentrated ammonium fluorosilicate.

For the second set of bottle tests, water composition was the only factor having a significant effect on log lead concentrations ($p = 0.000$). Estimated median and confidence intervals for the median lead concentration of each water is displayed in Fig. 3. From the individual confidence intervals (Fig. 3) and Tukey's LSD results (Table 2) it can be seen that CA + 50% excess NH + FSA (#6) and CA + ammonium fluosilicate (#9) produced the two highest lead concentrations. CA + 50% excess NH; CA + FSA; CA + concentrated FSA produced intermediate concentrations. CL alone, CA alone, DI water alone, and the combination of NH + FSA produced the lowest concentrations. Unlike the first set of bottle tests at pH 7, CA alone produced significantly lower lead concentration than CL alone. CA + concentrated FSA produced a higher concentration than CA + pre-diluted FSA but the difference was not statistically significant.

Table 2
Estimated median lead concentrations for bottle test 2

Water code	Estimated median
3	9.4
1	9.9
8	11.1
2	17.0
5	23.3
4	27.2
7	30.8
9	58.8
6	72.3

Combinations covered by the same line are not significantly different using Tukey's LSD statistic with a significance level of $\alpha = 0.05$.
1, deionized water; 2, CL only; 3, CA only; 4, CA and 50% excess NH; 5, CA and FSA; 6, CA + 50% excess NH + FSA; 7, CA with 26% FSA added; 8, FSA + NH; 9, CA + concentrated ammonium fluosilicate.

The interactive effects of CA and FSA on lead leaching cannot be directly examined from the set of waters in the second set of bottle tests. However, effects of excess NH, FSA and their interactive effect can be evaluated for the CA waters. The four conditions: (3) CA only; (4) CA + 50% excess NH; (5) CA + FSA; and (6) CA + 50% excess NH + FSA make up a two-way full factorial experimental design. ANOVA analysis on just those four conditions found that in the presence of CA, both 50% excess NH ($p = 0.000$) and FSA ($p = 0.000$) were positively related to log lead concentration, but their interactive effect ($p = 0.662$) was not significant. Thus, in the presence of CA, 50% excess NH and FSA had positive additive effects on log lead concentrations in the second bottle study.

Median lead concentration for CA + 50% excess NH + FSA was greater than for CA + ammonium fluosilicate, but not significantly so. Thus, it seems reasonable to believe that CA + 50% excess NH + FSA leaches lead through a mechanism similar to that for CA with ammonium fluosilicate as such.

4.2. Phase II results (8% leaded-brass meters)

ANOVA analysis of the log-transformed lead concentrations during exposure to conditioning water (1.0 ppm CL/DI water solution) found no significant differences in the median lead concentrations between the groups of meters selected for the seven different water treatments (p-value = 0.771). The estimated median lead concentration for meters exposed only to the conditioning regime was 84.0 μg/L.

On Day 13 after treatment began, a meter receiving CL + FSA was reported to have a stagnation water lead concentration of 2.9 ppb, while the other two meters had values of 49.5 and 62.4. The 2.9 outlier was not included in any analyses. The median ($n = 3$, except for CA + FSA where $n = 2$) lead concentrations for each day were obtained for each water chemistry. Fig. 4 displays the median lead concentrations for the CA-type waters over the 28 days of testing. Fig. 5 displays the median lead concentration for the CL-type waters over the 28 days.

The median range in lead concentrations for the three (or, in one case two) meters subjected to the same waters for the entire study period (not displayed on the figure) was 9.0 μg/L over all days and waters. Some day-water sets had much higher meter-to-meter ranges. For example, days 1–3 for CL + FSA water had ranges of 115.5, 217.3, and 136.4 μg/L, respectively, but these were measured for water lead data in the 1000 ppb regime. As expected for data that is approximately log-normally distributed, day-water combinations with larger medians tended to also have larger ranges.

Figs. 6 and 7 illustrate the variability of the stagnation water lead concentrations produced over the entire test period by meters receiving, respectively, CA + 100% extra NH and meters

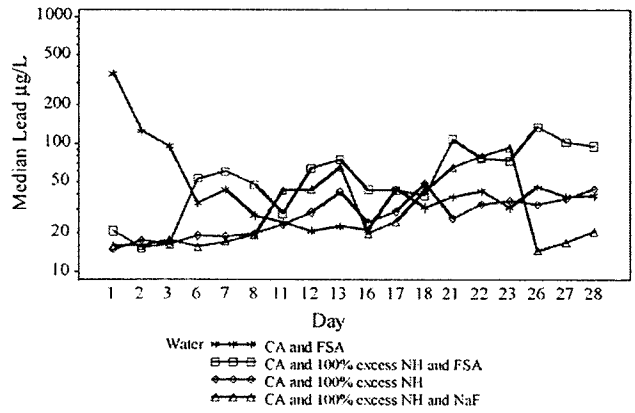

Fig. 4. Mean lead concentrations for the CA-based waters.

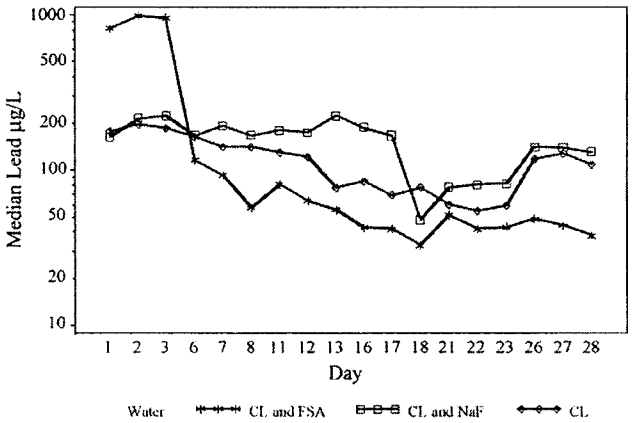

Fig. 5. Mean lead concentrations for CL waters by day.

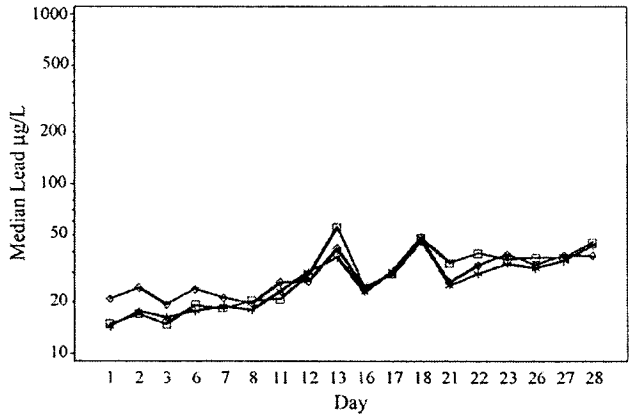

Fig. 6. Lead concentrations for meters using CA + 100% excess NH.

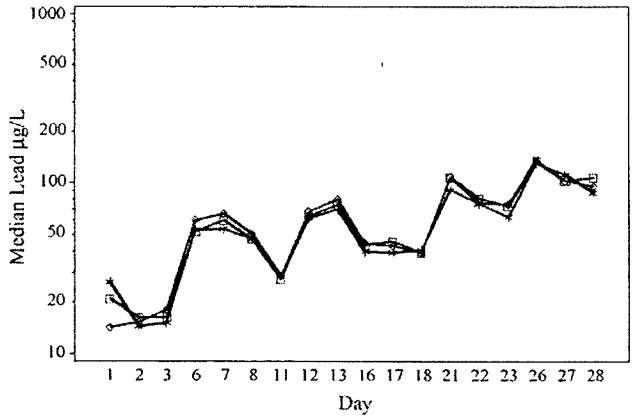

Fig. 7. Lead concentrations for meters using CA + 100% excess NH + FSA.

Table 3
Estimated median lead concentrations of meters by type of water averaged over day for the first 3 weeks of the study

Water	N meters	Estimated median (95% confidence interval)
CA and 100% extra NH	3	23.3 (21.0–25.9)
CA, 100% extra NH and NaF	3	28.1 (25.3–31.2)
CA, 100% extra NH and FSA	3	42.6 (38.4–47.3)
CA and FSA	2	83.1 (73.1–94.5)
CL	3	145.9 (131.4–162.0)
CL and NaF	3	185.3 (166.6–205.7)
CL and FSA	3	362.8 (326.7–402.9)

Water types that are covered by the same line are not significantly different from each other using Tukey's LSD with a significance level of $\alpha = 0.05$.

receiving CA + 100% extra NH + FSA. Clearly, for both treatment waters there is very little meter-to-meter variation in eluted lead on any one day. Such consistency is reasonable assurance that all the data reflect real effects, not merely random "chemical noise." Based on the premise that observed differences in stagnation water lead are not statistical aberrations, but due to explicable causes, day-to-day and week-to-week variability requires comment, which will be provided in the following section.

In the analyses shown in Figs. 4–7 a "week" is 5 days, since flushing occurred only on weekdays, and thus essentially no aging was considered to be occurring over weekends. Thus, Days 1–28 as shown in these figures represent 6 weeks of sampling on 3 successive days. Table 3 shows the median lead concentrations for all seven water chemistries averaged over the first 3 (5-day) weeks and Table 4 shows the corresponding results for the last 3 weeks of the study.

Fig. 4 and Tables 3 and 4 provide insights into corrosivity of CA-based test waters. The most corrosive of the CA-based test waters for the first 3 weeks was CA + FSA, while CA + excess NH + FSA was the most corrosive for the last 3 weeks and the only water showing a strong trend of increasing corrosivity over the whole experiment. Water with CA + excess NH and water with CA + excess NH + NaF were similar to one another in corrosivity which was significantly lower than the other waters. CA + excess NH + NaF displayed more variability over time than the other CA-based waters.

Fig. 4 and Tables 3 and 4 indicate that the water with CL + FSA was most corrosive for the first 3 weeks and CL + NaF was most corrosive for the last 3 weeks and second most corrosive for the first three weeks. Water with CL only was third most corrosive for the first 3 weeks and second most corrosive for the last 3. Water with CL + FSA showed a decreasing trend over the course of the experiment, while the other two CL-based waters displayed irregular, slightly decreasing trends.

Table 4
Estimated median lead concentrations of meters by type of water averaged over day for the last 3 weeks of the study

Water	N meters	Estimated median (95% confidence interval)
CA and 100% extra NH	3	34.8 (31.9–37.9)
CA and FSA	2	36.8 (33.1–40.9)
CA, 100% extra NH and NaF	3	43.2 (39.6–47.1)
CL and FSA	3	43.3 (39.7–47.2)
CA, 100% extra NH and FSA	3	79.2 (72.6–86.3)
CL	3	84.0 (77.0–91.6)
CL and NaF	3	115.2 (106.1–126.1)

Water types that are covered by the same line are not significantly different from each other using Tukey's LSD with a significance level of $\alpha = 0.05$.

Comparing the CL-based waters to CA-based waters, CL-based waters were most corrosive over the first 3 weeks of the study and three of the four most corrosive over the last three. This is in contrast to the results of Phase I in which the CA-based waters tended to be associated with greater stagnation water concentrations. However, it should be borne in mind that Phase I elbows were only 2% lead while Phase II meters were 8% lead. The possible effect of this difference on water lead will be discussed below along with comments about why in Phase II the highest lead level was a spike to over 1000 ppb extracted by FSA + 2 ppm CL water after initial meter conditioning with 1 ppm CL water.

5. Discussion of findings

5.1. Consistency of test results

Meter-to-meter difference in stagnation water lead values was very low on any one day and for virtually all test waters. The few instances where above median meter-to-meter variation was found, the median lead values were also on the high side. In other words, meter-to-meter differences in water lead values were about the same percent of median water lead values for most days and water formulas.

Day-to-day lead values within any week were often consistent or with a trend up or down. Such trends might be explained by loss of volatiles from a given batch of water without make-up under laboratory conditions that would not occur in a water plant where composition is in constant make-up mode. For example, excess NH alone might gradually decline, with one effect on lead extraction and excess CL alone might decline with the same or another effect. Given the fact that CL and NH combine to form chloramine, neither NH nor CL would be lost, which would have its own effect.

On the other hand, notable shifts in lead extraction occurred when a fresh batch of treatment water was prepared, typically between weeks. The most extreme shift in the entire experiment occurred when meters were first exposed to CL + FSA after having been conditioned to 1% CL/DI water. Along with the 17 other meters, the lead released by this group of 3 during the conditioning process had reached around 100 ppb. The first day these meters were exposed to CL + FSA following meter conditioning, lead concentration leaped to 800 ppb and increased to over 1000 ppb by the third day.

The second week batch of FSA + CL water was made up with the same FSA composition as the first batch, but the CL charge was different, in that sodium hypochlorite solution had been adjusted to compensate for change in the CL stock solution over time. The first day of the second week, water lead was down to 100 ppb, the same level as that at the end of the conditioning period. Thereafter, lead levels dropped in the

next 2 weeks and settled into a consistent 50 ppb day-to-day and week-to-week.

A crucial fact about the make-ups of the first and second FSA + CL batches is that the first had a pH of 7.56 with 1 g sodium bicarbonate added while the second had a pH of 7.30 with 6 g of sodium bicarbonate added. It is doubtful that this pH difference accounts for first week lead starting on day 1 at 800 ppb, increasing to over 1000 by the third day. The fact that one sixth the amount of base in batch 1 than in batch 2 produced a higher pH in batch 1 than that of batch 2 suggests fluosilicate dissociation status was not the same for both batches.

There also could have been serious error in batch preparation or analytical technique. But these explanations are at odds with the consistency of lead extraction measured during conditioning and very low meter-to-meter variation illustrated in Figs. 6 and 7. Neither human error in batch preparation, nor flaws in instrument performance can account for the high first day lead and upward trend that followed. A better explanation is that the combined action of CL and FSA in the first water batch started out very efficient and improved in the next 2 days. This would be consistent with release of particulate lead from the brass alloy in the first week leaving the remaining potentially mobilizable lead shielded from corrosive attack.

A similar, but less dramatic, effect was observed when FSA was added to CA with excess NH. After settling at the 100 ppb level during conditioning, the first day of exposure the test water produced a stagnation lead level of 350 ppb. Then, in this case, without any change in batch formula, after the first-day 350 ppb spike, the second day lead was 120 ppb, and the third day 100 ppb. Thereafter, for 5 successive weeks, with new batches each week, stagnation lead settled down to a very consistent 40–50 ppb.

On an obviously different scale, the same sensitivity of leaded-brass to corrosion by FSA + CL or FSA + CA can be expected in a water plant. It may not be observed when very tight controls are kept on treatment chemical compositions, but the results reported here are very much like what was found in the DC experience.

Considering the several different additives used in the plant, it is a forgone conclusion that deviations from an ideal dosage of any one additive are inevitable. The important data in this report should, therefore, be treated as providing reasonable confidence, not absolute proof of what would actually occur in a water plant.

6. Conclusions

In the "fluoridation debate" proponents frequently argue that the 1 or 2 ppm of fluoride in drinking water is so trivial that it cannot be a health danger. When one translates the ppm involved into molar concentrations, 2 ppm of fluoride is about twice the concentration of 2 ppm of chloride.

Ironically, the switch from CL to CA for disinfection that was made for health reasons, may have created a high water lead health problem. Published evidence has shown that chloramine used instead of chlorine for water disinfection enhances lead extraction from leaded-brass plumbing devices and faucets. Prior to the present study, no one had looked at brass corrosion by combinations of either chlorine or chloramine with water fluoridating agents. Several factors applicable to such combinations can produce more corrosion than either of the disinfectants or fluoridating agents alone.

One such factor is that fluosilicic acid, the most widely used fluoridating agent, is a good solvent for lead. Another is that chlorine, ammonia, and chloramine are all hostile to copper in that they induce copper stress cracking and/or can dissolve it. A third factor is that ammonia added to chlorine to produce chloramine will also react with fluosilicic acid to produce ammonium fluosilicate, an established solvent for copper alloys.

Besides these chemical factors, the lead in brass is present as nodules, so that any attack on the copper matrix of brass would render particulate lead readily accessible for mobilization. Whatever the exact mechanism may be for the combined effect of CA and fluoridating agents on increased levels of waterborne lead, the fact is that SiFs (FSA and NaFSA), commonly used to fluoridate water, have been associated with elevated blood lead levels in children (Coplan et al., in press; Masters et al., 2000). In a related sense, it was recently found that the North Carolina water systems that use FSA and chloramine are associated with elevated blood lead levels in children (Allegood, 2005; Clabby, 2006; Miranda et al., 2006). EPA has claimed a year-long evaluation they conducted did not find a national problem comparable to that in DC, but EPA also acknowledged the need to update specific areas of the LCR and guidance materials (USEPA, 2006). That ought to include lead from brass (Dudi et al., 2005; Renner, 2006) (see Coplan et al., in press).

Acknowledgment

This paper is dedicated to Dr. Richard P. Maas 1952–2005.

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Confirmation of and explanations for elevated blood lead and other disorders in children exposed to water disinfection and fluoridation chemicals

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Abstract

Silicofluorides (SiFs), fluosilicic acid (FSA) and sodium fluosilicate (NaFSA), are used to fluoridate over 90% of US fluoridated municipal water supplies. Living in communities with silicofluoride treated water (SiFW) is associated with two neurotoxic effects: (1) Prevalence of children with elevated blood lead (PbB > 10 μg/dL) is about double that in non-fluoridated communities (Risk Ratio 2, χ^2 $p < 0.01$). SiFW is associated with serious corrosion of lead-bearing brass plumbing, producing elevated water lead (PbW) at the faucet. New data refute the long-prevailing belief that PbW contributes little to children's blood lead (PbB), it is likely to contribute 50% or more. (2) SiFW has been shown to interfere with cholinergic function. Unlike the fully ionized state of fluoride (F-) in water treated with sodium fluoride (NaFW), the SiF anion, [SiF6]2- in SiFW releases F- in a complicated dissociation process. Small amounts of incompletely dissociated [SiF6]2- or low molecular weight (LMW) silicic acid (SA) oligomers may remain in SiFW. A German PhD study found that SiFW is a more powerful inhibitor of acetylcholinesterase (AChE) than NaFW. It is proposed here that SiFW induces protein mis-folding via a mechanism that would affect polypeptides in general, and explain dental fluorosis, a tooth enamel defect that is not merely "cosmetic" but a "canary in the mine" foretelling other adverse, albeit subtle, health and behavioral effects. Efforts to refute evidence of such effects are analyzed and rebutted. In 1999 and 2000, senior EPA personnel admitted they knew of no health effects studies of SiFs. In 2002 SiFs were nominated for NTP animal testing. In 2006 an NRC Fluoride Study Committee recommended such studies. It is not known at this writing whether any had begun.

Keywords: Elevated blood lead; Silicofluoride; Fluosilicate; Brass corrosion; Enzyme inhibition; Fluorosis

1. Introduction

Chronic ingestion of water bearing 1 ppm of fluoride ion (F^-) from NaF was thought harmless to humans when municipal water fluoridation began in 1945. NaFSA was substituted in 1947 and endorsed in 1950 by the US Public Health Service without prior animal testing because rats grew just as fast, their teeth got as much F^- as from NaF, and a community could save 4 cents per year per resident (McClure, 1950).

FSA (H_2SiF_6) and NaFSA, its sodium salt (Na_2SiF_6), share the $[SiF_6]^{2-}$ anion, a fluoride complex herein called "silicofluoride" (SiF) which dissociates in water, releasing F^-. The dissociation was predicted to be "virtually complete" at 1 ppm of F^- so that SiFW would be "just like" NaF treated water (NaFW). Today, 92% of US fluoridated drinking water is SiFW (CDC, 1993). Senior EPA personnel have found no evidence SiFW was ever tested for adverse health effects (Fox, 1999; Thurnau, 2000). In 2002, SiFs were "nominated" for animal tests (NTP, 2002) that had not begun as of July 2006.

The NRC report, "Fluoride in Drinking Water...A Scientific Review of EPA's Standards" (NRC, 2006) emphasizes the importance of such testing with questions about

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incompletely dissociated $[SiF_6]^{2-}$ end-products in human diets. It recommends study of SiFW of different hardness, mineral content, and silica native to the water, taking into account the reversible equilibrium aspects of $[SiF_6]^{2-}$ dissociation.

Neurotoxic and related effects associated with chronic ingestion of SiFW that have heretofore escaped attention are discussed here.

2. Effects associated with SiFW

2.1. Association of elevated PbB with SiFW

PbB data for 400,000 children (250,000 in Massachusetts, 150,000 in New York State, and 6,000 from NHANES III), consistently showed a statistically significant Risk Ratio around 2 for PbB > 10 μg/dL in SiFW communities compared with either non-fluoridated or NaFW communities. (Masters and Coplan, 1999; Masters et al., 2000) (see Tables 1–5 and Figs. 1 and 2). SiFW could increase PbB by (a) exacerbating plumbing corrosion, and/or (b) facilitating

Table 1
Community-Based PbB Parameters in 250,000 Massachusetts Children aged 0–5

WF status	Mean capillary PbB (μg/dL)	Prevalence PbB VB > 10 μg/dL (%)
Non-fluoridated	2.02	1.9
Sodium fluoride used	2.09	1.6
Sodium fluosilicate used	2.66	3.0
Fluosilicic acid used	2.78	2.9

Table 2
Comparison of matched Massachusetts communities

	30 non-fluoridated communities	30 SiF fluoridated communities
Total population	837,300	845,100
Pop children 0–5	57,031	56,446
Children tested (N)	37,310	39,256
VB PbB > 10 μg/dL (n)	283	762
Prevalence rate	283/37,130 = 0.76%	762/39,256 = 1.94%
Risk ratio	1.94/0.76 = 2.55	Chi sq $p < 0.001$

Table 3
Counties in NHANES III by fluoridation status

	County	State	Persons	Percent receiving fluoridated water
Low fluoride: counties: 8, total pop: 1.9 million, % on Fl: 4.0	Nassau	NY	1287348	0.0
	San Bernardino	CA	1418380	0.0
	San Diego	CA	2498016	0.1
	Fresno	CA	667490	0.4
	Ventura	CA	669016	3.9
	Bexar	TX	1185394	4.7
	Los Angeles	CA	8863164	4.8
	Orange	CA	2410556	9.9
Medium fluoride: counties: 8, total pop: 1.1 million, % on Fl: 51.6	Santa Clara	CA	1497577	11.6
	Palm Beach	FL	863518	16.8
	Westchester	NY	874866	27.3
	Maricopa	AZ	2122101	58.1
	Delaware	PA	547651	58.2
	Harris	TX	2818199	65.6
	Oakland	MI	1083592	68.6
	Middlesex	MA	1398468	77.1
High fluoride: counties: 19, total pop: 3 million, % on Fl: 97.2	King	WA	1507319	81.0
	El Paso	TX	591610	81.1
	Tarrant	TX	1170103	86.4
	Hamilton	OH	866228	88.0
	Dade	FL	1937094	96.3
	Cook	IL	5105067	98.5
	Dallas	TX	1852810	99.0
	Kings	NY	2300664	100.0
	New York	NY	1487536	100.0
	Philadelphia	PA	1585577	100.0
	Queens	NY	1951598	100.0
	Duval	FL	672971	100.0
	Wayne	MI	2111687	100.0
	Cuyahoga	OH	1412140	100.0
	Providence	RI	596270	100.0
	Alameda	CA	1279182	100.0
	Allegheny	PA	1336449	100.0
	Erie	NY	968532	100.0
	St. Louis	MO	993529	100.0

Table 4
NHANES III study participants by race and silicofluoride exposure ages 3–17 with PbB test and poverty/income information

	Number	Black-Non-Hisp	White Non-Hisp	Mexican-American
Total	6645	2375	1876	2394
Probability of SiF Exposure				
95%	1361	754	320	287
50/50	544	164	183	197
5%	1085	137	111	837
Unknown	3655	1320	1262	1037

Table 5
NYS Study ORs as Function of Criterion for Elevated PbB

Criterion (μg/dL)	Exposed to SiF		Not exposed to SiF		OR
	N	Prev %	*N*	Prev%	
5	4991	57.5	3,710	38.8	2.1
10	1786	20.6	673	7.0	3.4
15	857	9.9	262	2.7	3.9
20	458	5.2	137	1.4	3.8

lead transport across the gut/blood membrane, whatever the lead source might be.

2.2. Elevated PbW effect on PbB

EPA and CDC have estimated that 14–20% of PbB for all ages is due to PbW (EPA, 1993). This estimate is too low for children, considering reduced exposure to other lead sources and new data on PbW due to brass plumbing corrosion. Besides, children ingest more water per pound of body weight than adults, and absorb a higher fraction of any lead in that water (Houk et al., 1989; CDC, 2004). Newborns are the most seriously affected (White, 2004).

A fetus will get lead from placental blood due to PbW in a mother's current diet or released from her bones absorbed many years earlier (Gomaa et al., 2002). The worst and most likely irreversible lead damage is done to a fetus in the first trimester (Hu et al., 2006).

A breast-fed infant may ingest lead in its mother's milk; a formula-fed infant's diet may include tap-water lead, and any child may ingest PbW in home- or commercially-prepared foods. PbW contributes at least half of an infant's PbB (Houk et al., 1989; Skipton and Hay, 2006). PbW should be considered as important as lead paint and low calcium intake (Goyer, 1996) as a factor in PbB, particularly for Blacks (Enattah et al., 2002).

2.3. Plumbing corrosion and PbW

Plumbing corrosion has been studied under many conditions other than fluoridation (EPA, 1993; Lyttle and Schock, 1996). Recent high PbW episodes prompted a University of North Carolina Environmental Quality Institute (EQI) study which found high PbW from brass corrosion by water treated with fluoridating agents combined with chlorine or chloramine disinfecting agents (Maas et al.,in press). (See companion article in this issue and Table 6 here). This comports with field experience of high PbW in Washington DC (Edwards, 2004) and Boston (Estes-Smargiassi, 2005) pointing to brass corrosion as the problem. The effect of chloramine alone on lead corrosion has been explored (Switzer et al., 2006) as well as the effect of chloramine combined with fluoridating agents on blood lead (Miranda et al., 2007).

CDC's Fluoridation Manual says SiFs may enhance plumbing corrosion but can be mitigated by the simple pH control used for chlorine or alum (Reeves, 1994). However, this doesn't account for pH effects of SiF dissociation. The same error was made in "proving" SiFW and NaFW are identical (Jackson et al.,2004). Neutralizing the initial two protons released from H_2SiF_6 still leaves 4 more to neutralize when the 6 F^- ions are released by dissociation of $[SiF_6]^{2-}$.

CDC also recommends a 60 second flush of water that has been stagnant a few hours (CDC, 2003). This is now considered insufficient in many common situations (eg schools and multi-story apartment buildings). In the wake of Washington DC's water lead debacle of 2001–2004, CDC also dismissed as "much ado about nothing" public concern about high PbB from ingesting PbW as high as 300 ppb (Renner, 2006). EPA's Lead/Copper Rule sets 15 ppb as a limit on PbW and specifies 60 second flushing of water stagnated for several hours (EPA,

Table 6
Maas 2007 Water Lead Data Illustrating Enhanced Brass Corrosion By Combinations of Water Fluoridation and Disinfection Agents

Agent[a] Combinations	Water Lead (ppb) Found after overnight dwell[b] during 6 weeks of flow-through exposure			
	Mean lead ppb for		Spike increment due to	
	All 18 samples	Last 6 samples	ppb	Added F agent
(a) CA + FSA	60	39	300	
(b) CA, extra NH_3 + FSA	61	98	150	b/c 2.1, 2.8, 3.0
(c) CA, extra NH_3	29	35	50	
(d) CA, extra NH_3 + NaF	36	51	100	d/c 1.2, 1.5, 2.0
(e) Cl_2 + FSA	202	45	1000	e/g 1.8, 0.5 5.3
(f) Cl_2 + NaF	151	107	210	f/g 1.3 1.2 1.1
(g) Cl_2 alone	115	88	190	

[a] Agents added as 2 ppm: CA: chloramine; Cl_2: chlorine; FSA: fluosilicic acid; NH_3: ammonia in solution; NaF: sodium fluoride in solution; pH held at 7.2–7.5.
[b] Three samples taken per week for six weeks.

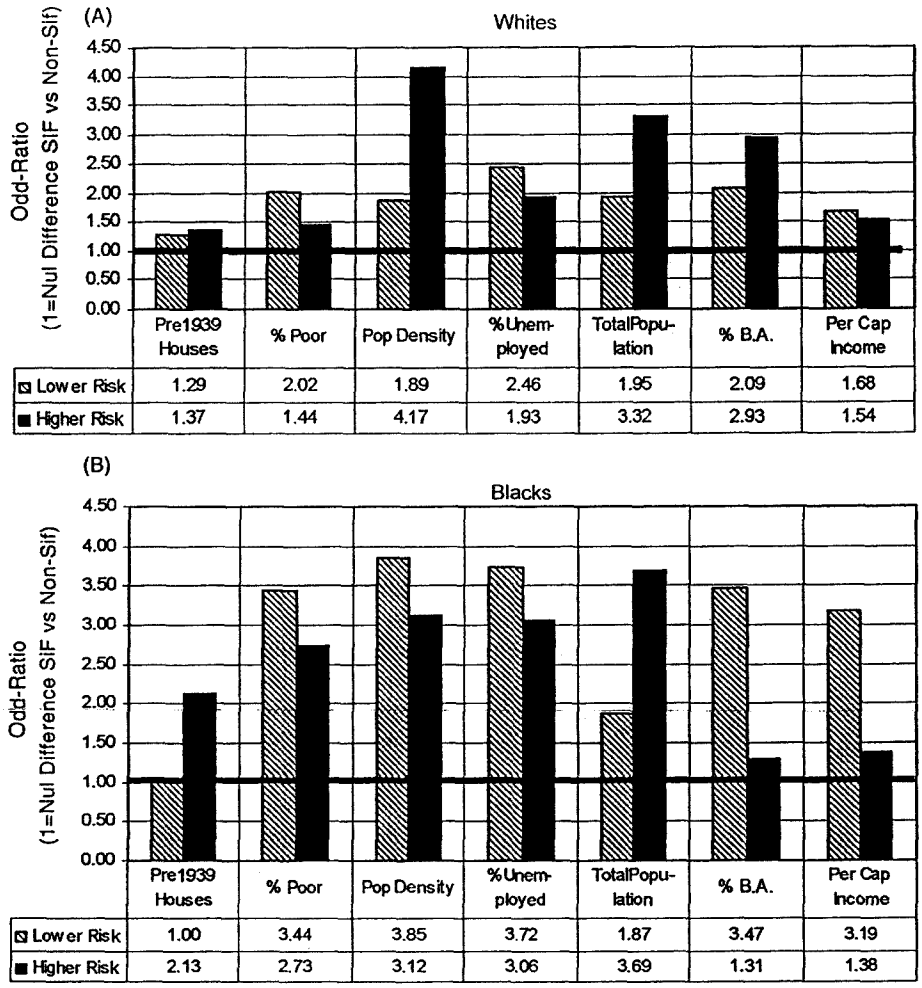


Fig. 1. Odds Ratios for VB > 10 μg/dL Comparing Children in 105 New York State Communities (pop. 15,000–75,000) With and Without SiF Treated Water Controlling for 7 Risk Factors for High Blood Lead.

2002; EPA, 2005). This might suffice for a ground floor faucet but not for upper floors of "triple-deckers" or apartment buildings (EPA, 1993). Several minutes may still be inadequate for some conditions. High PbW would explain association of elevated PbB with population density due to multi-story buildings. Similar considerations would apply to school PbW (Arizona, 2004; EPA, 2005; Karr et al.,2004).

Table 7
Average daily fluoride metabolism experience of young male rats (derived by MJ Coplan from Kick et al Data Cited by McClure)

	Fluoride Source		Na_2SiF_6/NaF
	Na_2SiF_6 (mgm)	NaF (mgm)	Ratio
Average Daily:			
(a) Fluoride "Dosage"	4.00	3.91	1.02
(b) Fluoride Absorbed (c + f)	2.60	1.75	1.49
(c) Total Fluoride Retained	1.22	1.27	0.96
(d) Total Fluoride Excreted	2.78	2.64	1.05
(e) Fluoride Excreted in Feces	1.40	2.16	0.63
(f) Fluoride Excreted in Urine	1.38	0.48	2.67

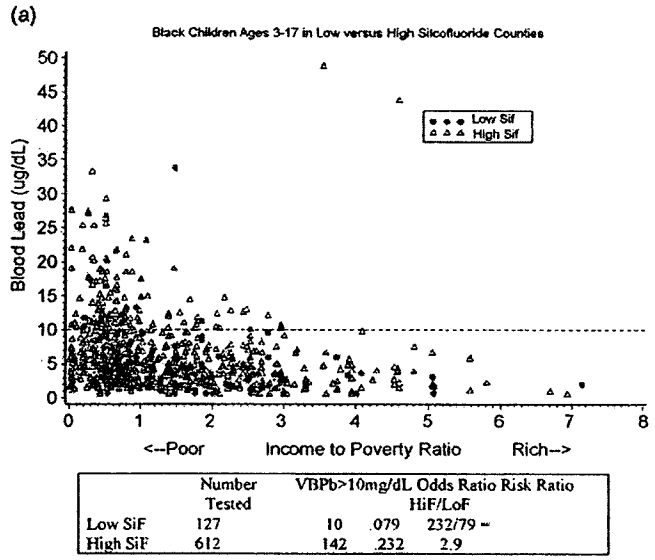
2.4. *Metabolism of ingested fluoride*

The 1950 US PHS endorsement of NaFSA noted that more F^- from SiFW was eliminated in urine of young male rats than from NaFW (Kick et al., 1935) (Table 7). A related metabolic effect was observed when urine of boys and men was collected (separately) in two communities from the start of fluoridation, one using NaF the other SiF. Urine F^- level was tracked until equilibrium with ingested F^- was reached. This occurred later for boys than men with no time difference for men exposed to NaF or SiF. However, a longer time to reach equilibrium was required for boys drinking SiFW compared with those drinking NaFW (Zipkin et al., 1956).

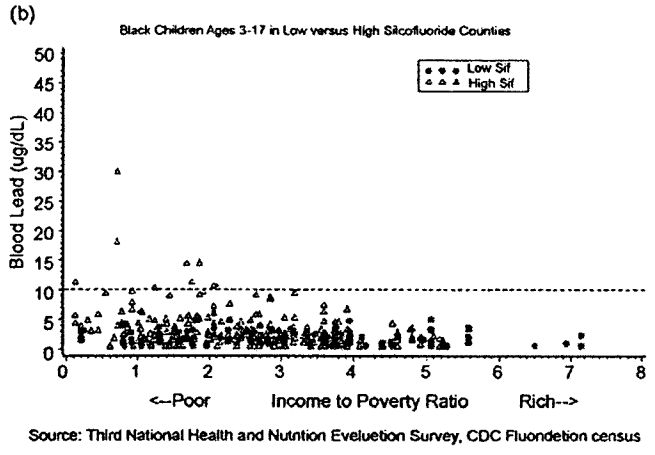
Kick et al., 1935; Zipkin et al., 1956 results imply that soft tissue of young male mammals suffer more exposure to F^- from SiFW than NaFW. This is consistent with reversible equilibrium chemistry since the fluoride-bearing dissociation species in SiFW should undergo re-association at stomach pH around 2, regenerating membrane-permeable fluorinated silicic acid (SA) derivatives that would not be produced from NaFW.

Table 8
Degree of complex dissociation at physiological conditions, pH 7.4, $T = 37$ °C

Complex used	Concentration [10^{-4}M] pH 7.4; $T = 37$ °C	Level of hydrolysis at saturation	Number of F ions separated per complex
$MgSiF_6$	5.7	0.593	4
$MgSiF_6$	1.01	0.622	4
$MgSiF_6$	0.232	0.625	4
$MgSiF_6$	0.116	0.630	4
K_2GeF_6	1.82	0.83	5
K_2SnF_6	1.42	1.00	6
Na_3AlF_6	1.76	0.659	4
KPF_6	1.67	0.0209	0
KBF_4	2.34	0.068	0



	Number Tested	VBPb>10mg/dL	Odds Ratio	Risk Ratio HiF/LoF
Low SiF	127	10	.079	232/79 =
High SiF	612	142	.232	2.9



Source: Third National Health and Nutrition Evaluation Survey, CDC Fluoridation census

	Number Tested	VBPb>10mg/dL	Odds Ratio	Risk Ratio HiF/LoF
Low SiF	110	0	.00	032/.00 =
High SiF	310	10	.032	infinite

Fig. 2. (a) Blood Lead Versus Poverty to Income Ratio of NHANESIII Children. Black Children Ages 3–17 in Low versus High Silicofluoride Counties. (b) Blood Lead Versus Poverty to Income Ratio of NHANESIII Children. White Children Ages 3–17 in Low versus High SilioDfluoride Counties.

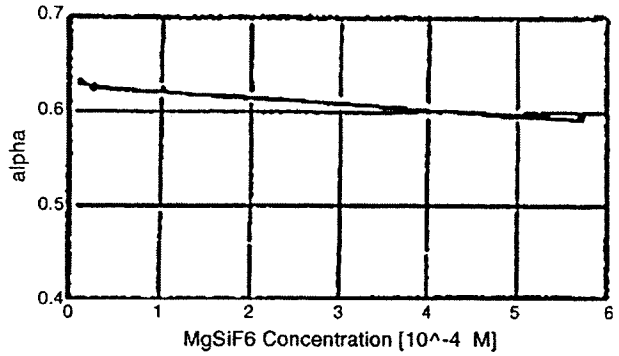


Fig. 3. Level of Hydrolysis of SiF_6^{2-} as a Function of Concentration (Westendorf Figure 17).

Kick's rat fluoride metabolism data were recently disputed (Whitford and Johnson, 2003, see also MCG Anon). Female rats were fed NaFW or SiFW for 4 months before their urine was tested. No difference was found for fluoride intake, excretion, and retention. However, Whitford's rat urine was collected from adult females and Kick's from juvenile males. Whitford himself had shown that children metabolize fluoride differently than adults (Whitford, 1999). It is also known that girls' boney tissue matures earlier than boys' (Gajewska et al., 2005).

Both acute short-term high level F^- and chronic low level F^- exposures impair calcium-dependent processes, including those associated with neural as well as kidney functions (Borke and Whitford, 1999). This is consistent with the damage found in squirrel monkeys exposed to SiFW at 1–5 ppm F^- for

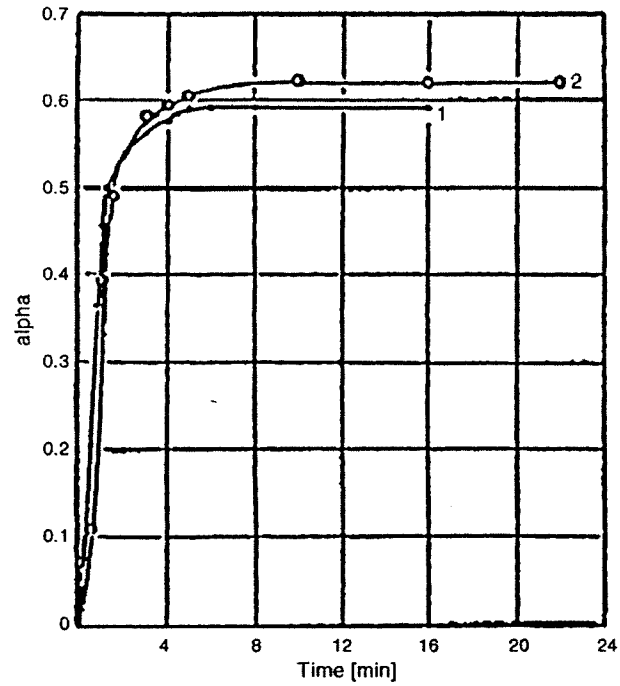


Fig. 4. Dependence of the Level of Hydrolysis of SiF_6^{2-} on Time. (Westendorf Figure 16). (1) 6.45 ppm of F^- (5.7×10^{-4} M of $MgSiF_6$). (2) 1.14 ppm of F^- (1.01×10^{-4} M of $MgSiF_6$). Hydrolysis is initially rapid; no more change occurred after 15 minutes.

18 months (Manocha et al., 1975). Early development of rat kidney is very sensitive to toxic insult (Alexander et al., 1997) with hypertension later in life a probable outcome of damage inflicted *in utero* (Alexander, 2006). The same F^- levels impairing rat kidney phospholipid cell membranes is associated with dental fluorosis (Guan et al., 2000). Similar damage to cell membranes results from prolonged low level exposure to reactive oxygen species produced by internal radiation, according to the "Petkau Effect" (Graueb, 1994).

2.5. Caries/Fluorosis/PbW/SiFW linkage

Contrary to common belief, fluorotic enamel *per se* does not prevent caries (Wondwossen et al., 2004). NRC, 2006 identifies fluorosis damage to tooth enamel as a cause of caries in about 10% of children living today where water F^- exceeds 3 ppm. Twenty years ago, the National Institute for Dental Research warned that fluorosis from natural F^- over 2 ppm could lead to caries (Heifetz et al., 1988). The CDC recently recommended that even very low levels of fluoride in infant formula should be avoided (CDC, 2006). Caries has also been associated with elevated PbB (Moss et al., 1999) and in newborn mice with lead in the dam's diet (Watson et al., 1997).

Before water fluoridation began (1945), 10–12% of children drinking 1 ppm F^- in natural fluoridated water exhibited dental fluorosis, mostly mild (Dean, 1938). In 1993, half the children in some fluoridated areas had fluorosis, 14% moderate-to-severe and some severe (NRC, 1993). Fluoridation advocates claim fluorosis is only "cosmetic" but NRC, 2006 says severe fluorosis is a "toxic effect that is consistent with prevailing risk assessment definitions of adverse health effects" and drinking water is a major fluoride source.

Dental fluorosis is believed due to inhibition of the enzyme that removes amelogenin after enamel formation in nascent tooth buds (Den Besten, 1986, 1999). Severe fluorosis was reported in India where naturally occurring fluoridated water had "silicon" in it (Anasuya et al.,1996). The silicon couldn't have been metallic or in sand, it had to come from natural SiFW (Ockerse, 1946; Sahlbom and Hinrichsen, 1906). Thus, chronic ingestion of SiFW is a major factor in the linkage of dental fluorosis with fluoridated water.

3. SiFW delivers more than fluoride

3.1. Incomplete dissociation of SiF in SiFW (Westendorf, 1975, 1974a,b)

Fluorides in general inhibit enzyme function. Voluntary and involuntary muscle action is stimulated by acetylcholine (ACh) which is cleaved by the enzyme acetylcholinesterase (AChE) to end the stimulation. Without that, muscle excitation would persist as spasm with potentially lethal effect, as caused by a nerve gas. ACh modulated by AChE also induces saliva flow. Intense salivation is a symptom of fluoride poisoning; less severe fluoride exposure should increase flow of fluoride bearing saliva. With caries prevention in mind, a German PhD study compared inhibition of AChE by NaFW and SiFW (Westendorf, 1975).

Inhibition was measured in cells or as purified products in 37 °C solutions buffered at pH 7.4 or 8.6, to which NaFW and SiFW were added at stoichiometric concentrations as low as 1 ppm F^-. HF from the F^- in NaFW caused "competitive" inhibition; (*ie* it blocked enzyme active sites). SiFW inhibition was more complicated. As measured by ion selective electrode *without* adding "TISAB" (see part B, below), only 2/3rds of the F^- bound in $[SiF_6]^{2-}$ was detected. The balance was present either in partially dissociated $[SiF_6]^{2-}$ such as $[SiF_2(OH)_4]^{2-}$ or in some fluorinated SA species (See Table 8 and Figs. 3 and 4).

Whichever one it was, it caused "non-competitive" inhibition by distorting enzyme shape making active sites inaccessible. Hence, for the same total fluoride in the system, SiFW competitive plus non-competitive AChE inhibition was more powerful than that by NaFW. Westendorf also studied butyrylcholinesterase (BuChE) and got the same result. Unlike AChE, the role of BuChE is not well-defined, but it is important as a scavenger of blood-borne toxins and back-up for AChE in some situations (Cokugras, 2003).

The mechanism for "non-competitive" enzyme inhibition by incomplete SiF dissociation products has broader implications than interference with normal AChE cholinergic activity. SA binds with a wide array of amides (Clark et al., 1957; Coradin and Livage, 2001; Coradin et al., 2005). Evidence of this is that LMW SA oligomers "denature" proteins (Iler, 1979).

When a stream of concentrated FSA or NaFSA enters water, $[SiF_6]^{2-}$ dissociates, releasing SA and F^- in close proximity and F^- catalyzes SA "oligomerization" (Rabinovich and Wood, 1986). Hydrogen bonding sites of linear SA oligomer molecules are spaced apart the same distance as polypeptide backbone repeat units. Properly juxtaposed, they are likely to zip together (Clark and Holt, 1957), creating the "mis-folded proteins" described by Ellis (2002) and Temussi et al. (2003). Protein mis-folding would explain SiFW non-competitive enzyme inhibition as well as other health problems associated with blood-borne polypeptides (Kayed et al.,2004).

3.2. SiF dissociation complexity; analysis for total fluoride

SiF dissociation has been studied for a century (e.g. Hudleston and Bassett, 1921) with little agreement other than that it involves a series of reversible equilibrium steps influenced by temperature and pH. Total fluoride in NaFSA (AWWA, 1994a) and FSA (AWWA, 1994b) is quantitatively assayed at pH 9 at the boil. The $[SiF_6]^{2-}$ anion is sufficiently stable at pH 7 to permit standard base titration of the ordinary hydronium ion acidity of FSA without producing SiF dissociation *per se* (Colton, 1958).

$$2OH^- + H_2SiF_6 \xrightarrow{pH\,7} [SiF_6]^{2-} + H_2O$$

The Ion Selective Electrode (ISE) method to measure ionic F^- in the water plant involves sample dilution with an equal volume of Total Ionic Strength Adjustment Buffer (TISAB).

The 2:1 dilution *per se* causes some dissociation but TISAB also carries a chelating agent to break up fluoride complexes. Other agents control pH at 5.5 (EPA, 1996). None of this is replicated in water plant treatments and certainly not in the digestive tract. Thus, it is unlikely the free F^- ion levels read on an ISE meter faithfully reflect SiF dissociation status in water leaving the plant, at the faucet, or in the digestive tract.

4. EPA's refutation of SiF adverse health effects

4.1. Urbansky and Schock

Without studying data or statistical methodology of the findings summarized in II A, EPA chemists produced an EPA "Work Product" detailing why SiFs are "almost completely" dissociated at 1 ppm $F^-$and can't influence PbB (Urbansky and Schock, 2000). The issue was framed as a question of residual concentration of the fluosilicate ion $[SiF_6]^{2-}$ after hydrolysis which should be predictable from reversible equilibrium thermodynamics and reaction kinetics theory. If theory applied, no $[SiF_6]^{2-}$ would remain in drinking water at equilibrium with 1 ppm of F^-.

However, residual $[SiF_6]^{2-}$, as such, is not the issue. Its total absence does not obviate survival of some fluorine-bearing SiF derivatives. Moreover, "at equilibrium" implies an end state that cannot be predicted without precise knowledge of SiF's *ab initio* status. As much as 30 ppm of silica may already be in raw water (ASTM, 1994) and silicates are sometimes used in the water plant for flocculation. Both of these conditions would influence dissociation end results. This is further confounded by what a phosphate fertilizer expert told an international technical conference of peers (Smith, 1999):

"The chemical formula of fluosilicic acid [FSA] is H_2SiF_6. However, things are not as simple as that due to the fact that rarely is fluosilicic acid present as pure H_2SiF_6. . .there are well reported references to the existence of $H_2SiF_6 \cdot SiF_4$. . . Hereon in this presentation, FSA means a mixture of HF, H_2SiF_6 and $H_2SiF_6 \cdot SiF_4$".

In 2001, EPA research managers concluded it was necessary to clarify SiF dissociation. In 2002, EPA issued a "Request for Assistance" (RFA) inviting research proposals on methods to detect and measure SiF dissociation products. For the benefit of prospective bidders, Urbansky wrote an extensive review of SiF dissociation studies (Urbansky, 2002), in which he concluded that hydroxo-fluoro SiF derivatives could survive in drinking water and opined:

> ". . . it is probably best to stop using qualified expressions such as 'virtually complete' or 'essentially complete' in favor of more rigorous and quantitative descriptions [of SiF dissociation] even if that hinders communication with the lay public."

In connection with a response to questions raised by a Congressional committee (Fox, 1999), an EPA Information Sheet (Donohue, 1999) defined "virtually complete" dissociation of $[SiF_6]^{2-}$ as 99%. This was based on a study that reported 99% dissociation at 1 ppm but it also reported only 95% dissociation at 2 ppm (Crosby, 1969).

At 99% dissociation, six of the 600 fluorine atom originally bound in 100 $[SiF_6]^{2-}$ anions remain bound to silicon in some species. If these fluorines are distributed randomly over 100 silicons, 6% of the 100 pre-dissociation $[SiF_6]^{2-}$ anions remain as some fluorine-bearing species. Without stipulating any particular one, it is reasonable to assume that if that species mobilizes a lead atom it does so1 to1. The atomic weight of lead being about ten times that of fluorine, for each ppm of silicon-bound fluorine, 10 ppm of lead would be mobilized.

With 1 ppm of free F^- and 1% of initial bound F^- remaining bound, 10 ppb (ie 0.01 ppm) of F^- would be in the lead mobilizing species, so 100 ppb of lead would be mobilized. For reasons discussed in Maas et al., in press, many water systems deliver water with 1.5–2 ppm of F^- which should magnify the 100 ppb several-fold. PbW at 100 ppb has been estimated to increase an infant's PbB by 15 μg/dL (White, 2004) and school age children's by 2.5 μg/dL (Karr et al.,2004). A recent mega-study (Lanphear et al., 2005) reports that PbB at 2.5 μg/dL reduces a child's IQ measurably.

4.2. Morris/Finney

NRC, 2006 mentions "Morris 2004," work funded pursuant to the EPA, 2002 RFA cited above, to be conducted in the laboratory of a Raman spectroscopy expert. The mission was to find better ways to quantify SiF dissociation end-products; results are reported in Finney et al., 2006. Although the EPA RFA did not ask for assistance in epidemiology or enzyme inhibition, the introduction of Finney et al., 2006 suggests another agenda, namely to prove that SiFW does not pose a public health danger.

The EPA contracted for work to be performed in the laboratory of an expert in Raman spectroscopy, but the published report does not mention the use of Raman spectroscopy. Instead, ^{19}F NMR spectroscopy was employed but it could not detect SiF hydrolysis intermediates because SA oligomers formed and interfered. Another approach was tried, FSA was added incrementally to buffered solutions and pH change, if any, measured.

Based on these data, Finney/Morris challenge Westendorf's enzyme inhibition results, claiming that added FSA lowered pH enough at 1 ppm F^- to explain the AChE inhibition he attributed to SiF derivatives. The problem is that, while their graph shows a line plotted from theory predicting pH shifts, the actual data points show no effect on pH from adding SiF at 1–5 ppm F^- and only a fraction of 0.1 pH unit at 50 ppm.

Finney/Morris had an easier way to refute Westendorf. They could have tried his way of measuring F^- released by $[SiF_6]^{2-}$ without the use of TISAB to see whether 67% dissociation was correct or not. They didn't, but in the course of their NMR experiments, they had inadvertently confirmed the formation of SA oligomers during $[SiF_6]^{2-}$ disociation.

Finney et al., 2006Finney, 2006 says Westendorf studied "effects of fluoride and hydrofluoric acid on AChE inhibition". Hydrofluoric acid is hydrogen fluoride dissolved in water, the

inhibitor derived from NaF. Unless the word "fluoride" was meant to denote $[SiF_6]^{2-}$, the phrase quoted above is equivalent to saying Westendorf studied "effects of hydrogen fluoride and hydrogen fluoride on AChE inhibition".

To summarize: Morris and Finney (a) misinterpreted Westendorf's experimental results; (b) didn't try to measure free F^- by ISE without TISAB to break up fluoride complexes, as Westendorf did; (c) didn't measure SiF derivatives by Raman spectroscopy; (d) tried NMR spectroscopy without success; and (e) measured pH as a secondary attribute of SiF dissociation, producing data that do not support their claims about AChE inhibition.

5. CDC's dismissal of an SiF/PbB linkage

CDC funded a former employee to either verify or refute Section 2.1 findings. Results first appeared as an abstract (Macek et al., 2003), the full report is published in Environmental Health Perspectives (Macek et al., 2006). NHANES III provided children's PbB data and the CDC Fluoridation Census (CDC, 1993) provided residence community fluoridation status (WFS). PbB data were grouped by WFS and housing age in four categories (See Appendix, Macek Table A.1). Other variables were similar to those underlying Section 2.1 analyses which Macek rejects on three counts:

(1) Macek says Section 2.1 analyses lacked data on covariates at the individual level. However, for the 150,000 NY State children (see Fig. 1A and B) Masters et al., 2000 says:

> "Controlling at the individual level for covariates usually associated with lead uptake, elevated blood lead was statistically significant with $p < 0.001$ and Hi/Low risk ratios in the range of 1.5–2.0 depending on age and race".

(2) Macek says Section 2.1 analyses used unclear sampling methods. However, the data for Massachusetts children were collected under State regulations by qualified agencies. Overall ascertainment rate was over 60% with 80% in most large communities (Masters and Coplan, 1999). The data were considered satisfactory by State health authorities for other epidemiologic analyses by Dartmouth-Hitchcock Hospital staff with partial CDC funding (Bailey et al., 1994; Sargent et al., 1995). Data for New York State children provided by the State Health Department for individuals included race, age, and poverty status. SiF exposure was determined from CDC information about community fluoridation status. The study was as close to case/control as possible (Masters et al., 2000). Moreover, the NHANES III data for the Section 2.1 analyses were collected the same way Macek's NHANES III data were collected.

(3) Macek objects to Section 2.1 analyses using skewed untransformed data to compare community mean PbB values. However, using log transformed, less skewed data to find a central tendency does injustice to worst case children. Suppressing the statistical effect of their high outlier PbB values makes it seem that the risk of elevated PbB where these children reside is closer to the norm for other communities. The CDC recently pledged "intensified efforts to target areas at highest risk, evaluate preventive measures, and improve the quality of surveillance data" (CDC Sept 12, 2003). Making comparisons based on geometric mean data does not foster that objective.

While Macek et al., 2006 claims its results are based on a superior analytic approach, it could not refute or confirm the results summarized in II A. The probable reason for this ambiguous outcome can be explained as follows:

(a) Macek used 5 μg/dL rather than 10 μg/dL as the criterion for elevated PbB. This downshifted OR values comparing the same two populations (Table 9). An OR based on 10 μg/dL can be 50% greater than if 5 μg/dL is used. Fig. 2a and b demonstrate how skewed untransformed PbB data can be used more effectively than GM values to reveal PbB problem populations. This isn't meant to suggest that PbB 5 μg/dL is acceptable for individuals. Rather, it means that using 5 μg/dL as an action trigger for alleviating community elevated PbB makes it easier to ignore the effect of PbW.

(b) While referring to the lead leaching capacity of "silicofluoride compounds" as a single class, Macek treated FSA and NaFSA as different SiF classes for statistical analysis. There was no reason to analyze PbB data for children drinking FSA treated water separately from those drinking NaFSA treated water. This produced two smaller groups of children exposed to SiFW. The OR confidence intervals in these smaller groups had to have broader band-widths with lower bounds near 1.0, based solely on an arbitrary statistical artifact.

Table 9
Blood lead data used by Macek et al., 2006 re-analyzed, confirming an association of silicofluoride with elevated blood lead for children aged 1–16 in NHANES III database

WF status	Number sampled	Number with[a] PbB > 5 μg/dL	Percentage with PbB > 5 μg/dL (%)
SiF treated[b]	3170	524	16.5
All other[c] known	4004	568	14.2

Odds Ratio: 1.20, Wald Chi-Square p-value: 0.006. Wald 95% Confidence Interval: (1.05, 1.36).

[a] The standard criterion for elevated blood lead is >10 μg/dL; an OR of 1.2 found using >5 μg/dL predicts an OR greater then 1.2 using >10 μg/dL as the criterion.

[b] Fluosilicic acid and sodium silicofluoride combined.

[c] Natural, NaF treated, and non-fluoridated combined.

(c) Macek used three dwelling age categories when two would have sufficed. A cut-off of 1940 or 1950 could have determined "older housing" as a risk of elevated PbB due to lead plumbing or lead paint. Data for pre-1946 housing were actually available and could have been used to compare with the others, rather than "1974-present".
(d) Macek required 90% of a county be fluoridated with a single agent (or none) for a child living there to be assigned any fluoridation status. Many children could arbitrarily remain unassigned in a county less than 90% fluoridated by either FSA or NaFSA alone when combined they could comprise 90% or more getting SiFW.

These flaws notwithstanding, Macek's raw data (see Appendix A) can be properly analyzed, using NHANES III weighting, to show that SiFW is associated with higher PbB levels than where other WFS prevails. For each housing age, the percent of children with PbB > 5 ug/dL multiplied by the number of children tested from any category gives the likely number of children with elevated PbB in a random sampling of that category. Since "Unknown/mixed" fluoridation category provides no data on SiF effects, it should be excluded.

PbB data for children in communities delivering NaFSA and FSA treated water combined as one SiF group and summed across all housing ages should be compared with PbB data for all other children, also summed across housing ages. The comparison in Table 9 shows that children getting SiFW had statistically significant higher PbB than all others combined.

The OR of 1.2 might seem inconsequential but it is based on 5 μg/dL, not the usual 10 μg/dL criterion. Since raising the cut-off of a continuous variable also raises an observed OR, if 1.2 is valid for 5 μg/dL the OR for 10 μg/dL would be meaningfully higher, possibly 2 or greater.

While Macek data analysis could neither confirm nor refute a PbB effect of SiF treated water, the same data does confirm that association when properly analyzed. On that count alone, under the Precautionary Principle, the two CDC policy positions embodied in the following quote are questionable, but Maas et al., in press also provides compelling evidence of the linkage between SiFW and PbB "demonstrated by additional research". Macek's policy position is not supported by the facts.

> "Efforts to decrease exposure to lead among children by targeting prevention efforts at high-risk communities and/or populations as well as efforts to prevent dental caries via the use of fluoridated drinking water should continue unless a causal impact of certain fluoridation methods on PbB concentration is demonstrated by additional research." (Macek et al., 2006).

6. Conclusions

The effect of PbW on elevated PbB in children has been underestimated on several counts: (a) a fetus *in utero* is exposed to lead in the mother's blood released from her bones and/or ingested with her food and drink; (b) a newborn ingests more water per pound of body weight than an adult; (c) all children absorb more of the lead they ingest than adults; (d) their developmental state renders them more susceptible to neurotoxic damage; (e) lead in drinking water extracted from brass plumbing fixtures by combinations of disinfection and fluoridation chemicals is more important than previously thought and has not had the attention it deserves.

The role of silicofluorides with significant capacity for enzyme inhibition and other bio-activity adverse to neural health, among other problems, has not been properly explored. On the other hand, several studies designed to rebut this conclusion not only fail to do so, they provide evidence of its validity.

It is proposed here that geometric mean values for community based blood lead levels obscure real problem populations by suppressing high side "outlier" cases that would otherwise be evident in simple scatter diagrams of non-transformed data for individuals.

Finally, an NRC Committee appointed in 2003 to review MCLG for fluoride (a) noted the widespread use of

Table A.1
Prevalence and adjusted odds of an elevated PbB concentration at the 5-μg/dL cut-off for U.S. children 1–16 years of age, by water fluoridation method and year in which dwelling was built, 1988–1934 (n = 9,477)[a]

Water fluoridation method[b]	Before 1946			1946–1973			1974-Present			Unknown		
	No.	Percent[c]	OR (95% CI)[d]	No.	Percent	OR (95% CI)	No	Percent	OR (95% CD	No.	Percent	OR (95%CI)
Unknown/mixed status	473	24.7	0.9 (0.4–1.9)	837	11.4	1.1(0.4–2.7)	674	8.3	1.2 (0.5–3.2)	319	21.9	3.8 (2.0–7.0)
Sodium silicofluoride	141	20.7[e]	0.9 (0.3–2.8)	420	16.8	0.8 (0.3–2.5)	289	6.5[e]	1.0 (0.4–2.5)	71	30.1	2.8 (0.8–9.8)
Hydrofluosilicic acid	448	30.1	1.2 (0.6–2.5)	839	14.7	1.4 (0.7–2.9)	605	5.4	1.7 (0.6–4.3)	257	24.7	5.3 (2.7–10.5)
Sodium fluoride	78	20.9	0.8 (0.3–1.7)	127	7.6[e]	1.5 (0.4–5.3)	81	6.0[e]	0.6 (0.1–4.6)	60	6.6[e]	1.0 (0.3–3.6)
Natural fluoride	113	19.4	0.3 (0.1–01.6)	419	17.3	1.5 (0.7–3.2)	413	7.3[e]	1.1 (0.3–3.8)	182	16.6	1.0 (0.4–2.2)
No fluoride	307	26.4	Reference	1176	16.0	Reference	707	6.4	Reference	341	18.4	Reference
Adjusted Wald-F p-value			<0.01			0.76			0.76			<0.01

[a] From the Third National Health and Nutrition Examination Survey (1988–1994) and 1932 Fluoridation Census.
[b] Weighted to reflect the civilian noninstitutionalized population of the United States; persons with unknown blood lead levels were excluded from analysis.
[c] Percentage of the population with an elevated blood lead concentration (≥5 μg/dL).
[d] Adjusted OR of an elevated blood lead concentration at the 5-μg/dL cut-off, controlling for age, sex, race/ethnicity, poverty status, urbanicity, and duration of residence.
[e] Does not meet the standard for statistical reliability.

silicofluorides in its March 22, 2006 report; (b) warned that a 4 ppm F^- MCLG was not protective of human health as regards dental and skeletal fluorosis; (c) included neurotoxic and behavioral issues in its studies; and (d) reinforced the need for the NTP to do animal tests of SiFs that have been on its "nomination" agenda since 2002.

Appendix A

Table A.1.

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Elevated Blood Lead in Young Children Due to Lead-Contaminated Drinking Water: Washington, DC, 2001–2004

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Incidence of EBL (blood lead ≥10 μg/dL) for children aged ≤1.3 years in Washington, DC increased more than 4 times comparing 2001–2003 when lead in water was high versus 2000 when lead in water was low. The incidence of EBL was highly correlated (R^2 = 0.81) to 90th percentile lead in water lead levels (WLLs) from 2000 to 2007 for children aged ≤1.3 years. The risk of exposure to high water lead levels varied markedly in different neighborhoods of the city. For children aged ≤30 months there were not strong correlations between WLLs and EBL, when analyzed for the city as a whole. However, the incidence of EBL increased 2.4 times in high-risk neighborhoods, increased 1.12 times in moderate-risk neighborhoods, and decreased in low-risk neighborhoods comparing 2003 to 2000. The incidence of EBL for children aged ≤30 months also deviated from national trends in a manner that was highly correlated with 90th percentile lead in water levels from 2000 to 2007 (R^2 = 0.83) in the high-risk neighborhoods. These effects are consistent with predictions based on biokinetic models and prior research.

Introduction

The Washington, DC "lead in drinking water crisis" was triggered by a change in disinfectant from free chlorine to chloramine in November 2000 (*1*). The switch in disinfectant reduced the concentration of potential carcinogens (a byproduct of chlorine disinfection) to levels below those specified by the U.S. Environmental Protection Agency (EPA). However, the chloramine also altered the water chemistry and unexpectedly caused lead to leach from lead service line pipes (*1*, *2*) and other plumbing materials such as leaded brass and solder (*1*). The resulting contamination affected water lead levels (WLLs) in homes throughout the city.

Two previous studies of blood lead levels (BLLs) relative to the high WLLs in Washington, DC have been published (*3*, *4*). While the high WLLs appeared to have some impact on the incidence of BLLs ≥ 5 μg/dL (*3*), no evidence was found of increased incidence over the 10 μg/dL level of concern set by the Centers for Disease Control and Prevention (CDC) for children aged <6 years. Blood lead levels exceeding the CDC level of concern are termed "elevated blood lead" (EBL) in this work.

A close examination of the two prior studies reveals noteworthy limitations. Neither study focused on infants, who are most vulnerable to harm from lead in water (*5*–*7*) due to their small body weight and heavy reliance on water as a major component of their diet in the case of infants using reconstituted formula. Moreover, both studies lumped all the blood lead data for Washington, DC together, an approach that can "mask disparities among communities and camouflage pockets of high risk" relative to smaller area analysis at the neighborhood or zip code level (*8*). This research addressed these limitations.

Methodology and Data

Environmental Data. *Water Lead Data.* Measurements of "total lead (*9*)" in potable water were collected by the local water utility using EPA-approved methodology. A "first draw" sample refers to a 1 L sample collected from a tap after greater than 6 h holding time in the household plumbing. After first draw samples are collected, water is flushed for a short time period (typically 30 s to 5 min) and a 1 L "second draw" sample is collected.

Two data sets of potable water lead concentrations were used throughout this research. Data on WLLs in homes with lead pipe during 2003 were collected by the local water utility from over 6000 Washington, DC homes with lead service line pipe. The WLL EPA Monitoring Data (2000–2007) were collected by the water utility specifically for compliance with EPA regulations. Compliance is determined by using the "90th percentile lead," which is the 90th percentile of the cumulative distribution of first draw lead samples collected within a given time period. The monitoring data were reorganized into calendar year time periods for which corresponding blood lead measurements were compiled. For example, the official 2002 EPA monitoring round at the utility included water samples collected between July 1, 2001 and June 30, 2002. The samples collected between July 1, 2001 and December 31, 2001 from that round were used in calculations of the 90th percentile WLLs for the second half of 2001. The remaining water samples from that round were included in calculations of 90th percentile WLLs for calendar year 2002. Several audits have been conducted on the utility's EPA monitoring data (*10*), and trends in 90th percentile lead used in this study are not strongly impacted by remaining unresolved errors in the data.

Lead Pipes by Zip Code and other Demographic Data. The number of lead pipes in each zip code was determined using a database provided by the CDC (*3*). Demographic data within each zip code were obtained from the U.S. Census.

Identification of Sensitive Population. *Predicted Impact of WLLs on BLLs.* In April 2004 the US EPA National Center for Environmental Assessment (NCEA) modeled the impact of high WLLs on the BLLs of children in the city (See Supporting Information Reports 1–3). The NCEA results and additional assumptions were used to make predictions of EBL incidence for children who had consumed formula reconstituted with tap water during their first year of life, and children aged 1–6 years who did not consume formula but drank tap water (see Supporting Information 1). A one-year-old infant living in a Washington, DC home with lead service line pipe and consuming formula made from tap water was predicted to have a 21% likelihood of EBL in 2003. The overall prediction was that there would be 600–700 cases of EBL for children under 6 years of age in 2003 due to the high WLLs. This estimate of 600–700 cases represents only

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The effect of lead on development of motor function was also investigated by Overman et al. (1981). Exposure of rat pups to lead was limited to the period from parturition to weaning and occurred through adulteration of the dams' drinking water with lead acetate (0.01% or 0.1%, equivalent to about 10 or 100 mg/kg/day). Overall, the ability to remain on a rotating rod was significantly impaired ($P < 0.01$) at 0.1%, and tended to be impaired ($0.10 > P > 0.05$) at 0.01%.

Generally similar results are obtained when suckling or young rats are exposed to lead directly (as opposed to indirect exposure via the mother). Tables 2 and 3 summarize some studies of this sort. Cory-Slechta and Thompson (1979) exposed Sprague-Dawley rats to 0.0025%, 0.015%, or 0.05% drinking water solutions of lead (as lead acetate) starting at post-natal day (PND) 20–22. Operant conditioning on a fixed-interval 30-second schedule of reinforcement (food pellet delivered upon the first bar-press occurring at least 30 sec after preceding pellet delivery) began at PND 55–60. Blood lead concentrations measured at approximately PND 150 were reported in graphic form roughly as follows: 0.0025% solution group, 5–10 μg/dL PbB; 0.015% group, 25–30 μg/dL PbB; 0.05% group, 40–45 μg/dL PbB. Subjects exposed to 0.0025% or 0.015% lead solutions showed a "significantly" (no probability value reported) higher median response rate than matched controls during the first 30 sessions of training; response rates continued to be significantly higher over the next 60 sessions for the 0.0025% group and over the next 30 sessions for the 0.015% group (at which points training terminated for each group). Moreover, latencies to the first response in the 30-sec interval (the beginning of the typical "fixed-interval scallop" cumulative response pattern) were significantly shorter in the 0.0025% and 0.015% groups. However, response rates for the group exposed to the 0.05% solution were significantly lower than the control group's rates for the first 40 sessions; correspondingly, response latencies were longer for the highest exposure group.

Other work by Cory-Slechta et al. (1981) repeated the earlier study's exposure regimen (using 0.005% and 0.015% solutions) and examined the effects on another aspect of operant performance. In this study the subjects were required to depress a bar for a specified minimum duration (0.5–3.0 sec.) before a food pellet could be delivered. Intersubject variability increased greatly in the lead-exposed groups. In general, though, treated subjects tended to shorten their response durations ($P = 0.04$ for the 0.005% group; $P = 0.03$ for the 0.015% group). This tendency would contribute toward a reduced rate of reinforcement, which is associated with (and perhaps accounts for) an observed tendency toward increased response latencies in the lead-exposed subjects ($P = 0.04$ in the 0.015% group).

Another form of developmental lag in gross activity around post-natal days 15–18, as measured in an automated activity chamber, was reported by Jason and Kellogg (1981). Rats were incubated on days 2 to 14 with lead at 25 mg/kg (PbB = 50.07 ± 5.33 μg/dL) or 75 mg/kg (PbB = 98.64 ± 9.89 μg/dL). In this case, the effect was a delay in

TABLE 1 (continued)

Exposure protocol	Estimated daily dose[b] (mg/kg/day)	Observed effects	Reference
0.2% $Pb(Ac)_2$ in dams' drinking water PND 0–21	200	1. 23% decrease in NE levels of hypothalamus and striatum 2. increased turnover of NE in brainstem	Goldman et al. (1980)
0.25% $Pb(Ac)_2$ in dams' drinking water PND 0–35	250	Decline in synthesis and turnover of striatal DA	Govoni et al. (1978a)
0.25% $Pb(Ac)_2$ in dams' drinking water PND 0–35	250	Increase in DA synthesis in frontal cortex and nuc. accumbens (10–30% and 35–45%, respectively)	Govoni et al. (1979, 1980), Memo et al. (1980a, 1981)
0.25% $Pb(Ac)_2$ in dams' drinking water PND 0–35	250	1. 50% increase in DA binding to striatal D_2 receptors 2. 33% decrease in DA binding to nuc. accumbens D_2 receptors	Lucchi et al. (1981)
0.25% $Pb(Ac)_2$ in dams' drinking water PND 0–42	250	1. 31% increase in GABA specific binding in cerebellum; 53% increase in GMP activity 2. 36% decrease in GABA-specific binding in striatum; 47% decrease in GMP activity	Govoni et al. (1978b, 1980)
0.25% $Pb(Ac)_2$ in dams' drinking water PND 0–21; 0.004% or 0.25% until PND 42	250	1. 12% and 34% elevation of GABA binding in cerebellum for 0.004% and 0.25%, respectively	Memo et al. (1980b)
0.4% $PbCO_3$ in dams' drinking water PND 0–30	400	Retardation in temporal sequence of hippocampal dendritic development	Alfano and Petit (1982)
0.5% $Pb(Ac)_2$ in dams' drinking water PND 0–2	500	10–15% reduction in number of axons in optic nerve; skewing of fiber diameters to smaller sizes	Tennekoon et al. (1979)

Adapted from USEPA (1984).

TABLE 1
Partial Summary of Studies of Lead Effects During Perinatal Development of Rats[a]

Exposure protocol	Estimated daily dose[b] (mg/kg/day)	Observed effects	Reference
0.004% $Pb(Ac)_2$ in dams' drinking water PND 0–35	4	Decline in synthesis and turnover of striatal DA	Govoni et al. (1979, 1980), Memo et al. (1980a, 1981)
0.02% $PbCl_2$ in dams' drinking water from gestation thru PND 0–21	20	1. Transient 30% reduction in cytochrome content of cerebral cortex 2. possible uncoupling of energy metabolism 3. delays in development of energy metabolism	McCauley and Bull (1978), McCauley et al. (1979), Bull et al. (1979), Bull (1983)
0.1% $Pb(Ac)_2$ in dams' drinking water PND 0–32	100	Significant inhibition in myelin deposition and maturation in whole brain	Stephens and Gerber (1981)
0.2% $PbCl_2$ in dams' drinking water from gestation thru PND 0	200	Less mature synaptic profile in cerebral cortex at PND 15	McCauley and Bull (1978), McCauley et al. (1979)
		30% reduction in synaptic density in cerebral cortex at PND 15 (returned to normal at PND 21)	McCauley et al. (1982)
0.2% $Pb(Ac)_2$ in dams' drinking water PND 0–25	200	15–30% reduction in synaptic profiles in hippocampus	Cambell et al. (1982)
0.2% $Pb(Ac)_2$ in dams' drinking water PND 0–20	200	More rapid appearance and increased severity of MES response	Fox et al. (1978, 1979)
0.2% $Pb(Ac)_2$ in dams' drinking water PND 0–21	200	1. Increased latencies and decreased amplitudes of primary and secondary components of VER 2. decreased conduction velocities in visual pathways 3. 25–50% decrease in scotopic visual acuity 4. persistent decreases in visual acuity and spatial resolution at PND 90	Fox et al. (1977), Impelman et al. (1982), Cooper et al. (1980), Winneke (1980), Fox and Wright (1982), Fox et al. (1982)

[a]Abbreviations: PND = post-natal day; $Pb(Ac)_2$ = lead acetate; MES = maximal electroshock seizure; VER = visual evoked response; NE = norepinephrine; DA = dopamine; GABA = gamma amino-butyric acid; GMP = guanosine monophosphate; 5HT = serotonin.

[b]Assuming daily consumption of 0.1 L of water per kilogram body weight (Arrington 1972).

state. Most common lead salts are either sparingly soluble (e.g., PbF_2, $PbCl_2$) or virtually insoluble (e.g., $PbSO_4$, $PbCrO_4$) in water, although a few salts are readily soluble (e.g., $Pb(NO_3)_2$, $Pb(CH_3COO)_2$). A large number of organolead compounds are known, and these nearly always involve the tetravalent (+4) oxidation state of lead. The chemistry of these compounds is complex, and is quite distinct from the chemistry of inorganic lead (USEPA, 1977).

Gastrointestinal absorption of lead is highly variable, depending on chemical form, dietary intake and age. For example, absorption from liquids is about five to eight times the absorption from food (USEPA, 1977). Typical gastrointestinal absorption of lead in adults is about 5% to 10%, but is higher (24% to 53%) in infants and young children (USEPA, 1977). Deposition and absorption of inhaled lead depend on particle size, chemical form, and rate and depth of breathing. In one study in human volunteers (Muir and Davies, 1967), 6% to 16% deposition was observed.

Absorbed lead distributes to all tissues of the body, but tends to accumulate in bone. In adults, over 94% of total lead body burden is in bone (Schroeder and Tipton, 1968). The major route of excretion of absorbed lead in humans is the urine (76%), with smaller amounts in feces (16%) and hair, nails and sweat (4%) (Rabinowitz et al., 1973). In human subjects fed a constant level of $_{240}Pb$, blood levels reached a steady state in about 100 days, and lead was cleared from the blood with a half-time of about 19 days after exposure ceased (Rabinowitz et al., 1973). The concentration of lead in soft tissues of humans usually reaches a steady state by age 20 (Barry, 1975), while levels in bone tend to increase at least until age 50 to 60 (Barry and Mossman, 1970).

Short-Term Exposure. Acute lead poisoning is not common, and is less thoroughly studied than chronic lead poisoning. Estimates of acute oral lethal doses in animals depend on chemical form, but are usually quite high (500 to 30,000 mg/kg) (NIOSH, 1983).

Studies in animals indicate that the perinatal period of ontogeny is a time of particular sensitivity to lead exposure. Table 1 is a partial summary of a large number of studies in which effects have been detected in offspring of mothers exposed to lead during gestation and/or after delivery. Ingestion by nursing females of water containing levels as low as 0.004% (40 mg/L, corresponding to a dose of about 4 mg/kg/day) causes effects on neurotransmitter metabolism in the offspring, and higher exposure levels result in a number of morphologic, biochemical and electrophysical changes.

Perinatal exposure to lead also produces a number of behavioral alterations in young animals. For example, Grant et al. (1980) exposed rats indirectly to lead *in utero* and during lactation through the mother's milk and, after weaning, directly through drinking water containing the same lead concentrations their respective dams had been given. There were delays in the development of surface righting and air righting reflexes in subjects given water containing 0.005% or 0.025% lead (50 or 250 mg/L, corresponding to about 5 or 25 mg/kg/day). Other reflexive patterns showed no effect. The median blood lead concentration for the 0.005% subjects at postnatal day 11 was 35 μg/L.

the ratio of the animal and human body weights. It is assumed that the average human body weight is 70 kg and that the average human consumes 2 liters of water per day. The multi-stage model is then fit to the equivalent human data to estimate the risk at low doses. The upper 95% confidence limit of this estimate is used. Excess cancer risks can also be estimated using other models, such as the one-hit model, the Weibull model, the logit model and the probit model. There is no basis in the current understanding of the biological mechanisms involved in cancer for choosing among these models. The estimates of low doses for these models can differ by several orders of magnitude.

The scientific database used to calculate and support the setting of risk rate levels has an inherent uncertainty. This is because the tools of scientific measurement, by their very nature, involve both systematic and random error. In most cases, only studies using experimental animals have been performed. There is thus uncertainty when the data are extrapolated to humans. When developing risk rate levels, several other areas of uncertainty exist, such as (1) incomplete knowledge concerning the health effects of contaminants in drinking water, (2) the impact of test animal age, sex and species and the nature of target organ systems examined on the toxicity study results and (3) the actual rate of exposure of internal targets in test animals or humans. Dose-response data are usually available only for high levels of exposure, not for the lower levels of exposure for which a standard is being set. When there is exposure to more than one contaminant, additional uncertainty results from a lack of information about possible synergistic or antagonistic effects.

Calculation of the ADI and AADI values for lead requires several changes in the normal approach described above. This is because most studies in humans evaluate the degree of lead exposure by measuring the concentration of lead in the blood (PbB), rather than by measuring lead ingestion. This requires that the NOAEL first be described in terms of a PbB, and then the oral exposure which produces that PbB must be calculated. The principal disadvantage to this approach is the additional uncertainty involved in such a calculation. On the other hand, since exposure is based on PbB levels, it is possible to consider studies in humans in which neither the route nor average daily exposure is known. For these reasons, in the following descriptions of studies on the toxicological effects of lead in humans, attention is focused on defining the PbB level which is not associated with adverse effects, regardless of route of exposure. From the PbB value selected as the NOAEL, an average oral daily intake level which would produce this PbB is calculated, and this is taken as the ADI.

NON-CARCINOGENIC EFFECTS

Lead is a gray-white metal that, because of its easy isolation and low melting point, was among the first of the metals to be utilized by humans. It is relatively abundant in the earth's crust, most commonly in the form of galena (lead sulfide). Lead is the heaviest member in Group IV-B of the periodic table (atomic number = 82, atomic weight = 207). Its inorganic chemistry is dominated by the divalent (+2) oxidation

3. A ten-day HA for 10-kg child.
4. A ten-day HA for 70-kg adult.
5. A lifetime AADI for a 70-kg adult.

The distinctions made between the HA calculations (items 1 through 4) are associated with the duration of anticipated exposure. Items 1 and 2 assume a single acute exposure to the chemical. Items 3 and 4 assume a limited period of exposure (possibly 1 to 2 weeks). The HA values will not be used in establishing a drinking water standard for the chemical. Rather, they will be used as informal scientific guidance to municipalities and other organizations when emergency spills or contamination situations occur. The AADI value (item 5) is intended to provide the scientific basis for establishing a drinking water standard based upon non-carcinogenic effects.

A NOAEL or LOAEL is determined from animal toxicity data or human effects data. For animal data, this level is divided by an uncertainty factor because there is no universally acceptable quantitative method to extrapolate from animals to humans. The possibility must be considered that humans are more sensitive to the toxic effects of chemicals than are animals. For human data, an uncertainty factor is also used to account for the heterogeneity of the human population in which persons exhibit differing sensitivity to toxic chemicals. An Office of Drinking Water (ODW) modification of the guidelines set forth by the National Academy of Sciences (NAS, 1977; 1980) is typically used in establishing uncertainty factors as follows:

- An uncertainty factor of 10 is used when good acute or chronic human exposure data are available and supported by acute or chronic toxicity data in other species.
- An uncertainty factor of 100 is used when good acute or chronic toxicity data identifying NOEL/NOAEL are available for one or more species, but human data are not available.
- An uncertainty factor of 1,000 is used when limited or incomplete acute or chronic toxicity data are available or when only the acute or chronic toxicity data identifying a LOAEL (but not NOEL/NOAEL) for one or more species are available.

The uncertainty factor used for a specific risk assessment is judgmental. Factors that cannot be incorporated in the NAS/ODW guidelines for selection of an uncertainty factor, but that must be considered, include: (1) the quality of the toxicology data, (2) the significance of the adverse effect and (3) the existence of counterbalancing beneficial effects.

If toxicological evidence requires the chemical to be classified as a potential carcinogen (there is evidence that some lead salts are carcinogenic following oral exposure in animals), mathematical models are used to calculate the estimated excess cancer risks associated with the ingestion of the chemical via drinking water. The bioassay data used in these estimates are from animal experiments. In order to predict the risk for humans, these data must be converted to an equivalent human dose. This conversion includes correction for non-continuous animal feeding, non-lifetime studies and differences in size. The factor that compensates for the size difference is the cube root of

LEAD HEALTH EFFECTS IN DRINKING WATER

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QUANTIFICATION OF TOXICOLOGICAL EFFECTS

The quantification of toxicological effects of a chemical consists of an assessment of the non-carcinogenic and carcinogenic effects. In the quantification of non-carcinogenic effects, an Acceptable Daily Intake (ADI) is calculated. An Adjusted Acceptable Daily Intake (AADI) and Health Advisory (HA) values for the chemical are then calculated to define the appropriate drinking water concentrations to limit human exposure. For ingestion data, this approach is illustrated as follows:

$$\text{ADI} = \frac{(\text{NOAEL or LOAEL in mg/kg/day})\ (\text{Body Weight in kg})}{\text{Uncertainty/Safety Factor}} = \text{mg/day}$$

$$\text{AADI} = \frac{\text{ADI}}{\text{Drinking Water Volume in L/day}} = \text{mg/L}$$

where:

NOAEL = no-observed-adverse-effect level.
LOAEL = lowest-observed-adverse-effect level.
Body weight = 70 kg for adult or 10 kg for child.
Drinking water volume = 2 L per day for adults or 1 L per day for children.
Uncertainty/Safety Factor = 10, 100 or 1,000.

Utilizing these equations, the following drinking water concentrations are developed for non-carcinogenic effects:

1. A one-day HA for 10-kg child.
2. A one-day HA for 70-kg adult.

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2. Key words: acceptable daily intake, blood lead, chronic poisoning, lead toxicity, neurological function.

3. Abbreviations: AADI, adjusted acceptable daily intake; ACH, acetylcholine; ADI, acceptable daily intake; ALA, aminolevulinic acid; ALAD, aminolevulinic acid dehydratase; DA, dopamine, FI, fixed interval; GABA, gamma aminobutyric acid; GMP, guanosine monophosphate; HA, health advisory; 5HT, serotonin; IARC, International Agency for Research on Cancer; IRT, inter-response time; LOAEL, lowest observed adverse effect level; MES, maximal electroshock seizure; NAS, National Academy of Sciences; NCV, nerve conduction velocity; NE, norepinephrine; NOAEL, no observed adverse effect level; ODW, Office of Drinking Water; $PB(Ac)_2$, lead acetate; PbB, blood lead concentration; PbD, daily dietary intake of lead; PbW, water lead concentration; PND, post-natal day; VER, visual evoked response.

Lead Health Effects in Drinking Water

William L. Marcus

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factors. Complex models of multivariate analysis are thus probably necessary. For example, when the presence or absence of SiF water treatment is considered along with other risk factors for lead uptake dichotomized at sample median, the analyses of variance (ANOVA) often show a significant interaction term ($p < .05$). Moreover, sometimes these interaction effects are different for children of different races. Whereas many studies of environmental toxins assume that variables of concern are causally independent, more attention thus needs to be devoted to multifactorial, interactive models of neurotoxicity (Masters and Coplan, 1999b). Recent research on the inhibitory effects of multiple sulfhydryl-reactive metals on the "adaptive and protective response to toxic metal exposure" by glutathione and metallothioneins (Quigg, 1998) confirms the importance of extending research to more complex mixtures and relationships than have typically been considered by either scholars or regulatory agencies (e.g., Calderon, 2000).

CONCLUSIONS

In the light of the foregoing discussion, the extent of subtle health effects associated with long term exposure to SiF is thus still unknown. Apart from the possibility of direct toxicity, freshly released monomeric silicic acid as well as fluoride ion bind calcium. If diets are low in milk products and other sources of calcium, the products of silicofluoride dissociation can exacerbate the competition between calcium and lead for bone and soft tissue sites. In addition, F^- has a high affinity for proteins (Emsley *et al.*, 1981) and is known to modify enzyme action (Westendorf, 1975; Padmanabhan and Shelanski, 1998) with potential for disrupting a wide range of endocrine, immune and neural processes. It has also been implicated in disturbing the functionality of calcium, both directly (ATSDR, 1993) and indirectly in interaction with vitamin D (Bayley *et al.*, 1990).

Studies of the long-term effects of exposure to fluorides in general and silicofluorides in particular must therefore take into account the strong possibility of multiple pathways and diverse mechanisms for intoxication. Even more important, recent biochemical findings link lead uptake in the brain to the replacement of zinc by lead in synaptotagmen, thereby changing its folding and greatly reducing its functional effectiveness (Godwin, 2000). Such new research apparently provides a precise neurochemical explanation for the well-known link between lead and lower cognitive ability (Needleman, 1992) and illustrates radically new methods to analyze lead neurotoxicity.

In the light of these facts and hypotheses, the congruent statistical findings from three populations totaling almost 400,000 children (over 238,000 in Massachusetts, over 150,000 in New York and over 4,000 in the NHANES III sample) indicate an urgent need for further study of the possible links between exposure to SiF and increased lead uptake as well as the behavioral dysfunctions associated with lead neurotoxicity. Given the paucity of direct knowledge about biomechanisms depending on exposure to commercial silicofluorides, and the magnitude of the potential risks–especially to poor and minority children–large-scale epidemiological studies, chemical analyses, and animal experimentation on silicofluorides and their effects deserve the highest priority.

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FIGURE 4: ORs for VBL>10µg/dL in Massachusetts SiF vs Non-SiF Communities Controlling For 4 Risk Factors

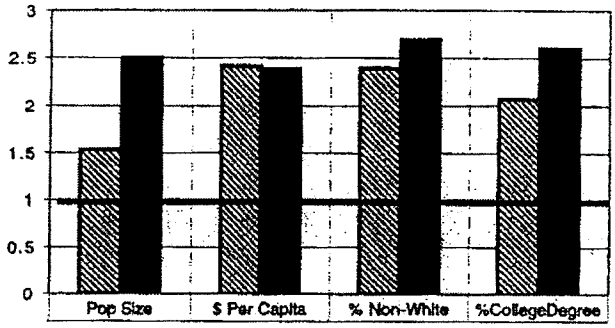

FIG. 4 ORs for VBL >10µg/dL in 30 SiF and 30 non-SiF Massachusetts comparable mid-size communities were determined for above and below median values of 4 risk factors: population size, per capita income, % non-white, and community average parent education level. ORs for VBL >10µg/dL in the communities with below median values for the referenced risk factor are represented by the gray bars; above the median, by the black bars. All ORs are close to 2.5 except for communities with below-median populations, where there is still a 50% greater chance of finding children with VBL > 10µg/dL if SiFs are used to treat the municipal water supply.

In Massachusetts we found risk ratios around 2.5 for elevated lead levels in venous and capillary blood tests by two independent methods. One compared the prevalence of VBL>10µg/dL among 37,000 children in 30 communities that use SiF with the prevalence of VBL>10µg/dL in 39,000 children living in 30 comparable communities that do not fluoridate. The other method compared mean capillary blood lead values of large populations of children in SiF and Non-F communities. To compare these results with the NY data, we re-analyzed our Massachusetts risk ratio data as OR values along the same lines as those reported here and depicted in Figure 1.

The Massachusetts OR results are summarized in Figure 4. Considering that similar ORs have now been found for two separate large populations from data collected and analyzed by entirely different agencies, it is unlikely that the results reported here are artifacts of sampling. Moreover, in a sample of over 30,000 criminals in 129 cities studied by the National Institute of Justice, behaviors associated with enhanced lead uptake vary in a manner consistent with the apparent effect of SiF (Masters and Coplan, 1999b).

Admittedly, in the absence of new chemical testing using contemporary methods of neurotoxicology, it is impossible to do more than hypothesize about the precise mechanisms that could account for these results. Nevertheless, these findings are extremely important for several reasons:

First, as noted above, it is not widely recognized that over 91% of fluoridated water has been treated with sodium silicofluoride or fluosilicic acid. Because less than 10% of the populace living in fluoridated communities receives water treated with sodium fluoride (NaF), the familiarity and widespread use of fluoridated toothpaste is not evidence of SiF safety. Despite MSDS data, (e.g., LCI, 1997) water plant managers and health agency personnel have little understanding of the toxic properties and behavior of the SiFs.

Second, it also true but not widely realized, that extensive tests on animals exposed to sodium fluoride in their diets have never been replicated by exposure to commercial grades of sodium fluosilicate or fluosilicic acid. Both federal health agencies and academic researchers customarily employ simple sodium fluoride in animal studies and extrapolate the results to humans ingesting silicofluoride treated water (Bucher *et al.*, 1997; Collins *et al.*, 1995; Dunipace *et al.*, 1998a; Dunipace *et al.*, 1998b; Dunipace *et al.*, 1995; Dunipace *et al.*, 1996; Jackson *et al.*, 1997; McClure, 1962; Sprando *et al.*, 1995; Sprando *et al.*, 1997; Sprando *et al.*, 1998). Moreover, this practice is reinforced by the habit of discussing "fluoridation" without *any* reference to the chemicals used (e.g., Newbrun and Horowitz, 1999). A detailed search of the literature which was relied upon for judging health safety of the SiFs for mass-fluoridation has produced only three comparative studies of the effects of sodium silicofluoride vs. sodium fluoride. One showed that rats and other animals process SiF differently from NaF (Kick *et al.*, 1935). Another showed that young children absorb fluoride from sodium silicofluoride over a substantially longer time than adults (Zipkin *et al.*, 1956). A third simply confirmed previous work of Zipkin, which had shown equivalent uptake of ingested fluoride by teeth and bones (Zipkin and McClure, 1951).

Third, officials at all levels responsible for water fluoridation often appear to lack an understanding of fundamental chemical principles relating to the use of silicofluorides. The silicofluoride anion that H_2SiF_6 and Na_2SiF_6 have in common must dissociate to release fluoride ion. This process is both complex and reversible, differing distinctly in many ways from release of fluoride from NaF by simple solution/ionization. Fluoridating water with either silicofluoride can thus create problems which are not experienced when adding NaF for that purpose (Reeves, 1994). The only extensive examination of the actual biochemical effects of SiF that we have encountered is a German study (Westendorf, 1975) — to our knowledge never cited before in the debates surrounding water fluoridation in the U.S. This research, which shows substantial changes in membrane permeability and enzymatic changes capable of substantially modifying neuronal excitability, suggests plausible chemical mechanisms that would be consistent with our empirical findings.

Finally, our findings (especially Figure 3) indicate that SiF treated water probably exacerbates the risks of absorbing lead from other known environmental risk

Figure 1: Odds Ratios of VBL>10µg/dL Comparing Children in 105 NY Communities (pop. 15,000-75,000) With and Without SiF Treated Water, Controlling for 7 Risk Factors for High Blood Lead

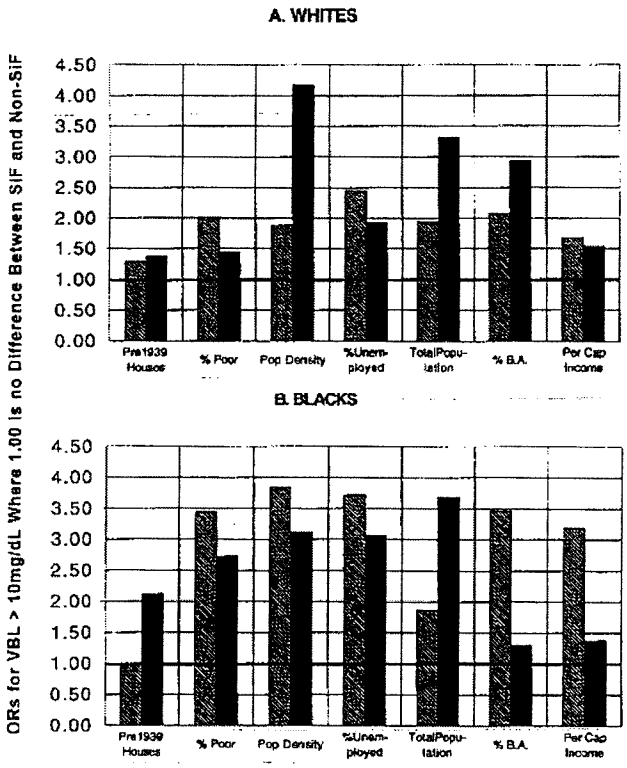

FIG. 1. Age adjusted logistic regression analysis of Odds Ratios for VBL > 10µg/dL in children 0-6 (Whites n = 72,542; Blacks, n = 18241) living in communities using and not using SiFs, ranked above and below the median for seven risk factors listed in Table 2. Light bars represent ORs for below the median of each identified risk factor and dark bars the ORs for above the median. An OR of 1.0 would mean that there is the same chance of VBL > 10µg/dL with and without SiF exposure; it does not mean low VBL.

UNRESOLVED ISSUES AND DISCUSSION

We are aware that statistical association should not be confused with causation. Moreover, since privacy limits have made it impossible to ascertain each individual's exposure to such risk factors as living in a house built before 1939, the assignment of community level covariates might artificially inflate significance levels (increasing the risk of Type I error). Though it is theoretically possible that there could be no effect, this conclusion seems unlikely when evidence from the present study is combined with congruent data from other studies. Prudence dictates, moreover, that a chemical delivered by government policy to 140 million people should have been tested and shown to be reasonably safe. As the Rio Declaration puts it in formulating the "precautionary principle," lack of "full scientific certainty shall not be used as a reason for postponing cost-effective measures to prevent environmental degradation" (Foster *et al.*, 2000).

Figure 2: Percent Tested with Venous Blood Lead>10µg/dLin New York State

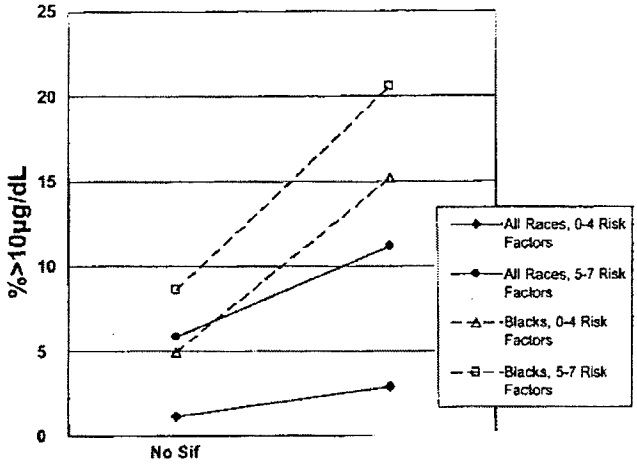

FIG. 2. Each of the ... factors from those listed in Table 2. ..., communities with five or more and those with 4 or fewer risk factors and further grouped by use or non-use of SiF. Broken lines indicate the prevalence of VBL >10µg/dL among Black children and the solid lines the prevalence of VBL >10µg/dL for all races

Figure 3: Percent Black Children Tested With >10µg/dL by Silicofluoride Use in NYState Communities Pop 15,000-75,000 with high % Old Housing and Childrens Poverty

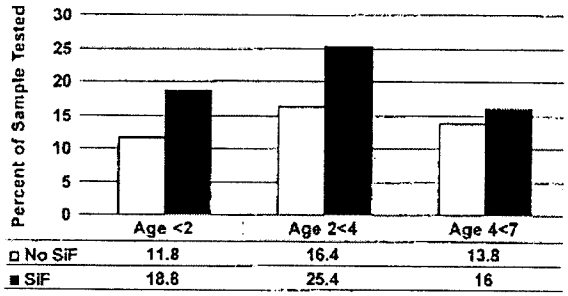

FIG. 3.

	n	Odds Ratio	CI	p <
All Ages	10,481	1.626	1.412, 1.873	0.0001
Age <2	4,076	1.729	1.354, 2.208	0.0001
Age 2<4	3,900	1.736	1.396, 2.150	0.0001
Age 4<7	2,505	1.277	0.959, 1.700	0.0949

Our analysis does not concern sodium fluoride, which is not used in a sufficient number of communities in this NY sample. Combined with our previous studies of data for children's blood lead in Massachusetts (Masters and Coplan, 1999a) and in the NHANES III sample (Masters, Coplan and Hone, in preparation) these findings point to a statistically significant risk of elevated blood lead associated with chronic ingestion of water treated with SiFs. In our experience, the data for NY State children reported here are not exceptions.

TABLE 2. Community Demographics and Risk Factors (Distribution of 1990 U. S. Census Variables in 105 NY State Communities of population 15,000-75,000, by SiF Status)

	SiF	Non-SiF
NUMBER OF COMMUNITIES	28	77
DEMOGRAPHICS		
Mean community population	34,778	25,627
Children 0-5 as % of population	8.5 %	8.0 %
No. of children 0-5 per community	2,960	2,046
TOTAL NUMBER OF CHILDREN TESTED 1994-1998		
Total number of VBL tests	58,934	94,291
Total number of capillary tests	36,791	68,357
Total of all blood lead tests	95,725	162,648
VBL Tests as % of total	61 %	58 %
RISK FACTORS ASSOCIATED WITH HIGH BLOOD LEAD		
Housing built before 1939	49.4 %	23.3 %
Children 0-5 in poverty	22.3 %	8.5 %
Community unemployment rate	3.5 %	2.5 %
Parents with Bachelor Degree	7.4 %	9.3 %
Population density (persons per sq. km.)	155	143
Total population of group	973,785	1,973,336
Per capita income	$14,698	$19,415

to each individual a value indicating whether his/her community was above or below the sample median for each covariate risk factor. We then used these as covariates in our analysis, dividing the sample of individuals into those who live in communities above and below the median of each covariate. Age-adjusted logistic regressions above and below the community median for each of the seven covariates (Figs 1a and 1b, and Appendix) showed, in most cases, a significant relationship between SiF use and elevated blood lead. An Odds Ratio (OR) of 1.0 (bold line in Figures 1a-1b) reflects an equal probability of elevated VBL for children living in communities with or without SiF treated water. In no case is an OR significantly lower than 1.0 where SiFs are in use. In contrast, for 49 of 56 regressions, the ORs are significantly over 1.0 for children exposed to SiF, with 35 over 2.0 (all significant), of which 23 are over 3.0. Moreover, ORs are significant in all 28 regressions for children in communities above the mean of a known risk-factor for lead uptake. Four of these ORs–all for Whites–are under 2.0 (range 1.4 - 1.9).

For Blacks and Hispanics exposed to both some other known risk-factor and SiF treated water, ORs range from 2.1 to 6.4 and are always significant. (See Appendix). ORs are significant 10 of 14 times for SiF use without any other risk factor. More striking, ORs are often doubled and sometimes quadrupled where Black and Hispanic children are exposed to both a risk factor such as old housing and SiF treated water.

The magnitude of some ORs might have been affected by sampling bias if conscious efforts were made to select children from high risk households in the SiF communities. We have, however, no information about such sampling procedures and sample size is robust (see Table 2). More important, because our statistical analysis considers risk-factors one at a time in accordance with standard multivariate methods, there is no reason to doubt the trend of ORs even should their magnitude sometimes be over-stated due to biased sampling.

To explore the relationship between SiF and combinations of the seven known risk factors ORs were computed by dividing the sample into those living in communities with four or fewer risk factors and those in communities with five or more risk factors. Exposure to five or more risk factors in combination always increases the risk of elevated blood lead. Where SiF is used to treat community water, exposure to this number of risks is associated with doubled values of OR (Figure 2). Although robust for all children tested (solid lines), the effect is substantially worse for Blacks (dashed lines).

Finally, it is commonly assumed that those at highest risk for high blood lead are poor Black children in old housing. For three age groups, logistic regressions indicated that if their community provided SiF-treated water, children in this group were at higher risk of elevated blood lead. Except for the age-group 4 to 6, which had the smallest sample, the effect was highly significant (Figure 3).

We chose seven variables from the U.S. census which tend to predict elevated VBL as likely covariates of elevated blood lead. Because children in communities with high levels of one or more of these variables are more likely to have high blood lead levels, our analysis computes a separate logistic regression for each race above and below the median of each covariate. By doing this one can determine whether there is an effect of SiFs independent of these covariates and, indeed, that was found to be the case.

RESULTS

As a measure of the likelihood of dangerous lead levels in children, we have selected the conventional cutting point of 10μg/dL in venous blood lead (VBL). To confirm that this choice would not bias our results, however, we first considered blood lead levels at increments of 5 μg/dL to assess effects of silicofluoride among Blacks in our sample. The data show that, for lead levels above 5 μg/dL, as the measured range increases from between 10 and 15 μg/dL to over 20 μg/dL, there are progressively higher odds ratios attributable to SiF (Table 1). These findings indicate that, for a dichotomous measure, the traditional cutting point (10 μg/dL) does not bias our findings and the choice of another measurement would not change the results in a material way. Indeed, using a higher cut-off point such as 15μg/dL (as proposed by Sargent, Dalton, and Klein, 1999) might be viewed as biased in favor of our hypothesis because odds ratios among Blacks are so much higher for lead levels of 15 to 20 μg/dL (3.5) or over 20 μg/dL (3.8) than for lead levels between 10 and 15 μg/dL (2.7) or between 5 and 10 μg/dL (1.3). Moreover, by focusing on the percent of children whose venous blood lead was in excess of 10μg/dL, we should minimize effects within the range of resampling error (Morris *et al.*, 1999; Sargent and Dalton, 1996).

Multivariate statistical analysis was performed for those variables which were known individually (age, race, exposure to SiF). Logistic regression was used to compare percentages of children with blood lead exceeding 10μg/dL in communities using SiF versus communities not using these chemicals. Because the odds ratios are consistently significant, for this sample as for others studied, the association between exposure to SiF and VBL>10mg/dL does not seem to be an artifact and deserves further study as a potentially serious issue of public health.

For each of the 105 communities, values for 7 risk factors were assigned based on the seven covariates listed in Table 2. To assess the overall vulnerability of those in high risk environments (cf. Binns *et al.*, 1999), we assigned

TABLE 1. Prevalence And OR of Elevated VBL for Black Children 0-6 in NY Communities of 15,000-75,000, Using And Not Using SiF to Treat The Municipal Water Supply

Number of communities	28 (SiF)	77 (Non-F)
Mean community sample size	310 (SiF)	124 (Non-F)
Total number tested	8,685 (SiF)	9,556 (Non-F)

A. Prevalence and Odds Ratios by VBL Interval

Interval	Using SiF		Not Using SiF		Odds Ratio
μg/dL	n	Prev %.	n	Prev %	SiF/Non-F
0-5	3,694	42.5	5,846	61.2	0.5
5-10	3,205	36.9	3,037	31.8	1.3
10-15	929	10.7	411	4.3	2.7
15-20	402	4.6	125	1.3	3.5
20 +	455	5.2	137	1.4	3.8

B. Prevalence and Odds Ratios by VBL Threshold Level

Threshold	Using SiF		Not Using SiF		Odds Ratio
μg/dL	n	Prev %.	n	Prev %	SiF/Non-F
< 5	3,694	42.5	5,846	61.2	0.5
> 5	4,991	57.5	3,710	38.8	2.1
> 10	1,786	20.6	673	7.0	3.4
> 15	857	9.9	262	2.7	3.9
> 20	455	5.2	137	1.4	3.8

independent relationships for blood lead levels and "median per capita income, percentage of housing built before 1950, percentage of the population who were Black, percentage of children screened, and a 'poverty index'." (Sargent *et al.*, 1995). Although not found in that study, other researchers have also found higher blood lead levels among Hispanics (Snyder *et al.*, 1995).

Recently, we carried out a further analysis of the Massachusetts blood lead data used by Sargent *et al. (op cit)*. We found that there was a statistically significant ($p = 0.001$ or better) correlation between community use of SiFs as reported by the CDC's fluoridation census (CDC, 1992) and a higher level of children's blood lead parameters, controlling for community factors commonly associated with blood lead, such as rates of housing built before 1950 and exposure to water containing over 15ppb first draw lead (Masters and Coplan, 1999a). Moreover, as predicted by our model (Masters and Coplan, 1999b), there were statistically significant interactions between these risk-factors and SiF usage as predictors of higher blood lead.

A second study has recently been completed, based on the Third National Health and Nutrition Evaluation Survey (NHANES III). Blood lead data for over 4,000 children living in 35 counties with a population over 500,000 was compared with county fluoridation status using the CDC's 1992 Fluoridation Census. The percent of each county population receiving silicofluoride treated water was calculated and each county was assigned to one of three groups. The "high" group comprised counties in which a total of 92% of the population received SiF-treated water. The "low" group comprised a population only 6% of which received SiF-treated water. A relatively small group of counties with "intermediate" exposure comprised a population with about a 50% chance of receiving SiF-treated water. Controlling at the individual level for covariates usually associated with lead uptake, elevated blood lead was statistically significant, again with $p = 0.001$ or better, with High/Low risk ratios in the range of 1.5 to 2.0 depending on age and race (Masters, Coplan and Hone, 1999 and in preparation).

The new analysis reported here is completely consistent with these earlier analyses of data from Massachusetts and NHANES III. Using New York State Health Department individual blood lead data for children 0-6, collected mostly between 1994 and 1998 by the Bureau of Children's Health', a multivariate study was done on data for over 151,225 children living in New York communities between 15,000 and 75,000 in population, of which 28 use SiF and 77 do not (one community using mixed chemicals had to be dropped from the analysis). We find that SiF is consistently associated with increased risk of having VBL>10mg/dL for virtually any race/age group, controlling for most factors commonly associated with increased blood lead.

METHODS

Over 1.2 million blood lead tests were collected by the State of New York, mostly between the years 1994 and 1998. We analyzed records in all communities between 15,000 and 75,000 in population (as determined using the 1990 US Census) which had at least 100 blood lead tests (five had fewer than 100 tests). Selecting communities in this size range avoided the complication of non-comparable large cities (such as New York City) and controlled for the effects of large population size as well as the environmental conditions in inner cities. For each community, we determined whether it uses SiF using the CDC 1992 Fluoridation Census. Olean, New York — the sole community using sodium fluoride — and towns using multiple chemicals were dropped from the multivariate comparisons, which focus on the contrast between non-fluoridated communities and those using one of the silicofluorides (fluosilicic acid or sodium silicofluoride) at 1.0 ppm. Levels of other factors associated with increased blood lead, such as age of housing and % aged 0-5 who are poor were determined from the 1990 U.S. Census. We are thus able to control at the individual level for factors such as race, age, community size and SiF exposure, but only indirectly for such factors as age of housing and poverty.

Of over 256,000 children tested, data for 151,225 were from venous blood tests, whereas the remaining 105,148 only had data from capillary blood. Because some researchers suggest greater reliability for the former (Sargent, Dalton and Klein, 1999; Morris *et al.*, 1999), we limit this paper to VBL (cf. Schlenker *et al.*, 1993; Hernandez-Avila *et al.*, 1998). In the future, it may be possible to study both methods. To minimize effects within the range of resampling error (Morris *et al.*, 1999; Sargent and Dalton, 1996), we focus on the percent of children whose venous blood lead was in excess of $10\mu g/dL$.

Researchers have differed widely on the threshold level of blood lead level representing danger to humans: although the conventional level for identifying threats to health and behavior has been $10\mu g/dL$, both higher and lower levels have been proposed. Some (e.g., Sargent, Dalton and Klein, 1999) claim it is not necessary to report levels below $15\mu g/dL$, others (e.g., Lanphear, 1999) suggest that any detectable lead in blood indicates unacceptable risks to development, health, and cognition. For evidence that this choice does not bias our findings, see Results below.

In a study such as this, it is also of utmost importance to test for covariance in order to avoid spurious correlation. We were able to control for race and age at the individual level by analyzing each age/race group separately. For other possible correlates of elevated VBL, we were only able to control at the community level.

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Association of Silicofluoride Treated Water with Elevated Blood Lead

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Abstract: *Previous epidemiological studies have associated silicofluoride-treated community water with enhanced child blood lead parameters. Chronic, low-level dosage of silicofluoride (SiF) has never been adequately tested for health effects in humans. We report here on a statistical study of 151,225 venous blood lead (VBL) tests taken from children ages 0-6 inclusive, living in 105 communities of populations from 15,000 to 75,000. The tests are part of a sample collected by the New York State Department of Children's Health, mostly from 1994-1998. Community fluoridation status was determined from the CDC 1992 Fluoridation Census. Covariates were assigned to each community using the 1990 U.S. Census. Blood lead measures were divided into groups based on race and age. Logistic regressions were carried out for each race/age group, as well as above and below the median of 7 covariates to test the relationship between known risk factors for lead uptake, exposure to SiF-treated water, and VBL >10μg/dL. RESULTS: For every age/race group, there was a consistently significant association of SiF treated community water and elevated blood lead. Logistic regressions above and below the median value of seven covariates show an effect of silicofluoride on blood lead independent of those covariates. The highest likelihood of children having VBL>10μg/dL occurs when they are both exposed to SiF treated water and likely to be subject to another risk factor known to be associated with high blood lead (e.g., old housing). Results are consistent with prior analyses of surveys of children's blood lead in Massachusetts and NHANES III. These data contradict the null hypothesis that there is no difference between the toxic effects of SiF and sodium fluoride, pointing to the need for chemical studies and comprehensive animal testing of water treated with commercial grade silicofluorides. © 2000 Intox Press, Inc.*

Key Words: Lead Neurotoxicity, Silicofluorides, Water Fluoridation, Venous Blood Tests, Public Health

INTRODUCTION

Over 91% of US fluoridated water is treated with either sodium silicofluoride (Na_2SiF_6) or fluosilicic acid (H_2SiF_6) — henceforth, the silicofluorides or SiFs. Less than 10% is treated with simple sodium fluoride (NaF). Whereas NaF was the model compound used in the 1940s for demonstrating safety and efficacy, and has been submitted to exhaustive animal testing for decades (e.g., McClure, 1962; Largent, 1961; Dunipace *et al.*, 1995, 1996, 1998a, 1998b; Jackson *et al.*, 1997), the same cannot be said of the SiFs. The Assistant Administrator of the EPA recently acknowledged (Fox, 1999) that his agency knew of no study of human health effects of chronic low-level exposure to either of the SiFs. Here we report data suggesting that SiF may enhance the uptake of lead from other environmental sources.

As is well known, many environmental and behavioral risk factors have previously been associated with increased lead levels among children. Among these are age, race, sex, income (or poverty), size of community (esp. between urban areas of greater and less than 1 million as well as between urban, suburban, and rural communities), location of community (including presence of soils with high lead levels), age of housing (presence of lead paint), lead in excess of 15 ppb in public water supplies, individual calcium or iron deficiency, maternal smoking, parental education and alcohol consumption (e.g., Needleman, 1992; Hense *et al.*, 1992; Cezard *et al.*, 1992; Weitzman *et al.*, 1993). Using community rates, a recent study of over 238,000 Massachusetts children found

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[25] This hypothesis may help unravel another paradox recently noted by an official of the EPA, who concluded a discussion of the impact of high levels of local soil lead on infant blood lead by remarking: "There is a mystery in all this. It's clear that soil-borne lead contributes to lead poisoning... What's not clear is why some soil-lead contamination contributes more to blood-lead levels than others ... Theories abound, based on such variables as the size of the lead particles or chemical form of the lead...", M. S. Stapleton, *Lead is a Silent Hazard* (Walker and Co., New York, 1994), p. 81. Our findings suggest that water treated with fluosilicic acid or sodium silicofluoride should be added to the list of suspects.

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[14] According to the *Merck Index*, 10th edition (Eds., M. Windholz, S. Budavari, R. P. Blumettl and E. S. Otterbein) (Rathway, NJ: Merck, 1983), p. 599, the uses of fluosilicic acid are: "a 1–2% soln is used widely for sterilizing equipment in brewing and bottling establishments. Other concns are used in the electrolytic refining of lead, in electroplating, for hardening cement, crumbling lime or brick work, for the removal of lime from hides during the tanning process, to remove molds, as preservative for timber. *Caution.* Severe corrosive effect on skin, mucous membranes" (#4089); uses of sodium silicofluoride (listed as sodium hexafluorosilicate) are: "in enamels for china and porcelain, manuf opal glass; as insect exterminator and poison for rodents, mothproofing of woolens" (#8459).

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[22] Towns using sodium fluorosilicate reported lower first draw water lead values (11.7 ppb) than unfluoridated towns (21.2 ppb) or towns using sodium flouride (17.5 ppb); communities using fluosilicic acid had significantly higher levels of lead than in others (39.3 ppb). Although the difference between usage of fluosilicic acid and all other treatment conditions is highly significant ($p < 0.0001$, DF 3, 223, F 9.32), differences in lead in first draw water cannot account for the fact that levels of children's blood lead are comparable in towns using sodium silicofluoride and fluosilicic acid. In any event, there is one order of magnitude difference between lead levels reported in water supplies (in parts per *billion* or 10^{-9}) and measures of lead uptake in blood (micrograms per deciliter are equivalent to parts per *one hundred million* or 10^{-8}).

[23] This result is all the more impressive because multiple regression reveals that percentage of housing built before 1940 is a significant predictor of which towns use silicofluorides (controlling for population density, % vacant housing, *per capita* income, racial composition, and other demographic variables).

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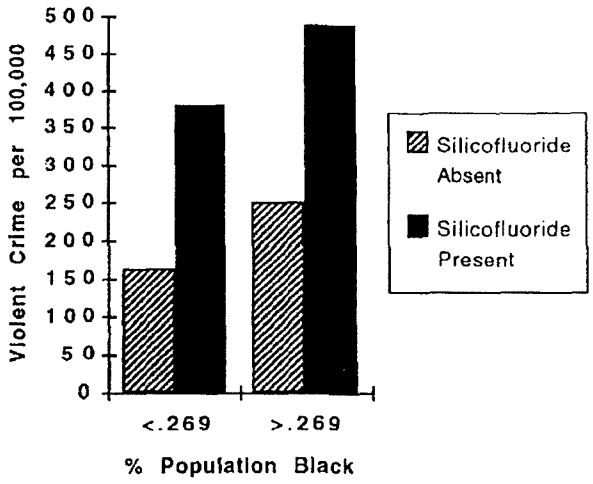
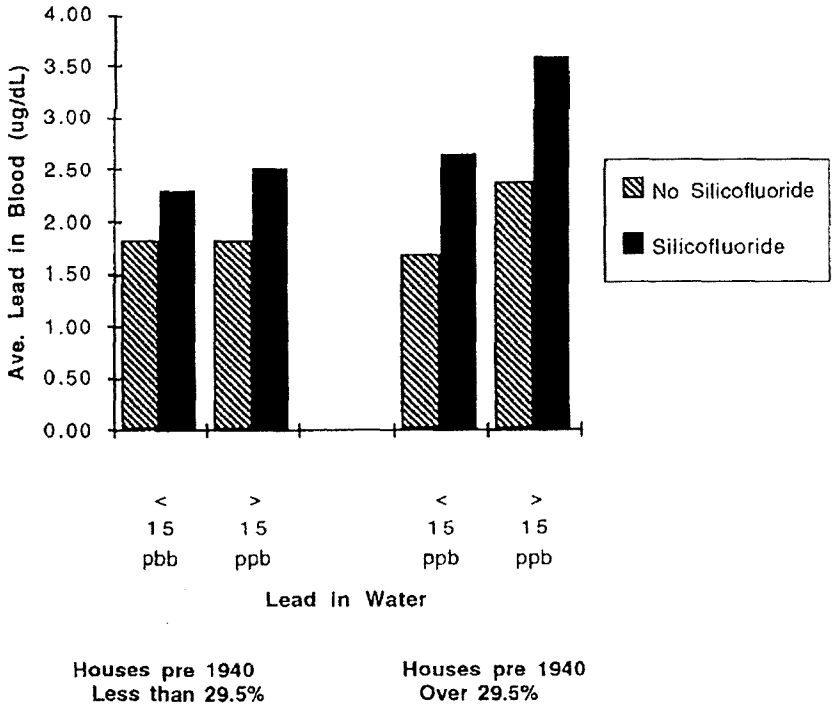
deficits in calcium and iron, income, race, and individual lifestyle [24], chemical agents used to treat public water supply appear to be an additional "risk factor" for lead toxicity [25]. It is noteworthy in this regard that rates of violent crime, elsewhere associated with lead neurotoxicity (Fig. 2), are significantly higher in communities that utilize silicofluorides in their water treatment systems not only in Massachusetts, but in Georgia, other states under consideration as this article goes to press, and a sample of cities with lead levels above 15 pbb [7, 26].



Rates of violent crime:
25 counties with no Silicofluoride water treatment and less than 26.9% black population = 161/100,000.
24 counties with no Silicofluoride water treatment and more than 26.9% black population = 249/100,000
52 counties with silicofluoride water treatment and less than 26.9% black population = 377/100,000
57 counties with silicofluoride water treatment and more than 26.9% black population = 486/100,000.
Georgia county ave. = 363/100,000. U.S. county ave. = 284/100,000):

Statistical Significance - Two way ANOVA:
effect of silicofluoride treatment: p = .0001, F 19.522, DF 1, 154
% blacks in population: p = .0561, F 3.704, DF 1, 154
Interaction = n.s.

FIGURE 2 Silicofluorides and Race as risk factors for violent crime–Georgia counties.



ANOVA Significance:
Main EFFECTS
% Houses pre 1940: p = .00901, F 21.17
90th percentile 1st Draw Lead > 15ppb: p = .0101, F 6.75
Silicofluoride use: p = .0177, F 5.63

Interaction effect
silicofluoride use * 1st Draw Lead in Water: p = .0422, F 4.18

FIGURE 1 Factors associated with children's blood levels – Massachusetts.

used for over half the population. Because communities where silicofluorides are in use were significantly under-sampled, moreover, it would be more accurate to compute estimated prevalence rates. On this basis, the risk-ratio of blood lead levels over 10 μg/dL is apparently increased by 1.46% in counties under 40,000, and 1.23 in counties from 40,000 to 250,000.

These findings suggest that the deleterious effect of the silicofluoride agents is not primarily due to a direct enhancement of lead in water, but rather through chemical effects that maintain lead in supension or biochemical effects that enhance lead uptake (or both). Although many other factors are involved in lead uptake, including dietary

TABLE II Correlation of lead in public water and fluoridation agents with children's blood lead

	None	*Sodium fluoride*	*Fluoridation agent*		*Average*
			Sodium Silico-fluoride	*Fluosilicic acid*	
Lead in Public Water					
< 15 ppb	1.97	2.11	2.37	2.31	2.08
n =	*86*	*31*	*6*	*26*	*149*
> 15 ppb	2.18	1.9	4.38	3.27	2.61
n =	*29*	*8*	*1*	*25*	*63*
Average	2.02	2.07	2.66	2.78	2.23

Analysis of Variance Lead in Public Water:: $p = 0.026$, F 5.06, DF2, 204; Fluoridation agent: $p = 0.004$, F 6.30, DF 3, 204; Interaction term: $p = 0.052$, F 2.62, DF 3, 204.

predictors of children's lead uptake is statistically significant ($p = 0.05$; DF 3,204, F2.62).

To confirm this effect, we assessed the extent to which silicofluoride usuage might increase the risk from lead paint in old housing as well as lead in the water. Towns were dichotomized according to whether they use silicofluoride agents, whether percent of houses built before 1940 was above the state median, and whether 90th percentile first draw water lead was over 15 ppb. In towns with both more old housing and high levels of lead in water, average blood lead is 3.59 μg/dL in 20 towns where silicofluorides are used, and only 2.50 μg/dL (slightly above the average of 2.23 μg/dL) in the 26 towns not using these agents. Controlling for other sources of lead, silicofluoride usage remains significant (Fig. 1).

Preliminary data from a parallel study at the county-level in Georgia confirm these effects. Since all large cities in Georgia use silicofluorides, data analysis that controls for population size must focus on counties under 250,000 population. In counties under 40,000 with untreated water, 14% of children screened had blood lead levels over 10 μg/dL, whereas in counties of this size where over half the population drank silicofluoride treated water, 16.8% had this level of lead in their blood. In intermediate sized counties (40,000 to 150,000), the rate of screened children with blood lead over 10 μg/dL was 7.1% if the county was not treated, and 10.5% where silicofluorides were

TABLE I Percent children screened with blood levels above CDC maximum (10μg/dL): matched sample of 30 nonfluoridated and 30 silicofluoride treated communities

	Total population (x 100,000)	*Children* 0–5	*# Children screened*	*# Lead risk (Pb > 10 μl/dL)*	*%Screened high risk*	*@ H_2O Pb (pbb)*	*4th Gr MEAP (average)*	*%Poor*	*%Non white*	*%AB*	*Income percap* ($1,000)	*Elem budget* ($1,000)
30 Non-fluoridated Communities	837.3	57031	37310	283	0.76	21	5440	4.6	6.6	23.6	16.6	3584
30 Fluorosilicic Acid or Sodium Fluorosilicate Treated Communities	845.1	56446	39256	762	1.94	30	5455	5.1	11.5	30.5	19.6	4067

8.408). Despite smaller samples tested, similar findings were obtained using venous blood uptake. These findings are independent of recorded 90th percentile first draw lead levels in the public water supplies [22].

To explore more fully the effect of both silicofluorides, a matched sample of 30 communities using these agents and 30 nonfluoridating towns was studied in detail (Tab. I). The matched groups are similar in total population (837,300 *versus* 845,100) and numbers of children between 0 and 5 (57,031 *versus* 56,446). While the non-fluoridated towns are slightly poorer (per capita income $16,600) than the communities using silicofluorides ($19,600) and have a lower percentage of residents with a college degree (23.6% *versus* 30.5%), they also have fewer residents who are either below the poverty line (4.6% *versus* 5.1%) or are nonwhite (6.6% *versus* 11.5%).

None of these factors would predict our finding that the communities using silicofluorides in water treatment have a disproportionate prevalence of children with dangerously high levels of blood lead: in our matched sample of similar communities, where silicofluoride was used, 1.94% of the children screened had over 10 μg/dL, whereas only 0.76% of the non-fluoridating towns had similarly high levels of lead in venous blood lead. This translates to a risk ratio of 2.55 ($p < 0.001$ by chi-square analysis).

DISCUSSION

Out findings suggest that chemical attributes of water treatment and delivery systems can mediate or enhance the deleterious effects of environmental exposure to lead. Such an hypothesis is confirmed by analysis of variance to assess interactions between fluoridation treatment agents and other sources of lead in the environment as factors influencing local variations in children's uptake of lead. When both fluoridating agents and 90th percentile first draw lead levels are used as predictors of lead uptake (Tab. II), the silicofluoride agents are only associated with substantially above average infant blood lead where lead levels in water are higher than 15 ppm. This interaction between the use of silicofluorides and above average lead in water as

Multivariate analyses provided a control for such socio-economic and demographic factors as population density, income, housing age, and race. Although the original survey included children from the entire state of Massachusetts (350 communities), multivariate data could only be analyzed for the 213 communities for which we could obtain water quality reports and census data as well as blood lead screening. This group, however, includes all but one fluoridated town and virtually all other communities over 3,000 population.

To control for the methodological questions that have been raised concerning multiple regression models in epidemiological research [21], a sample of 30 communities with populations between 17,000 and 48,000 (total population 837,300) using silicofluorides was compared with a matched set of 30 unfluoridated communities (total population 845,100). This sample excludes Boston and other large cities, where higher rates of poverty, crime and educational failure might confound the analysis. Because most of the localities not included in at least one of these data analyses are extremely small and have populations that often use household wells for their water supplies, the resulting findings incorporate all major towns and cities and the vast majority of the Massachusetts population.

RESULTS

Whereas a community's average uptake of lead by children is weakly associated with the so-called "90th percentile first draw" levels of lead in public water supplies (adjusted $r^2 = 0.02$), the fluoridation agents used in water treatment have a major effect on lead levels in children's blood. Average levels of lead in capillary blood were 2.78 μg/dL in communities using fluosilicic acid and 2.66 μg/dL in communities using sodium silicofluoride, while they were significantly lower in communities that used sodium fluoride (2.07 μg/dL) or did not fluoridate (2.02 μg/dL) (one way ANOVA, $p = 0.0006$; DF 3, 209, F 6.073). The prevalence rate of individuals whose capillary blood lead was above the maximum permissible level of 10 μg/dL was also significantly higher in the communities using either of the silicofluoride compounds (fluosilicic acid = 2.9%, sodium silicofluoride = 3.0%; sodium fluoride = 1.6% untreated = 1.9%; $p < 0.0001$; DF 3,212, F

levels of fluoride in the water prior to treatment; such communities are allowed to distribute water with up to 4 ppm of fluoride, whereas when fluoridation agents are used, water is adjusted to between 0.8 and 1.2 ppm fluoride.

Data on both fluoridation agent and average first draw lead levels in water were available for 227 towns: 51 using fluosilicic acid, 40 using sodium fluoride, 7 towns using sodium silicofluoride, and 129 not using a fluoridation agent.

3. Demographic and Socio-economic Measures

WTown-level data were compiled from the U.S. census, FBI, and Massachusetts Department of Education. Variables studied include population density, per capita income, ethnic composition, housing age and quality, percentage of households on public welfare, percentage high school dropouts (per capita), and rates of both violent and property crime. Because census data on high school dropouts reflects economic opportunity and social class as well as educational failure, standardized test scores (MEAP) and dollar expenditures per town were also used. (Special education budgets would be unreliable for this purpose, both because towns vary greatly in educational policies such as mainstreaming and because the influence of lead on IQ has been shown to be continuous, with no threshold effects allowing a segregation of the population potentially affected [20].

4. Statistical Analyses

Analysis of variance and multivariate models were used to asess the associations between children's blood levels of lead and the lead levels in their community's water supply, between lead levels in water supplies and fluoridation agents, and between behavioral deficits otherwise associated with lead uptake and children's blood lead levels. Because this is an ecological study and sampling error in each community's average level of blood lead was a matter of concern, different measures of lead toxicity (average levels of lead in capillary blood, and percent screened with venous blood above 10 μg/dL, the current CDC threshold) were used, as were a variety of behavioral factors associated with lead neurotoxicity.

statewide multivariate statistical analysis. To confirm that these findings were not due to confounding factors, including possible contamination of capillary blood, venous blood was used to assess the prevalence rate of children between 0 and 4 whose blood lead level was above the CDC recommended maximum of 10 μg/dL. (Selection bias, due to retest of children with high levels of lead in capillary blood, need not confound this measure as it does for community averages of blood lead uptake).

2. Public Water Supplies

Water quality reports from Massachusetts towns for levels of lead and other toxic metals are routinely maintained by Federal law [18]. Our analysis is based on reports provided by the Massachusetts Department of Environment's Drinking Water Program, listing each community's average 90th percentile first draw levels of lead – that is, the highest decile of reported lead (in ppb) when tested household taps are first turned on in the morning. Although so-called "service" levels, after water has been allowed to run, are typically much lower than first draw, our measure is the nationally accepted measurement of safe water quality. Inspection of raw data from local water systems shows, however, that these measures vary considerably over time as well as between one household and another in the same neighborhood. Because statistical analysis using reported water lead levels as a continuous variable may be less reliable than has often been assumed, we have also divided the sample by quartile in order to compute analyses of variance.

Methods of fluoridation are classified and reported by the United States Public Health Service census of water treatment facilities [19]. Three principal agents are utilized in the U.S.: 30% fluosilicic acid (H_2SiF_6), a liquid which is injected into a main water stream *via* a flow rate control device; sodium silicofluoride (Na_2SiF_6), a dry powder which is made up to a saturated solution which is then metered into the main stream, and sodium fluoride (NaF), the agent used in most toothpastes, which is also made up to a saturated solution for metering into the main water stream. The two silicofluorides require addition of neutralizing agent since they both produce significant acidity. Some communities are also considered to be "naturally" fluoridated due to

The relation between municipal potable water and enhanced lead uptake was analyzed for all communities in Massachusetts by combining: (i) data compiled in an extensive survey of children's blood lead levels conducted in 1990–91 by researchers at Dartmouth College and Mary Hitchcock Hospital [13], (ii) reports of "first draw" water lead monitored in 1992–93 under the EPA "Lead/Copper Rule", (iii) data from the 1990 US National Census; and (iv) water fluoridation methods employed by individual communities as tabulated in the "CDC Fluoridation Census, 1992."

The use of silicofluorides–fluosilicic acid (H_2SiF_6) and sodium silicofluoride (Na_2SiF_6)–is suspect for several reasons [14]. First, dissociation of these compounds into free fluoride anion (F^-) and the associated acidic cation (H_3O^+) is almost certainly not complete under the conditions of normal water treatment, leaving residues of toxic silicofluorides in the water stream [15]. Consequently, there is a strong likelihood that additional F^- and H_3O^+ will be liberated into the water after it has left the water treatment facilities, with attendant chemical reactions that increase acidity (lower pH) beyond the expectations established by current practice. Acid water in turn can extract lead from pipes, solder and fixtures made of lead-bearing alloys, and increase the bioavailability of lead from water at the tap [16]. In addition, traces of undissociated fluorinated silica residues may complex dissolved lead and facilitate its transport from the gastrointestinal tract to the blood stream [17].

METHODS

1. Blood Lead Levels

The Dartmouth-Hitchcock Hospital study sampled children ages 0 to 4 for capillary blood levels in μg/dL; a much smaller sample of venous blood lead levels in μg/dL was also recorded. (For detailed methods, see earlier published reports) [12, 13]. Because the total number of children screened for venous blood was so small (median of 8 children per community, with the lowest quartile towns averaging only 2 children between the ages of 0 and 4) and reflected potential selection bias, community average levels of capillary blood were used for

to which these chemicals are risk co-factors for lead uptake and the hazardous effects it produces.

Keywords: Lead; toxicity; pollution; children's health; public water supplies

INTRODUCTION

Although it has been known since antiquity that lead is harmful to human health, its precise neurotoxic effects have only recently been studied in detail [1, 2]. Lead exposure is now considered a risk factor in fetal and early childhood developmental deficits, premature birth, low cranial circumference, lower IQ, learning deficits, attention deficit disorder (ADD) or hyperactivity (ADHD), and reduced impulse control [3]. Lead neurotoxicity has also been linked with higher rates of aggressive or criminal behavior in both correlational [4] and prospective studies [5]. Although acute exposure to lead also increases susceptibility to hypertension, heart disease, renal disease, or cancer, evidence of disease risk from chronic, low-level exposure is mixed (suggesting the importance of other risk co-factors) [6]. For some behavioral effects of lead neurotoxicity, data based on individual levels of lead absorption have been confirmed by ecological comparisons between communities or counties in the U. S. that vary in the exposure to lead pollution [7, 8].

Apart from occupational hazards, attention was long focused on leaded gasoline and house paint as the principal vectors of public exposure to lead. The prohibition of these products has been associated with a significant drop in children's blood lead levels [9]. Nevertheless, chronic low level lead intoxication remains a major problem of public health and behavioral dysfunction in the United States [10].

Recent research has shifted to other sources of lead, including urban soils with lead residues from gasoline additives [11] and industrial pollution [12]. The possibility that supplied municipal water might be an additional vector was suggested in a recent study of over 2800 counties in the continental United States in which – controlling for such socioeconomic and demographic variables as population density, income, and race as well as industrial pollution – the percentage of households on public water supplies was an additional risk factor for high school dropout, welfare dependence, and violent crime [7].

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WATER TREATMENT WITH SILICOFLUORIDES AND LEAD TOXICITY

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Toxic metals like lead, manganese, copper and cadmium damage neurons and deregulate neurotransmitters like serotonin and dopamine (which are essential to normal impulse control and learning). Earlier studies show that–controlling for socio-economic and demographic factors–environmental pollution with lead is a highly significant risk factor in predicting higher rates of crime, attention deficit disorder or hyperactivity, and learning disabilities. Exposure and uptake of lead has been associated with industrial pollution, leaded paint and plumbing systems in old housing, lead residues in soil, dietary habits (such as shortages of calcium and iron), and demographic factors (such as poverty, stress, and minority ethnicity). We report here on an additional "risk co-factor" making lead and other toxic metals in the environment more dangerous to local residents: the use of silicofluorides as agents in water treatment. The two chemicals in question–fluosilicic acid and sodium silicofluoride–are toxins that, despite claims to the contrary, do not dissociate completely and change water chemistry when used under normal water treatment practices. As a result, water treatment with siliconfluorides apparently functions to increase the cellular uptake of lead. Data from lead screening of over 280,000 children in Massachusetts indicates that silicofluoride usage is associated with significant increases in average lead in children's blood as well as percentage of children with blood lead in excess of 10 μg/dL. Consistent with the hypothesized role of silicofluorides as enhancing uptake of lead whatever the source of exposure, children are especially at risk for higher blood lead in those communities with more old housing or lead in excess of 15 ppb in first draw water samples where silicofluorides are also in use. Preliminary findings from county-level data in Georgia confirm that silicofluoride usage is associated with higher levels of lead in children's blood. In both Massachusetts and Georgia, moreover, behaviors associated with lead nurotoxicity are more frequent in communities using silicofluorides than in comparable localities that do not use these chemicals. Because there has been insufficient animal or human testing of silicofluoride treated water, further study of the effect of silicofluorides is needed to clarify the extent

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incidence of EBL declined 50% nationally in the same time period (*11*). Indeed, the original CDC study did find a slight (but insignificant) increase in incidence of EBL in 2001 versus 2000 for residents living in homes with lead pipe (*3*). When the CDC 2001 data are broken into halves according to the approach of this work, the second half of 2001 has an anomalous increase in EBL incidence relative to what occurred in 1999 or 2000 for all ages tested (Figure 1; Supporting Information 6). The results for second half of 2001 are deserving of increased scrutiny in light of the very high WLLs throughout the city in July and August 2001 (Supporting Information Figures S4 and S14).

There are two other studies that examined the impact of WLLs on BLLs of DC residents. Guidotti et al. (2007) report a low incidence of EBL in a population tested well after high WLLs were front page news (*4*). Another portion of the CDC (2004) study reported no cases of EBL in 2004 for residents living in homes where second draw WLLs were over 300 ppb (*3*). In both of these studies there was a delay of months to a year between the time that consumers were first informed of hazardous WLLs and the actual measurement of their BLL (*21*). Since the half-life of lead in blood is 28–36 days, these results cannot be construed to indicate lack of harm from exposure to the lead contaminated water (*22*).

The Guidotti et al. (2007) study also erroneously identified critical dates and facts regarding the lead in water contamination event that skewed interpretations (*4*). For example the authors state that:

(1) chloramine was first added to the water supply in November 2002 [the actual date for addition of chloramines was November 2000 [see Supporting Information 7]
(2) WLLs showed an "abrupt rise" in 2003 [the WLLs had risen by the second half of 2001 as per Figure 1]
(3) the Washington, DC population had been protected by "massive public health interventions" starting in 2003 [the significant public health intervention did not begin until after the story was front page news in early 2004, see Supporting Information 7].

This may explain why the conclusions of Guidotti et al. (2007) differ from those of Miranda et al. (2006), who found a significant correlation between children's BLLs and a switch to chloramine disinfection in North Carolina (*14*).

Overall, this research demonstrates that the experience in Washington, DC is consistent with decades of research linking elevated WLLs to higher BLL and EBL (*23, 24*). Studies in France (*25*), Scotland (*26*) and Germany (*27*) correlated WLLs to adult BLLs, even for adults drinking water after corrosion control markedly reduced water lead levels. Lanphear has also noted a correlation between BLLs and higher WLLs in a U.S. city in which no system-wide problem with WLLs was occurring (*28*). Lead in potable water is therefore a viable explanation for some of the 30% of elevated BLL cases that occur nationally for which no paint source can be found (*20*), and may even be a significant contributor to EBL in cases where lead paint is identified as a hazard in the home. Assumptions by the CDC that high WLLs are rarely the cause of EBL in children should be re-evaluated.

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Supporting Information Available

Seven supporting analyses and three reports (EPA, 2004). This material is available free of charge via the Internet at http://pubs.acs.org.

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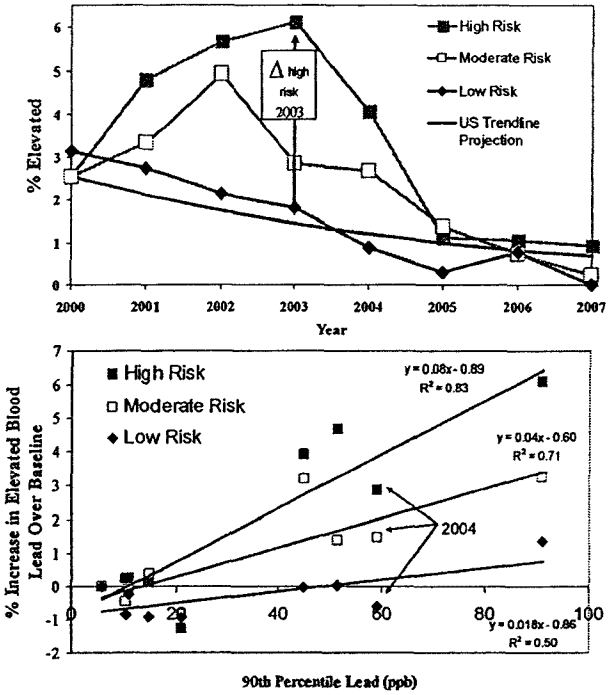

FIGURE 2. Temporal trends in incidence of EBL for children age ≤30 months. The deviation from the U.S. trendline is determined by the difference between the actual data and the projected U.S. trendline (top). Correlation between increased incidence of elevated blood lead in Washington, DC children aged ≤30 months and 90th percentile lead (bottom).

in the moderate-risk part of the city, and 4.4 times higher than in the low-risk part of the city.

If the 2001 data are not split into a first and second half, R^2 in the high-risk part of the city drops from 0.83 to 0.65, R^2 in the moderate-risk part of the city drops from 0.71 to 0.45, and R^2 in the low-risk part of the city drops from 0.50 to 0.18. (Supporting Information 5). The 2004 data also deviate significantly from the trendline (Figure 2 bottom), in that the high WLLs did not increase the percentage of children aged ≤30 months with EBL to the same extent as they did in 2001–2003. This is to be expected, since public health interventions were implemented in early 2004. If 2004 were treated as a transitional year and excluded from the analysis, R^2 would increase for the correlations (Supporting Information 5).

Discussion

EBL Cases Attributed to High WLLS Versus Predictions of Bio-Kinetic Model. The estimated number of children with EBL in Washington, DC due to the lead-contaminated water can be roughly estimated using the results of Figure 2 and the population of children aged ≤30 months in each part of the city (low-, moderate-, and high-risk neighborhoods). It is estimated from the CNMC analysis that 342 children in DC aged ≤30 months had EBL in 2003 due to high WLLs, and that 517 additional children aged ≤30 months had EBL from high WLLs in 2002. The corresponding exposure model predictions including all 1 year old, all 2 year old, and 50% of the 3 year old category (to approximate children aged ≤30 months) is for 170 cases in 2003 (Supporting Information 1). The discrepancy (342 estimated cases of EBL using the CNMC analysis vs 170 predicted for children aged ≤30 months) is not large given the model assumptions. It is not even unexpected, since the exposure model predictions did not include cases for which BLLs would be raised above 10 μg/dL from a combination of sources that include water. The most significant impacts of the high WLLs on EBL incidence probably occurred in the second half of 2001 (Figure 1, Figure 2, Supporting Information Figures S13 and S14), but calculating an increased number of EBL cases in that time period is beyond the scope of this work.

Lack of Monitoring Data for the Population Most Vulnerable to High WLLs. CDC recommends that BLL blood lead of children be screened at 1 and 2 years of age, "based on the fact that children's blood lead levels increase most rapidly at 6–12 months age and peak at 18–24 months (*18*)." These guidelines were developed from studies conducted in Cincinnati and elsewhere, where lead dust and lead paint were the predominant sources of exposure and water lead levels were low (*19*). In contrast, previous research has demonstrated that BLLs begin to rise rapidly when infant formula contains elevated lead ((*7*), see Supporting Information 2 Figure S6). Thus, when lead contaminated water is the sole or main source of lead exposure for infants, it is logical to expect that blood lead levels would tend to peak at ages much younger than 18–24 months (Supporting Information 1).

In earlier research on effects of high WLL on EBL for Washington, DC residents, it was stated that the blood lead monitoring was "focused on identifying children at highest risk for lead exposure (*3*)." This statement is correct from the perspective of lead paint and lead dust, but it is not necessarily accurate from the perspective of exposure to lead from water. Indeed, because so little blood lead data had been collected in Washington, DC for the population most vulnerable to high WLLs, no statistically valid conclusions are possible relative to incidence of EBL for children aged ≤1 year. The data presented herein for children aged ≤1.3 years (which are actually mostly data for children aged 1–1.3 years) supports the prediction that the impacts would be highly significant.

Other Considerations and Biases. A significant number of children aged ≥30 months was likely to have EBL during 2000–2003 because of exposure to high WLLs (Supporting Information 1). Moreover, even in the low-risk neighborhoods many children probably had EBL due to exposure to high WLLs. But the increased incidence of these cases cannot be readily detected in the BLL monitoring data in this work for reasons discussed previously. It is also inevitable that some misclassification of children's addresses will occur in a study of this nature, in that some children in high-risk neighborhoods would be misclassified as living in low-risk neighborhoods and vice versa. To the extent that such random bias occurred, it would tend to make the reported correlations between EBL and WLLs less significant than they actually were.

The Literature Revisited. Differences in conclusions between this work and the earlier CDC study (*3*) are mostly attributed to the type of analysis and interpretation, as opposed to discrepancies between the two databases discussed previously. In a recent discussion of the original CDC results, Levin et al. (2008) noted that the percentage of BLL measurements ≥5 μg/dL declined by 70% from 2000–2003 across the U.S., but did not decline at all in Washington, DC during the period of high WLLs (*20*). The obvious implication is that the high WLLs in Washington, DC countered the expected decline in BLLs that would have otherwise occurred, even for the general population that was analyzed in the CDC report.

Applying the Levin et al. (2008) logic to a closer examination of the CDC (2004) data suggests that the rate of decline in BLL measurements >10 μg/dL across the city was also reduced during the time period that WLLs were high. For example, the CDC study reported that from 2000 to 2003, the incidence of BLL measurements >10 μg/dL in homes with lead pipe declined by 28%, whereas

TABLE 1. Summary Data for Neighborhoods of High, Moderate, and Low Relative Risk of Exposure to High Elevated WLLs

relative exposure risk	est. lead pipes	% of total pop. in city	pop. (1000)	% pop. with lead pipe	% 1st draw over 100 ppb	% pop. above indicated WLL (ppb)		
						1st draw >100	2nd draw >100	1st draw >400
high	10086	22	126.3	17.6	15.0	2.63	3.43	0.13
moderate	14743	55	314.3	10.3	9.4	0.97	1.59	0.02
low	1318	23	131.4	2.2	12.8	0.28	0.37	0.00

are not expected to be in agreement, because the CDC included multiple measurements of blood lead for children which tends to skew the EBL incidence higher. CDC data for 2003 are not plotted on the graph, because only 90 children were identified as age 1−16 months for that year, of which 31 had elevated blood lead (34% EBL incidence).

Data from the CDC study (*3*) were then compared to the blood lead data (>28,000 records) from CNMC. In theory, the CNMC data are a subset of the more expansive data compiled and maintained by the DC Department of Health and which were used in the CDC study. However, a comparison of records between the two databases for the year 2003 revealed an error rate of more than 50%. That is, there was less than a 50% chance that a given record in the CNMC database matched a record in the CDC data in 5 domains: sample collection date, subject age, sample recording date, zip code, and BLL. Because repeated attempts to resolve this and other discrepancies in the CDC data were not successful, only the CNMC data were used for analyses and conclusions in this work.

Correlation Between EBL and WLLs for Children Aged ≤30 Months. No strong temporal trends or correlations between EBL incidence and the varying WLLs were observed for children aged ≤30 months if the data were analyzed across the entire city (data not shown). A neighborhood analysis of the data was then conducted.

High-, Moderate-, and Low-Exposure-Risk Neighborhoods. During 2003, the local utility conducted intensive sampling in Washington, DC homes with lead service pipe. Contrary to the popular perception that lead leaching to water is a fairly reproducible phenomenon from home to home, WLLs present in the first and second draw (flushed) samples from home to home vary dramatically (*9, 17*). For instance, in homes known to have lead service line pipe the second draw samples collected from 33% of homes had WLLs below the 15 ppb EPA action level. But 17% were above 100 ppb, 1% were above 1,000 ppb, and one sample contained 48,000 ppb.

A Freedom of Information Act request of the water utility revealed that a "geographic phenomena" was identified that played a key role in the observed variability of water lead in homes throughout the city (Supporting Information 3). Specifically, certain neighborhoods were "hot spots" for high water lead. While the utility would not provide documentation of the neighborhood analysis, their 2003 lead in water data were scrutinized for geographic trends based on zip code.

The analysis demonstrated that relative risk of exposure to high lead in water was a strong function of zip code (see Supporting Information 4). To capture the risk of exposure to high WLLs for the different neighborhoods, while also pooling data to maintain sufficient statistical power, the city was demarcated into neighborhoods that had relatively high risk (22% of the population), moderate risk (55% of the population), and low risk (23% of the population). In the high-risk part of the city, 2.63% of the population had first draw WLLs above 100 ppb (Table 1). This is 9.4 times higher than the 0.28% of the population having first draw WLLs above 100 ppb in the low-risk part of the city, and 2.7 times higher than in the moderate-risk part of the city (Table 1). The population living in the high-risk neighborhoods also had much greater likelihood of exposure to second draw lead over 100 ppb or to first draw lead over 400 ppb when compared to the moderate- and low-risk neighborhoods (Table 1).

Temporal Trends in EBL. The incidence of EBL for children aged ≤30 months had strong temporal trends that differed based on neighborhood risk level (Figure 2). In the high-risk neighborhoods EBL incidence increased from 2.5% in 2000 when WLLs were low, to 6% in 2003 after WLLs had been high for a few years. Thus, the incidence of EBL cases increased 2.4 times in 2003 versus 2000 in the high risk neighborhoods. The incidence of EBL dropped rapidly in the high-risk neighborhoods beginning in 2004. In the moderate-risk part of the city the EBL incidence was higher in each of the years 2001−2003 when water lead levels were high, relative to 2000 when water lead levels were low. But in neighborhoods of the city with the lowest risk of exposure to high WLLs, the percentage of children aged ≤30 months with EBL dropped steadily from 2000 to 2007.

Comparing the high-risk part of the city to the low-risk part of the city using a proportions test in *R* shows no significant difference in EBL incidence for the year 2000 (before WLLs were high) or for 2001 ($p = 0.544$ and 0.330, respectively). But utilizing the same test in 2002, 2003, and 2004 shows a statistically higher incidence of EBL in high-risk neighborhoods relative to low-risk neighborhoods ($p =$ 0.024 for 2002, 0.037 for 2003, and 0.006 for 2004). This analysis shows that the high WLLs had a very significant impact on EBL incidence for children aged ≤30 months in the neighborhoods with high WLLs.

Comparison of EBL in Washington, DC to the U.S. Trend in BLLs, 2000−2007. National trends in EBL incidence from 2000 to 2006 (*11*) are reasonably fit by an exponential decay model with an annual rate constant of −0.1867/year ($R^2 =$ 0.99). Extrapolation of this trendline using the year 2000 as time = 0 provides a basis for relating the Washington, DC blood lead data to the national trend. For example, the calculated "Δ high risk 2003" (Figure 2), is the difference between the U.S. trendline and the DC data. This represents the increased incidence of EBL in the high-risk DC neighborhoods in 2003, compared to what would have occurred if the national trend had been followed.

Correlation between WLL and Deviations from National BLL Trends. The correlation between the increased incidence of EBL in DC children aged ≤30 months versus national trends, and the reported 90th percentile WLL concentrations for the city, was dependent on the neighborhood risk level (Figure 2). In neighborhoods with the highest WLLs a strong positive linear correlation was established between the increased incidence of EBL and the 90th percentile WLL concentration ($R^2 = 0.82$). In the moderate-risk section of the city the slope and correlation were slightly lower ($R^2 =$ 0.71). The weak correlation ($R^2 = 0.50$) in the low-risk section of the city is to be expected, because the population in these neighborhoods had relatively low likelihood of exposure to high WLLs (Table 1). The slope of the trend-line in the highest risk part of the city is approximately double that observed

TABLE 2
Behavioral Effects of Direct Lead Exposure in Young Rats[a]

Reference	Lead Exposure: Pb conc. (medium)	Lead Exposure: Period (route)	Estimated daily dose[b] (mg/kg/day)	paradigm (task)	Age at testing	Behavioral results
Overmann (1977)		PND 3–21 (gavage)	C = 0 Pb_1 = 5 Pb_2 = 15 Pb_3 = 45	Adversive conditioning	26–29 days	Pb_3-*Ss* sig. slower in acquisition and extinction of active avoidance response; no sig. diffs. for passive avoidance. All Pb groups failed to inhibit responses as well as C-*Ss*. No sig. diffs. on E-maze tasks, except $Pb_{2,3}$-*Ss* sig. worse than C-*Ss* when on tactile discrim.
Dietz et al. (1978)		PND 3–30 (gavage)	C = 0 Pb = 200	Operant (minimum 20-sec pd. between bar-presses)	90 days	Short IRTs (<4 sec) more prevalent in P-b-*Ss* than in C-*Ss*, but did not result in different reward rates; Pb-*Ss* showed higher variability in response-rate under d-amphetamine treatment.

[a]Abbreviations:
C = control group
FI = Fixed interval
IRT = Inter response time
PND = Post-natal day
Ss = Subjects
? = Information not given

[b]Assuming consumption of 100 mL water/kg body weight (Arrington 1972).

TABLE 2 continued

Reference	Lead Exposure: Pb conc. (medium)	Lead Exposure: Period (route)	Estimated daily dose[b] (mg/kg/day)	paradigm (task)	Age at testing	Behavioral results
Cory-Slechta and Thompson (1979)	C = 0 Pb_1 = 0.0025% Pb_2 = 0.015% Pb_3 = 0.05% (water)	a. PND 20–70 or b. PND 20–150	C = 0 Pb_1 = 2.5 Pb_2 = 15 Pb_3 = 50	Operant (FI-30 sec)	55–140 days	Increased response rate and inter-*S* variability in groups $Pb_{1a,b}$ and Pb_2; decreased response rate in group Pb_3; effects in Pb_{1a} reversed after exposure terminated.
Cory-Slechta et al. (1981)	C = 0 Pb_1 = 0.005% Pb_2 = 0.015% (water)	PND 21–?	C = 0 Pb_1 = 5 Pb_2 = 15	Operant (minimum duration bar-press)	55–? days	Pb groups impaired: decreased response durations; increased response latencies; failure to improve performance by external stimulus control.
Milar et al. (1981b)		PND 4–31 (gavage)	C = 0 Pb_1 = 25 Pb_2 = 100 Pb_3 = 200	Operant (spatial alternation levers)	50 days	No sig. differences between C-*S*s and Pb-*S*s.

[a]Assuming consumption of 100 mL water/kg body weight (Arrington 1972).
Adapted from USEPA (1984).

TABLE 3
Neurological Effects of Direct Lead Exposure in Young Rats[a]

Exposure protocol	Estimated daily dose[b] (mg/kg/day)	Observed effects	Reference
0.1% Pb^{++} in chow PND 0–90	50	Decreased density of oligodendrocytes in cerebral cortex	Reyners et al. (1979)
Gastric intubation PND 2–14	75	1. 20% decline in striatal DA levels at PND 35 2. 35% decline in striatal DA turnover by PND 35 3. Transient depression of DA uptake at PND 15 4. Possible decreased DA terminal density	Jason and Kellogg (1981)
0.25–1% $Pb(Ac)_2$ in drinking water PND 0–60	250–1,000	1. 40–50% reduction of whole-brain ACh by PND 21 2. 36% reduction by PND 30 (return to normal values by PND 60)	Modak et al. (1978)
0.5–1% $Pb(Ac)_2$ in drinking water PND 0–60	500–1,000	1. Increased sensitivity to seizures induced by GABA blockers 2. Increase in GABA synthesis in crotex and striatum 3. Inhibition of GABA uptake and release by synaptosomes from cerebellum and basal ganglia 4. 70% increase in GABA-specific binding in cerebellum	Silbergeld et al. (1979, 1980a)
1% $PbCO_3$ in chow PND 0–60	500	Retardation of cortical synaptogenesis over and above any nutritional effects.	Averill and Needleman (1980)

[a]Abbreviations:
PND = Post-natal day
GABA = gamma aminobuyric acid
ACH = Acetyl choline
DA = Dopamine

[b]Assuming consumption of 100 mL water/kg/day and 50 g chow/kg/day (Arrington 1972).
Adapted from USEPA (1984).

the characteristic decrease in activity that normally occurs in pups at that age (Campbell et al., 1969; Melberg et al., 1976); thus, lead-exposed pups were significantly more active than control subjects at post-natal day 18.

These alterations in behavior are indicative of altered neural functioning in the CNS, but whether such alterations represent significant impairment in overall functioning of the lead-exposed subjects is not clear. As some studies indicate, lead-treated subjects may actually perform better than non-treated control subjects on certain learned tasks (Winneki et al., 1982b; Driscoll and Stegner, 1976; Gross-Selbeck and Gross-Selbeck, 1981). Taken as a whole, these studies suggest that lead exposure results in a tendency to respond more rapidly or "excessively," regardless of whether or not such responding is appropriate for the reinforcement contingencies of an experiment. However, this idea remains to be firmly established.

Acute lead intoxication resulting from a single high dose of lead is rare in humans. Symptoms of acute lead poisoning include vomiting, abdominal pain, hemolysis, liver damage and reversible tubular necrosis. Ingestion of a large but undetermined amount of red lead (lead tetroxide) by a 21-year-old male resulted in severe hemolysis and blood lead levels of 130 μg/dL before treatment was begun three days after ingestion (Nortier et al., 1980). Free hemoglobin levels in the serum were normal six days after exposure, and liver function tests were normal after four weeks.

A similar response to lead was observed after four drug addicts injected themselves intravenously with opium and lead acetate suspended in water (Beattie et al., 1979). Blood lead levels of 180 μg/dL were observed upon admission of one subject to the hospital ten days after injection of about 900 mg lead acetate. Symptoms included severe abdominal pain, headache and generalized limb pains. The time interval between injection of the drugs and onset of symptoms varied from two days in the two most severe cases to two months in the patients with mild illnesses. Liver lead values ranged from 111 to 824 mmol/kg liver wet weight (23 to 170 g/kg), as compared with values of 4.2 to 24.6 mmol/kg (0.87 to 5.1 g/kg) found in cases of industrial lead poisoning.

Subacute exposure may produce a syndrome of neurologic disorders. Early signs of neuropathy (which may develop within weeks of initial exposure) include dullness, restlessness, irritability, poor attention span, headaches, muscular tremor, hallucinations and loss of memory. These symptoms may progress to delirium, mania, convulsions, paralysis, coma and death. The onset of such symptoms can often be quite abrupt, with convulsions, coma and even death occurring very rapidly in patients who shortly before appeared to exhibit much less severe or no symptoms of acute lead intoxication (Cumings, 1959; Smith et al., 1938). Symptoms of lead encephalopathy indicative of severe CNS damage and posing a threat to life are generally not seen in adults except at blood lead levels well in excess of 100 to 200 μg/dL (Smith et al., 1938; Kehoe 1961a,b,c).

Long-Term Exposure. The main symptoms of chronic lead poisoning are abdominal pain, colic, vomiting, constipation, anemia and peripheral neuropathy. Whereas en-

cephalopathy and renal damage are considered the most serious complications of chronic lead toxicity in man (Nortier et al., 1980), the hematopoietic system may be more sensitive. Anemia resulting from decreased erythrocyte production and increased cell destruction is often the earliest manifestation of chronic lead poisoning.

Effects of Lead on Hematopoiesis. Lead inhibits several enzymes involved in heme biosynthesis. In adult humans, levels of 15 to 20 μg/dL and 25 to 30 μg/dL result in 40% and 70% inhibition of delta-aminolevulinic acid dehydratase (ALAD) activity, respectively. In children, 40% inhibition occurs at PbB values of 5 to 20 μg/dL, and 70% inhibition is observed at 20 to 25 μg/dL (Zielhuis, 1975). Granick et al. (1973) observed ALAD inhibition at PbB values of 15 μg/dL and higher, and similar results were reported by Hernberg and Nikkanen (1970). Measuring a decrease in erythrocyte ALAD activity correlates better with PbB values and is more sensitive than monitoring a decrease in hemoglobin (Selander and Cramer, 1970; Tola et al., 1973). Inhibition of ALAD results in increased urinary concentrations of delta-aminolevulinic acid (ALA). A significant increase in urinary ALA occurs at PbB values of 40 to 50 μg/dL (Tola, 1973; Selander and Cramer, 1970; Haeger-Aronsen et al., 1974).

Lead also interferes with the final step in heme biosynthesis, the chelation of iron by protoporphyrin IX. The work of Zielhuis (1975) shows that elevation of free erythrocyte porphyrin is a more sensitive indicator of lead exposure than is inhibition of ALAD. However, increased free erythrocyte porphyrin is not a specific indicator of lead-induced anemia (Roels et al., 1978).

A few studies have reported effects of lead on hemoproteins other than hemoglobin. For example, the rate of cytochrome P-450-mediated drug metabolism was depressed in two cases of lead poisoning (PbB = 60 and 72 μg/dL), but not in ten cases where lead levels ranged from 20 to 60 μg/dL (Alvares et al., 1975). Although other hemoproteins may be affected by lead, the evidence suggests that they are not sensitive to relatively low levels of exposure.

Effects of Lead on Blood Pressure. Hypertension is an important element in the etiology of cerebrovascular deaths. Dingwall-Fordyce and Lane (1963) reported a marked increase in the cerebrovascular mortality rate among heavily exposed lead workers as compared with the expected rate. These workers were exposed to lead during the first quarter of this century, when working conditions were poor.

Cramer and Dahlberg (1966) studied the incidence of hypertension in a population of 364 industrially exposed men in Sweden, 265 of whom had had long-term exposure to lead (ten or more years). They subdivided the workers into lead-affected and non-lead-affected groups, based on results from urinary coproporphyrin tests. There was no statistically significant difference in blood pressure between the groups, nor was the incidence of hypertension higher than that expected for nonexposed men in a similar population in Norway. Similar results (no evidence of increased hypertension among lead workers) have been described by Lane (1949) and Dreessen et al. (1941).

Beevers et al. (1980) reported results from an epidemiological study of hypertensive and normotensive persons in an area of Scotland where drinking water hardness is low

and water lead levels are high. They found a significant association for males between high PbB values and high blood pressure ($P < 0.05$). The mean PbB values for hypertensive and normotensive men were 1.31 and 1.14 μmol/L (27.1 and 23.7 μg/dL), respectively; for hypertensive and normotensive women, they were 1.09 and 0.94 μmol/L (219 and 194 μg/dL). In a similar study in England, ingestion of soft water with high lead levels did not result in elevated PbB values, and no relationship was found with high blood pressure.

In a review of five fatal cases of lead poisoning in young children, degenerative changes in heart muscle were reported to be the proximate cause of death (Kline, 1960). It is not clear, however, that such morphological changes are a specific response to lead intoxication. The overall evidence does not suggest that the heart is a critical target for lead effects.

Effects of Lead on the Renal System. Severe effects of lead on the renal system are seen both in persons dying of acute lead poisoning and in persons suffering from lead-induced anemia and/or encephalopathy. Effects are usually restricted to non-specific degenerative changes in renal tubular epithelial cells, with some degree of cellular necrosis. Cells of the proximal convoluted tubules are most severely affected.

Dysfunction of the proximal convoluted tubules (Fanconi's syndrome) is manifested by aminoaciduria, glycosuria and hyperphosphaturia. Chisolm (1962, 1968) found that nine of 23 children with lead encephalopathy had aminoaciduria, glycosuria and hypophosphatemia. Aminoaciduria was related to the severity of clinical toxicity and was most marked in children with encephalopathy. The aminoaciduria disappeared after treatment with chelating agents (Chisolm, 1962).

Clarkson and Kench (1956) investigated renal function in men who were occupationally exposed to lead fumes (lead oxide and/or lead chromate). They found no clinical evidence of renal disease; however, they did observe a modest but significant increase in aminoaciduria. The duration of exposure to lead and the PbB values were not given, but up to 500 μg/L of lead was excreted in the urine. In a series of 15 infants hospitalized for lead poisoning, three had aminoaciduria with PbB values of 246, 399 and 1,798 μg/dL (Chisolm, 1968).

Cramer et al. (1974) reported on a group of seven workers who had been exposed from less than one year to up to 30 years. Air lead concentrations in the workplace were not measured over the entire period, but were believed to exceed 0.2 mg/m^3. The average PbB was 100 μg/dL, ranging from 71 to 138 μg/dL, and all subjects had strikingly high urinary ALA excretion. Although aminoaciduria was not found and inulin clearance and renal blood flow were reported normal, some individuals with very long exposures were reported to have interstitial and peritubular fibrosis.

Cramer et al. (1974) suggested that there are at least two stages in the response of the human kidney to chronic lead exposure: (1) formation of nuclear inclusion bodies (probably reversible) with no impairment of renal function after short exposure; and (2) formation of interstitial fibrosis in the proximal tubular cells after four or more years of exposure. This latter stage is not characterized by any gross impairment of

renal function, but it is doubtful whether the morphological changes are completely reversible. Whether or not a third stage exists (i.e., renal failure) remains questionable (Cramer et al., 1974).

Reduced glomerular filtration with an attendant rise in serum urea concentration is generally considered to be symptomatic of chronic lead nephropathy. It is accompanied by interstitial fibrosis, obliteration of glomeruli and vascular lesions (Morgan et al., 1966) and occurs at low levels of lead exposure relative to the levels associated with aminoaciduria. For example, in Cramer's study of seven lead workers, none had aminoaciduria, while three had low renal clearance of inulin (Cramer et al., 1974). In another study of men with occupational lead exposure (air lead levels not stated), four of eight individuals with PbB values of 48 to 98 μg/dL had reduced glomerular filtration rates (Wedeen et al., 1975). Biopsies on three of the subjects showed proximal tubule abnormalities. One subject with a PbB of 48 μg/dL showed asymptomatic renal failure (elevated blood urea nitrogen and serum creatinine values). The PbB values of subjects with preclinical renal dysfunction (reduced glomerular filtration rate, but no elevation in blood urea or serum creatinine) ranged from 51 to 98 μg/dL. The average period of lead exposure was 4.25 years. On the basis of these studies, Wedeen et al. (1975) suggested that nephropathy may be an important occupational hazard in the U.S. lead industry.

In a series of 102 cases of lead poisoning studied by Lilis et al. (1968), undercompensated or decompensated renal failure was found in 17 patients, most of whom had been exposed to lead for more than 10 years. The mean PbB of the entire series was approximately 80 μg/dL, with a range from 42 to 141 μg/dL. The majority of this group had a history of several attacks of colic. Arterial hypertension followed chronic renal failure in 13 cases, with the renal impairment generally preceding the rise in blood pressure by several years.

Tabershaw and Cooper (1974) considered 7,032 workers who had been exposed to lead as a result of employment in either the battery or the lead-smelting industry between 1947 and 1970. Many were found to have PbB values in excess of 80 μg/dL. From a study of death records during the study period, the authors reported that there was excess mortality due to "chronic nephritis and other renal scleroses" and "other hypertensive disease" (uremia, nephrosclerosis and other renal disease).

Renal effects have also been observed in persons exposed to lead in domestic water supplies. In 970 households studied in Scotland, Campbell et al. (1977) found an association between elevated PbB values and renal insufficiency, as indicated by elevated serum urea concentration. Of the 970 households from which water samples were obtained, 249 had lead levels greater than 100 μg/L. Many samples exceeded this limit by a large margin, with the highest value being 8,500 μg/L. Among 54 age- and sex-matched pairs of subjects, those with elevated serum urea levels had PbB values in the range of 10.4 to 103 μg/dL. This compared to PbB values ranging from 10.4 to 62 μg/dL for those without elevated serum urea levels.

From these and other studies, it appears that the kidney is sensitive to lead-induced glomerular-vascular damage, with a threshold perhaps below a PbB value of 50 μg/dL. These renal conditions are considered to be precursors of hypertension.

Reproductive and Teratogenic Effects of Lead. It had been known since before the turn of the century that lead may exert adverse effects on human reproduction. Legge (1901) reported an increased frequency of miscarriages and stillbirths in women working in the lead trades during the latter half of the nineteenth century. Lane (1949) reported on the outcome of 15 pregnancies among 150 women working under conditions of moderate lead exposure (via air). Three of these women had miscarriages (an incidence seven times normal), although the numbers were too small to be statistically significant. A Japanese study among women with PbB values of 110 to 317 μg/dL indicated a greater incidence of miscarriages and reduced fertility, but there was no significant correlation with the PbB values (Nogaki, 1958).

The incidence of premature fetal membrane rupture in term and preterm infants was higher in an area 30 to 50 miles west of a lead mining area of Missouri urban area remote from lead mining activities (1% versus 0.41%) (Fahim et al., 1976). Maternal and fetal PbB values at birth differed significantly for normal births versus births with premature membrane rupture. Mean maternal and fetal PbB values for the normal deliveries were about 14 and 4 μg/dL, respectively, whereas the mean PbB values were about 26 and 13 μg/dL, respectively, for mothers and infants with membrane rupture.

Wibberley et al. (1977) examined a series of 126 births in Birmingham, England, and found that placental lead levels in the case of stillbirth or neonatal death were significantly higher than in the case of normal births. Placental levels were greater than 1.5 μg/g in only 7% of the normal births, whereas placental levels were greater than this in 61% of the stillbirths or neonatal deaths.

There is some evidence that lead affects fertility as well as the viability of the fetus. Lancranjan et al. (1975) reported that significant levels of teratospermia occurred among men working in a lead storage battery factory. Their PbB values were 41.0 to 74.5 μg/dL. The extent to which abnormally shaped spermatozoa affect fertilization, however, has not been established.

Effects of Lead on the Nervous System. It has been recognized for many years that chronic exposure to lead may result in severe neurological disorders. Overt neuropathy is usually associated with PbB values in excess of 60 μg/dL (Lilis et al., 1977; Irwig et al., 1978; Dahlgren et al., 1978; Baker et al., 1979; Haenninen et al., 1979; Spivey et al., 1979; Fischbein et al., 1980; Hammond et al., 1980). A number of workers have sought to assess the neurotoxic effects of lead using criteria more sensitive than overt neuropathy and there is a considerable body of data of this sort derived from animal studies. However, this material will not be discussed here, because there is also a large body of data from human studies that can be used, thereby avoiding the uncertainty associated with extrapolating from animals to humans.

Lead-Induced Neuropathy in Adults. Morgan and Repko (1974) reported deficits in hand-eye coordination and reaction time in an extensive study of behavioral functions in 190 lead-exposed workers (mean blood lead level = 60.5 μg/dL). The majority of the subjects were exposed for 5 to 20 years. Similar studies (Arnvig et al., 1980; Grandjean et al., 1978; Haenninen et al., 1978; Mantere et al., 1982; Valciukas et al., 1978) have found disturbances in visual motor performance, IQ test performance, hand dexterity, mood, nervousness and coping in lead workers with blood lead levels of 50 to 80 μg/dL. On the other hand, Milburn et al. (1976) found no differences between control and lead-exposed workers as judged by numerous psychometric and other performance tests.

Seppalainen et al. (1975) observed slowed nerve conduction velocity (NCV) in lead workers whose PbB levels were as low as 50 μg/dL and had never exceeded 70 μg/dL during the entire exposure period (mean × 4.6 years). Similarly, Melgaard et al. (1976) observed a clear association between lead exposure and peripheral nerve dysfunction in automobile mechanics exposed to tetra-ethyl lead and other compounds in lubricating and high-pressure oils. Half of the workers (10 of 20) had elevated PbB levels (60 to 120 μg/dL) and showed definite electromyographic deficits. The mean blood lead level for the control group was 18.6 μg/dL.

More recent studies by Araki et al. (1980), Ashby (1980), Bordo et al. (1982), Johnson et al. (1980), Seppalainen et al. (1979) and Seppalainen and Hernberg (1980, 1982) have confirmed a dose-dependent slowing of NCV in lead workers with PbB levels below 70 to 80 μg/dL. Sappalainen et al. (1979) observed NCV slowing in workers with blood lead levels ranging from 29 to 70 μg/dL, and Seppalainen and Hernberg (1980, 1982) found NCV slowing in workers with maximum PbB levels of 30 to 48 μg/dL, but not among workers with levels below 30 μg/dL. Buchthal and Behse (1979), Lilis et al. (1977) and Paulev et al. (1979), in contrast, found no signs of neuropathy at PbB levels below 80 μg/dL. Thus, considerable evidence exists that peripheral nerve dysfunction occurs in adults at PbB levels as low as 30 to 50 μg/dL. The question as to whether these reflect mild, reversible effects (Buchthal and Behse, 1981) or are true early warning signals of progressively more serious peripheral neuropathies (Feldman et al., 1977; Sappalainen and Hernberg, 1980) is still a matter of some dispute. Nevertheless, it is clear that these effects represent a departure from normal neurologic functioning, and their potential relationship to more serious effects establishes a basis for prudence in interpreting their potential health significance.

Lead-Induced Neuropathy in Children. Overt symptoms of encephalopathy similar to those that occur in adults have been reported to occur in infants and young children (Prendergast, 1910; Blackfan, 1917; McKhann and Vogt, 1926; Giannattasio et al., 1952; Cumings, 1959; Tepper, 1963; Chisolm, 1968). A markedly higher incidence of severe encephalopathic symptoms and death among lead-exposed children than adults may reflect the greater difficulty of recognizing early symptoms in young children (Lin-Fu, 1973). Available data indicate that PbB levels associated with acute encephalopathy symptoms are lower among children than adults. The most extensive

compilation of information on a pediatric population is a summarization by NAS (1972) of data from Chisolm (1962, 1965) and Chisolm and Harrison (1956). This data compilation relates occurrence of acute encephalopathy and death in children in Baltimore to PbB levels determined by the Baltimore City Health Department. Signs of encephalopathy (hyperirritability, ataxia, convulsions, stupor and coma) were associated with PbB levels of approximately 90 to 800 μg/dL (mean = 330 μg/dL). The distribution of PbB levels associated with death (mean = 327 μg/dL) was essentially the same as for levels associated with encephalopathy. These data suggest that PbB levels capable of producing death in children are essentially identical to those associated with acute encephalopathy, and that such effects are usually manifested in children starting at PbB levels of approximately 100 μg/dL. Other evidence from scattered medical reports (Gant, 1938; Smith et al., 1938; Bradley et al., 1956; Bradley and Baumgartner, 1958; Cumings, 1959; Rummo et al., 1979) suggests that acute encephalopathy in the most highly susceptible children may be associated with blood lead levels in the range of 80 to 100 μg/dL.

Lasting neurotoxic sequelae of overt lead intoxication in children in the absence of acute encephalopathy have also been reported. Byers and Lord (1943), for example, reported that 19 out of 20 children with previous lead poisoning later made unsatisfactory progress in school, presumably due to sensorimotor deficits, short attention span and behavioral disorders. These effects have since been confirmed in children with known high exposures to lead, but without a history of life-threatening forms of acute encephalopathy (Chisolm and Harrison, 1956; Cohen and Ahrens, 1959; Kline, 1960).

Perlstein and Attala (1966) reported neurological sequelae in 140 of 386 children (37%) following lead poisoning without encephalopathy. Sequelae included mental retardation, seizures, cerebral palsy, optic atrophy and visual-perceptual problems with minimal intellectual impairment. The severity of sequelae was related to the severity of the earlier observed symptoms.

De la Burde and Choate (1972, 1975) observed neurological dysfunctions, fine motor dysfunction, impaired concept formation and altered profiles in 70 preschool children exhibiting pica. These children displayed elevated PbB levels (above 30 μg/dL in all cases; mean = 59 μg/dL) in comparison with matched control subjects not engaging in pica. Subjects were drawn from the Collaborative Study of Cerebral Palsy, Mental Retardation and Other Neurologic Disorders of Infancy and Childhood (Broman et al., 1975). In a follow-up study on the same children (at seven to eight years of age), De la Burde and Choate (1975) reported continuing CNS impairment in the lead-exposed group, as assessed by a variety of physiological and neurological tests. In addition, seven times as many lead-exposed children were repeating grades in school or being referred to the school psychologist, despite the observation that many of their PbB levels had by then decreased significantly from the initial study.

Odenbro et al. (1983) studied the psychological development of children (aged three to six years) seen in Chicago Department of Health clinics (August, 1976 to February,

1977). These workers evaluated scores from the Denver Development Screening Test (DDST) and Wechsler IQ scale (WPPSI) in relation to PbB levels obtained by repeated sampling during the three previous years. A significant correlation ($r = 0.435$, $P < 0.001$) was reported between decreased perceptual-visual-motor ability and mean PbB levels. Statistically significant ($P < 0.005$) deficits in verbal productivity and perceptual-visual-motor performance (measured by the WPPSI) were found for children with mean PbB levels of 30 to 40 μg/dL, versus control children with mean PbB levels below 25 μg/dL. These results are highly suggestive that neuropsychologic deficits are associated with PbB levels of 30 to 60 μg/dL in preschool children.

A population study was reported by Needleman et al. (1979), who used shed deciduous teeth as an index of lead exposure. Teeth were donated from 70% of a total population of 3,329 first and second grade children from two towns near Boston. Almost all children who donated teeth (2,146) were rated by their teachers on an eleven-item classroom behavior scale devised by the authors to assess attention disorders. A preliminary analysis indicated a dose-response relationship between dentine lead content and behavior test results. A more detailed analysis was performed on 100 children with low dentine lead (<10 ppm) and 58 children with high dentine lead (>20 ppm). After consideration of a number of significant covariates (e.g., parental IQ and education), a significant difference ($P < 0.05$) between the low-lead and high-lead groups was found for two different IQ tests, 9 of the 11 behavioral tests and several measures of perceptual-motor behavior. In a subsequent report, Needleman (1982) demonstrated that the entire distribution of IQ scores was shifted downward in the high-lead group, with none of the children having a verbal IQ over 125.

Yule et al. (1981) studied the effects of lead exposure in a population of 195 children (aged 6 to 12 years). The PbB concentrations ranged from 7 to 32 μg/dL, and the children were assigned to one of four quartiles having the following PbB values: 7 to 10 μg/dL; 11 to 12 μg/dL; 13 to 16 μg/dL and 17 to 32 μg/dL. The tests of achievement and intelligence were similar to those used by Needleman et al. (1979). There were significant associations between PbB levels and scores on tests of reading, spelling and intelligence, but not of mathematics (Yule et al., 1981). In a subsequent report, Yule et al. (1983) dealt with the results pertinent to the attention deficits. While there were few differences between groups on the Rutter Scale, the summed scores on the Needleman questionnire across PbB groupings approached significance ($P < 0.096$). Three of the questionnaire items showed a significant dose-response function ("Day Dreamer," "Does not Follow Sequence of Direction" and "Low Overall Functioning"). Nine of the eleven items were highly correlated with the children's IQ. Therefore, the Needleman questionnaire may be identifying IQ-related attention deficits, as opposed to measures of conduct disorders and socially maladaptive behavior (Yule et al., 1983). The authors noted that caution is necessary in interpreting their findings, in view of the crudity of the available measures of social factors and differences between countries in diagnosing attention deficit disorders.

Yule and Landsdown (1983) reported the results from a second and better designed study using similar methods and procedures with 194 children. These subjects lived

near a busy roadway in a predominantly lower middle-class area of London. In contrast to the first pilot study, no statistically significant relationships were observed between PbB values and test achievement. Still, the authors stated there was some relationship between PbB levels and intelligence in working-class groups, but whether these are of a causal nature in either direction is unclear.

In a study by Winneke et al. (1982a), incisor teeth were donated by 458 children, aged seven to ten years, in Duisburg, Germany. Two extreme exposure groups were formed: a low-lead group, with 2.4 μg/g mean tooth lead level ($n = 26$), and a high-lead group, with 7 μg/g mean tooth lead level in ($n = 16$). The authors found a marginally significant decrease ($P < 0.10$) of five to seven IQ points and a significant decrease in perceptual-motor integration ($P < 0.05$).

Winneke et al. (1982b) carried out a study which involved 115 children living in the lead smelter town of Stolberg. Tooth lead (mean value = 6.16 ppm, range = 2.0 to 38.5 ppm) and PbB levels (mean = 13.4 μg/dL) were significantly correlated ($r = 0.47$; $P < 0.001$) for the children studied. Using stepwise multiple regression analysis, the authors found significant ($P < 0.05$) or marginally significant ($P < 0.10$) associations between tooth lead levels and measures of perceptual-motor integration, reaction time performance and four behavioral rating dimensions, including distractability.

Burchfiel et al. (1980), using computer-assisted spectral analysis of a standard EEG examination of 41 children from the study of Needleman et al. (1979), reported significant EEG spectrum differences in percentages of low-frequency delta activity and in spontaneous alpha activity of the high-lead children. Percentages of alpha- and delta-frequency EEG activity and results for several psychometric and behavioral testing variables (e.g., WISC-R full-scale IQ and verbal IQ, reaction time under varying delay) for the same children were then employed as input (or "features") in direct and stepwise discriminant analyses. The separation determined by these analyses for combined psychological and EEG variables ($P < 0.005$) was reported to be strikingly better than the separation of low-lead from high-lead children obtained using either psychological ($P < 0.041$) or EEG ($P < 0.79$) variables alone. Unfortunately, no dentine lead or PbB values were reported.

The relationship between low-lead exposure and neurological function (including electrophysiological response) in children aged 13 to 75 months was extensively explored in a study conducted at the University of North Carolina in collaboration with the U.S. Environmental Protection Agency (Milar et al., 1980; 1981). Psychometric evaluation revealed lower IQ scores for children with PbB levels of 30 μg/dL or higher compared with children with PbB levels under 30 μg/dL. The observed IQ deficits were confounded by poor home caregiver environment scores in children with elevated PbB levels (Milar et al., 1980). No relationship between blood lead and hyperactive behavior (as indexed by standardized playroom measures and parent-teacher rating scales) was observed in these children (Milar et al., 1981).

Electrophysiological assessments, including analyses of slow potentials during sensory conditioning (Otto et al., 1981) and EEG spectra (Benignus et al., 1981), did provide

evidence of CNS effects of lead in the same children. In contrast to psychometric and behavioral findings, a significant linear relationship between PbB (ranging from 6 to 59 μg/dL) and slow wave (SW) voltage was observed (Otto et al., 1981). Analyses of quadratic and cubic trends in SW voltage did not reveal any evidence of a threshold for this effect. The slope of the PbB versus SW voltage function decreased with age. No effect of blood lead levels on EEG power spectra or coherence measures was observed, but the relative amplitude of synchronized EEG between left and right hemispheres (gain spectra) increased relative to PbB levels (Benignus et al., 1981). A significant cubic trend for gain between the left and right parietal lobes was found, with a major inflection point at 15 μg/dL. These findings suggest that EEG gain is altered at PbB levels as low as 15 μg/dL, although the clinical and functional significance of this measure has not been established.

A follow-up study of slow cortical potentials and EEG spectra in a subset (28 children, aged 35 to 93 months) of the original group was carried out two years later (Otto et al., 1982). Slow wave voltage during sensory conditioning again was a linear function of blood lead, even though the mean PbB level had declined by 11.4 μg/dL (from 32.5 μg/dL to 21.1 μg/dL). The similarity of SW results obtained at initial and follow-up assessments suggests that the observed alterations in this parameter of CNS function are persistent, despite a significant decrease in the mean PbB level during the two-year interval. No significant relationship between a child's IQ and EEG measures was found in the initial (Benignus et al., 1981; Otto et al., 1981) or follow-up studies. Slow wave voltage and EEG gain thus appear to provide CNS indices of lead exposure effects that may be more sensitive than and independent from the standardized psychometric measures used in other studies.

Further evidence for lead-induced peripheral nerve dysfunction in children is provided by a study by Landrigan et al. (1976) of children living in close proximity to a smelter in Idaho. No clearly abnormal conduction velocities were observed, although a statistically significant negative correlation was found between peroneal NCV and PbB levels ($r = -0.38$, $P < 0.02$ by one-tailed t-test). These results provide evidence for significant slowing of NCV (and, presumably, for advancing peripheral neuropathy) as a function of increased PbB levels, but do not allow clear statements to be made regarding a threshold for pathologic slowing of NCV.

Prenatal exposure of the fetus at PbB levels below those causing fetotoxicity may result in damage to the brain. Beattie et al. (1975) identified 77 retarded children and 77 normal children matched for age, sex, and geography. Of 64 matched pairs, 11 of the retarded children came from homes in which the concentration of lead in the "first flush" water exceeded 800 μg/L. By contrast, none of the control children came from such homes. In a follow-up study, PbB values of the mental retardates, taken during the second week of life, were found to be significantly higher than those of control subjects (25.5 ± 8.9 vs. 20.9 ± 7.9 μg/dL) (Moore et al., 1977). When compared with the studies of children presumably suffering neurobehavioral deficits from direct exposure to lead, these studies suggest that the brain of the fetus is considerably more sensitive to the toxic effects of lead than the brain of the young child.

QUANTIFICATION OF NON-CARCINOGENIC EFFECTS

One-Day Health Advisory. No studies were located which were suitable for calculation of a one-day Health Advisory for either children or adults. Values calculated below for ten-day Health Advisories will be taken as conservative estimates of appropriate one-day Health Advisories.

Ten-Day Health Advisory. Effects of short-term lead exposure are not well studied, but a number of workers have observed behavior or neurological changes in young animals exposed to lead through food or water at various times from post-natal day zero through post-natal day 150 (see Tables 2 and 3). The studies of Cory-Slechta and Thompson (1979) and Cory-Slechta et al. (1981) have been selected to serve as the basis for calculation of the ten-day Health Advisory for children. These studies show that in weanling rats exposed to lead via drinking water, detectable behavioral effects are produced by water concentrations of lead as low as 0.0025% (25 mg/L) after as few as 30 days of exposure. This corresponds to an average daily dose of about 2.5 mg/kg/day (assuming daily water consumption of 100 mL/kg) (Arrington, 1972). Other studies (Tables 2 and 3) were not selected, since the estimated exposure levels were higher. No suitable study regarding short-term exposure in children was located. Gastrointestinal absorption rates do not need to be considered in this calculation, since the LOAEL is based on effects observed following oral ingestion.

The ten-day HA for the 10-kg child is calculated as follows:

$$\frac{(2.5 \text{ mg/kg/day})\ (10 \text{ kg})}{(1 \text{ L/day})\ (1000)} = 0.025 \text{ mg/L } (25\ \mu\text{g/L})$$

where:

2.5 mg/kg/day = LOAEL, based on behavioral effects observed in weanling rats exposed to lead *via* drinking water (Cory-Slechta and Thompson, 1979).

10 kg = assumed weight of a child.

1 L/day = assumed water consumption by a 10-kg child.

1000 = uncertainty factor. This factor was selected in accordance with ODW/NAS guidelines for use with a LOAEL from an animal study.

The studies by Govoni et al. (1979, 1980), Memo et al. (1980a, 1981) and Grant et al. (1980) have been selected to serve as the basis for calculation of the ten-day Health Advisory values for adults. These studies provide evidence for altered nuerotransmitter metabolism and behavior traits in neonatal rats exposed to lead by supplying the dams with water containing 0.004% to 0.005% lead acetate during gestation and/or during post-natal development. This corresponds to an average daily dose to the mother of about 4 to 5 mg/kg/day (assuming consumption of 100 mL/kg/day) (Arrington, 1972). On this basis, 4 mg/kg/day is taken as the LOAEL in animals. No suitable short-term study in humans was located. Gastrointestinal absorption rates do not need to be considered in calculating the HA values, since the LOAEL is based on effects observed following oral exposure via drinking water.

The ten-day HA for the 70 kg adult is calculated as follows:

$$\frac{(4\ \text{mg/kg/day})\ (70\ \text{kg})}{(2\ \text{L/day})(1000)} = 0.14\ \text{mg/L}\ (140\ \mu\text{g/L})$$

where:

4 mg/kg/day = LOAEL, based on altered neurotransmitter metabolism and behavior in rat pups born to dams exposed to lead via drinking water during gestation (Govoni et al., 1979, 1980; Memo et al., 1980a, 1981; Grant et al., 1980).

70 kg = assumed weight of an adult

2 L/day = assumed water consumption by a 70 kg adult

1000 = uncertainty factor. This uncertainty factor was selected in accordance with ODW/NAS guidelines for use with a LOAEL from an animal study.

Adjusted Acceptable Daily Intake. Because so many body systems are affected by chronic exposure to lead, and because so many studies provide data regarding the PbB threshold for toxic effects, it does not seem appropriate to select a single study as the basis for calculation of the ADI. Rather, it appears more appropriate to identify a "consensus" PbB value that represents the collective indication of the NOAEL based on all relevant studies in humans. Tables 4 and 5 summarize the adverse or potentially adverse effects associated with various PbB levels in adults and children, respectively. From these tables, it is apparent that effects on the enzymes of heme synthesis occur at very low PbB levels (10 μg/dL), and that subtle signs of neurological disorder begin to appear at 15 to 20 μg/dL in children and at 25 to 30 μg/dL in adults. A number of more obvious effects become manifest in several body systems at PbB levels of 40 μg/dL and higher. Taken as a whole, this body of data suggests that PbB values of 15 μg/dL should be exceeded in children, and values of 25 μg/dL should not be exceeded in adults. In order to protect the fetus, however, it is necessary to set the PbB level in adults at 15 μg/dL as well, since several studies (Harris and Holley, 1972; Gershanik et al., 1974; Hubermont et al., 1978) indicate that the ratio of fetal/maternal PbB values is close to 1:1. Thus, a PbB level of 15 μg/dL is selected as the NOAEL.

Using a PbB value of 15 μg/dL as the NOAEL in humans, the average daily oral exposure that produces this PbB value can be calculated. A number of reports have been published which describe the relationship between average daily oral ingestion of lead and PbB values.

Studies in infants and toddlers are generally simpler than studies in older children or adults, due to the limited dietary intake (formula or breast milk) and the lower incidence of pica (which represents a significant source of lead ingestion in older children). Unfortunately, in three studies in infants and toddlers (Table 6), significant differences were observed in relationships (i.e., the slope of the line) between oral ingestion of lead from the diet (PbB) and blood lead levels (PbB). For example, it is not even clear whether the relationship is linear or curvilinear (dependent on the 1/3 power of daily intake). Most of the subjects in the Sherlock et al. (1982) and United Kingdom Central

TABLE 4
Summary of Health Effects in Adults Following Chronic Lead Exposure[a]

Lowest observed effect level (PbB)	Heme synthesis and hematological effects	Neurological effects	Renal system effects	Reproductive function effects	Gastrointestinal effects
100–120 μg/dL		Encephalopathic signs and symptoms	Chronic renal nephropathy		Overt gastrointestinal symptoms (colic, etc.)
80 μg/dL	Frank anemia				
60 μg/dL					
50 μg/dL	Reduced hemoglobin production	Overt subencephalopathic neurological symptoms		Altered testicular function	
40 μg/dL	Increased urinary ALA and elevated coproporphyrins				
30 μg/dL		Peripheral nerve dysfunction (slowed nerve conduction)			
25–30 μg/dL	Erythrocyte protoporphyrin (EP) elevation in males				
15–20 μg/dL	Erythrocyte protoporphyrin (EP) elevation in females				
<10 μg/dL	ALA-D inhibition				

[a]Abbreviations: PbB = blood lead concentrations.
Adapted from USEPA (1984).

TABLE 5
Summary of Health Effects in Children Following Chronic Lead Exposure[a]

Lowest observed effect level (PbB)	Heme synthesis and hematological effects	Neurological effects	Renal system effects	Gastrointestinal effects
80–100 μg/dL		Encephalopathic signs and symptoms	Chronic nephropathy (aminoaciduria)	Colic, other overt gastrointestinal symptoms
70 μg/dL	Frank anemia			
60 μg/dL		Peripheral neuropathies		
50 μg/dL				
40 μg/dL	Reduced hemoglobin	Cognitive (CNS) deficits		
	Elevated corpoporphyrin	Peripheral nerve dysfunction (slowed NCV's)		
	Increased urinary ALA			
30 μg/dL			Vitamin D metabolism interference	
15 μg/dL	Erythrocyte protoporphyrin elevation	Altered CNS electrophysiological responses		
10 μg/dL	ALA-D inhibition			
	Py-5-N activity inhibition			

[a]Abbreviations: PbB = blood lead concentrations; Py-5-N = pyrimidine-5'-nucleotidase.
Adapted from USEPA (1984).

Directorate on Environmental Pollution (1982) studies received relatively high PbB levels (>300 μg/day). The fitted cube root equations give high slopes at lower PbB levels. On the other hand, the linear slope of the United Kingdom Central Directorate on Environmental Pollution (1982) study is probably an underestimate of the slope at low PbD levels. In the study by Ryu et al. (1983), a steady-state PbB value of about 7 μg/dL was observed in infants ingesting milk from cartons (total PbD = 16 μg/day). In another group of infants, a PbB value of about 14 μg/day was attained when the milk was supplied from cans (total PbD = 61 μg/day). From the increase in PbB (7 μg/dL) resulting from the increase in PbD (45 μg/day), the relationship shown in Table 6 (PbB = 0.16 PbD) is derived. Since these PbD values are relatively low (16 to 61 μg/day) and involve PbB values in the range of interest (around 15 μg/dL), this equation is judged to be most appropriate for relating oral lead intake levels in infants and children to PbB values.

The relationship between oral ingestion of lead and PbB values in adults is even less certain. Tables 7, 8 and 9 summarize some of the relevant studies. For dietary intake (Table 7), most slopes relating PbD to PbB are about 0.02 μg/dL per μg/day. The relation between PbB values and water lead concentration (PbW) is not clearly defined and is often described as nonlinear (Tables 8 and 9). Many authors choose to fit cube root models to their data, although polynomial and logarithmic models are also used.

Unfortunately, the form of the model employed greatly influences the estimated contributions to PbB from relatively low PbW values. The only study that determines the relationship based on low drinking water lead values (<100 μg/L) is that by Pocock et al. (1983). That data from this study suggest that in this lower concentration range, the relationship is linear. Furthermore, the estimated contributions to PbB levels from this study are roughly consistent with the polynomial models from other studies. For these reasons, the Pocock et al. (1983) slope of 0.06 is considered to be the most appropriate for use at low water lead concentrations.

Calculation of ADI for Infants and Children:

Using the relationship derived from the data of Ryu et al. (1983) (PbB = 0.16 PbD), the ADI for infants and children may be calculated as follows:

$$\frac{(15\ \mu g/dL)}{[1\ \mu g/dL)/(0.16\ \mu g/day)](5)} = 19\ \mu g/day$$

where:

15 μg/dL = blood lead level (PbB) at which no adverse effects are observed (see Tables 4 and 5).

$\frac{1\ \mu g/dL}{0.16\ \mu g/day}$ = relation between lead intake in the diet (PbD) and blood lead level (PbB) in infants (Ryu et al., 1983).

5 = uncertainty factor. This factor was selected to account for intra-human variability, uncertainty with respect to selection of the NOAEL (as PbB) and uncertainty with respect to calculation of an oral intake equivalent to the NOAEL. The rationale for this uncertainty factor is detailed below.

TABLE 6
Empirical Relationships between Dietary Lead Intake and Blood Lead Levels in Infants and Toddlers

Reference	Relationship[a]
Sherlock et al. (1982)	$PbB = 2.5 + 5(PbD)^{1/3}$
UKCD (1982)	$PbB = 17.1 + 0.56(PbD)$ $PbB = 3.9 + 4.6(PbD)^{1/3}$
EPA (1984), based on Ryu et al. (1983)	$PbB = 0.16\,PbD$

[a]Abbreviations: PbB = blood lead concentrations (μg/dL).
PbD = daily dietary intake (μg/day).
Adapted from USEPA (1984).

According to ODW/NAS guidelines, an uncertainty factor of 10 is normally employed when a NOAEL in humans is used as the basis for calculation of the ADI and AADI values. This factor is intended to account for intra-human variation in sensitivity to the toxicant. However, in this case, the NOAEL has been selected in order to protect the most sensitive portion of the population (infants and pregnant women). Therefore, it is judged that no uncertainty factor is required to account for this consideration.

There are, however, two other sources of uncertainty associated with this calculation that do require consideration. The first source of uncertainty is selection of the blood lead level (PbB) which represents a NOAEL in children and pregnant females. The adverse effects of lead appear to be described by a dose-response continuum with no clear threshold value. Because the PbB value selected (15 μg/dL) is associated only with subtle, pre-clinical effects, it is judged that there is relatively little uncertainty associated with selection of this value. The second source of uncertainty lies in the calculation of an oral intake which corresponds to the selected PbB value. Since there is considerable variation in the mathematical relationships which have been derived between oral lead intake and PbB, it is judged that significant uncertainty exists in this calculation. An overall uncertainty factor of 5 was selected to account for all of these considerations.

Calculation of ADI for Adults:

Using the relationship derived from the data of Pocock et al. (1983) (PbB = 0.062 PbW), the ADI for adults may be calculated as follows:

$$\frac{(15\ \mu g/dL)}{[1\ \mu g/dL)/(0.062\ \mu g/day)](5)} = 48\ \mu g/day$$

where:

15 μg/dL = blood lead level at which no adverse effects are observed (see Tables 4 and 5).

$\frac{1\ \mu g/dL}{0.062\ \mu g/day}$ = relation between lead intake in water (PbW) and blood lead level (PbB) in adults (Pocock et al., 1983).

TABLE 7
Studies Relating Blood Lead Levels to Experimental Dietary Intakes

Study	Subjects	Exposure	Chemical Form	Blood Level		Slope[a] (μg/dL per μg/day)
				Initial	Final	
Stuik (1974)	5 adult male students	20 μg Pb/kg/day—21 days	Lead acetate	20.6	40.9	0.017[b,c]
	5 adult female students	20 μg Pb/kg/day—21 days	Lead acetate	12.7	30.4	0.018[b,c]
	5 adult male students	Controls	Placebo	20.6	18.4	—
	5 adult female students	20 μg Pb/kg/day	Lead acetate	17.3	41.3	0.002
	5 adult male students	30 μg Pb/kg/day	Lead acetate	16.1	46.2	0.014
	5 adult female students	Controls	Placebo	~17.0	~17.0	—
Cools et al. (1976)	11 adult males	30 μg Pb/kg/day ~7 days	Lead acetate	17.2	26.2	0.027[c]
	10 adult males	Controls	Placebo		~19.0	—
Schlegel and Kufner (1979)	1 adult male	50 μg Pb/kg/day—6 wk	Lead nitrate	16.5	64.0	0.014[b,c]
	1 adult male	70 μg Pb/kg/day—13 wk	Lead nitrate	12.4	30.4	0.004[d]
Gross (1979) analysis of Kehoe's experiments	1 adult male	300 μg/day	Lead acetate		−1	[0]
	1 adult male	1,000 μg/day	Lead acetate		+17	0.017
	1 adult male	2,000 μg/day	Lead acetate		+33	0.016
	1 adult male	3,000 μg/day	Lead acetate		+19	0.006[e]

[a]Exposure (μg/d) = Exposure (μg/kg/day) × 70 kg for males, 55 kg for females. Slope = (Final − Initial Blood Lead)/Exposure (μg/d).

[b]Corrected for decrease of 2.2 μg/dL in control males.

[c]Assumed mean life 40d. This increases slope estimate for short-term studies. Stuik Study I would be 0.042, 0.044 respectively for males, females.

[d]Assumed limited absorption of lead.

[e]Removed from exposure before equilibrium.

Adapted from USEPA (1984).

TABLE 8
Studies Relating Blood Lead Levels (μg/dL) to First-Flush Water Lead (μg/L)

Study	Analysis	Model[a]	Estimated blood lead at PbW = 0	Predicted blood lead contribution (μg/dL for a given water lead (μg/L)			
				5	10	25	50
Worth et al. (1981) study of 524 subjects in greater Boston. Water leads (standing water) ranged from <13 to 1,108 μg/L. Blood leads ranged from 6 to 71.	Worth et al. (1981)	$\ell n(PbB) = 2.729\,PbW - 4.699\,(PbW)^2 + 2.116\,(PbW)^3$ + other terms for age, sex, education, dust (PbW is in mg/L).	20.5	0.3	0.6	1.4	2.7
	EPA	$\ell n(PbB) = \ell n(.041\,PbW = .000219\,(PbW^2)$ + other terms for age, sex, education, dust)	21.1	0.2	0.4	1.0	2.1
Moore et al. (1979) study of 949 subjects from different areas of Scotland. Water leads were as high as 2,000 μg/L.	Moore et al. (1979)	$PbB = 11.0 + 2.36\,(PbW)^{1/3}$	11.0	4.0	5.1	6.9	8.7
Hubermont et al. (1978) study of 70 pregnant women in rural Belgium. Water leads ranged from 0.2 to 1,228.5 μg/L. Blood leads ranged from 5.1 to 26.3 μg/dL.	Hubermont et al. (1978)	$PbB = 9.62 + 0.756\ell n\,(PbW)$	8.4[b]	2.4	3.0	3.7	4.2
U.K. Central Directorate (1982) study of 128 mothers in greater Glasgow. Water leads ranged from under <10, 1,060 μg/L. Blood leads ranged from 2 to 39 μg/dL.	U.K. Cen. Dir. (1982)	$PbB = 13.2 + 1.8\,(PbW)^{1/3}$	13.2	3.1	3.9	5.3	6.6
	U.K. Cen. Dir. (1982)	$PbB = 18.0 + 0.009\,PbW$	18.0	0.0	0.1	0.2	0.4
	EPA	$\ell n(PbB) = \ell n(14.2 + 0_2033\,PbW = 0.000031\,PbW^2)$	14.2	0.2	0.3	0.8	1.6

U.K. Central Directorate (1982) study of 126 infants (as above). Blood leads ranged from 1 to 51 μg/dL.	U.K. Cen. Dir. (1982)	$PbB = 9.4 + 2.4 (PbW)^{1/3}$	9.4	4.1	5.2	7.0	8.8
	U.K. Cen. Dir. (1982)	$PbB = 17.2 + 0.018 PbW$	17.1	0.1	0.2	0.4	0.9
	EPA	$\ell n(PbB) = \ell n(12.0 + 0.050 PbW - 0.000045 PbW^2)$	12.0	0.2	0.5	1.2	2.4
Thomas et al. (1979) study of 115 adult Welsh females. Water leads ranged from <10 to 2,800 μg/dL. Blood leads ranged from 5 to 65 μg/dL.	EPA	$\ell n(PbB) = 14.9 + 0.041 PbW - 0.000012 PbW^2)$	14.9	0.2	0.4	1.0	2.0
Moore (1977) study of 74 residents of a Glasgow tenement.	Moore (1977)	$PbB = 15.7 + 0.015 PbW$	15.7	0.1	0.2	0.4	0.8
Pocock et al. (1983) study of 7,735 men aged 40–59 in Great Britain. Water leads restricted to <100 μg/L.	Pocock et al. (1983)	$PbB = 14.48 + 0.062 PbW$	14.5	0.3	0.6	1.6	3.1

[a]Abbreviations: PbB = blood lead concentration (μg/dL), PbW = water lead concentration (μg/L).
[b]Minimum water lead of 0.2 μg/dL used instead of 0.
Adapted from USEPA (1984).

TABLE 9
Studies Relating Blood Lead Levels (μg/dL) to Running Water Lead (μg/dL)

Study	Analysis	Model[a]	Estimated blood lead at PbW = 0	Predicted blood lead contribution (μg/dL for a given water lead (μg/L)			
				5	10	25	50
Worth et al. (1981) study of 524 subjects in greater Boston. Water leads (standing water) ranged from <13 to 1,108 μg/L. Blood leads ranged from 6 to 71.	EPA	ℓn (PbB) = (0.0425 PbW + other terms for age, sex, education and dust)	21.3	0.2	0.4	1.1	2.1
Moore et al. (1981) study restricted to 390 subjects aged 20 or older.	U.S. EPA 1980)	PbB = 14.33 + 2.541 $(PbW)^{1/3}$	14.3	4.4	5.4	7.4	9.4
	EPA	ℓn (PbB) = ℓn (18.6 + 0.071 PbW)	18.6	0.4	0.7	1.8	3.6
	EPA	ℓn (PbB) = ℓn (0.073	18.8	0.4	0.7	1.8	3.7
Worth et al. (1981) study restricted to 249 females ages 20 to 50.	U.S. EPA (1980)	PbB = 13.38 + 2.487 $(PbW)^{1/3}$	13.4	4.3	5.4	7.3	9.2
	EPA	ℓn (PbB) + ℓn (17.6 + 0.067 PbW)	17.6	0.3	0.7	1.7	3.4
	EPA	ℓn (PbB) = (0.067 PbW + other terms for education and dust)	17.6	0.3	0.7	1.7	3.4
U.K. Central Directorate (1982) study of 128 mothers in greater Glasgow. Water leads ranged from 20 to 720 μg/L. Blood leads ranged from 1 to 39 μg/dL.	U.K. Cen. Dir. (1982)	PbB = 12.8 + 1.8 $(PbW)^{1/3}$	12.8	3.1	3.9	5.3	6.6
	U.K. Cen. Dir. (1982)	PbB = 18.1 + .014 PbW	18.1	0.1	0.4	0.4	0.7
	EPA	ℓn (PbB) = ℓn (13.4 + 0.071 PbW $-0.000104\ PbW^2$)	13.4	0.4	0.7	0.7	3.3

Moore (1977) study of 75 residents of a Glasgow tenement.	Moore (1977)	PbB = 16.6 + 0.02 PbW	16.6	0.1	0.2	0.5	1.0
Sherlock et al. (1982) study of 114 adult women. Blood leads ranged <5 to >61 μg/dL. Kettle water leads ranged from <10 to >2,570 μg/L.	Sherlock et al. (1982)	PbB = 4.7 + 2.78 $(PbW)^{1/3}$	4.7	4.8	6.0	8.1	10.2
	EPA	$\ell n(PbB) = \ell n(11.5 + 0.033\,PbW - 0.00001\,PbW^2)$	11.5	0.2	0.3	0.8	1.6
Sherlock et al. (1984) follow-up study.	Sherlock et al. (1984)	PbB = 5.6 + 2.62 $(PbW)^{1/3}$	5.6	4.5	5.6	7.7	9.7

[a]Minimum water lead of 0.2 μg/dL used instead of 0.
Adapted from USEPA (1984).

5 = uncertainty factor. The basis of this factor is detailed above, in the calculation of the ADI for children.

Calculation of AADI: Using 19 μg/day as the ADI in infants and children, and assuming consumption of one liter of water per day by a 10-kg child, the AADI for infants and children is calculated as follows:

$$\frac{19\ \mu g/day}{1\ L/day} = 19\ \mu g/L$$

Using 48 μg/day as the ADI in adults, and assuming consumption of two liters of water per day by a 70-kg adult, the AADI for adults is calculated as follows:

$$\frac{48\ \mu g/day}{2\ L/day} = 24\ \mu g/L$$

Choosing the lower valuc in order to protect both groups, the AADI for both children and adults is taken to be 19 μg/L.

Consideration of Relative Source Data. The calculations above assume that 100% of the lead exposure occurs from ingestion of drinking water. This is nearly correct for infants consuming formula prepared with water. However, studies of the routes of lead exposure in older children and adults have found that inhalation of air-borne lead, ingestion of lead in food and ingestion of lead in dust and other non-food substances (paint chips, etc.) are significant sources of exposure. Table 10 summarizes the average baseline exposure from these sources. This baseline exposure is considered unavoidable without a generalized reduction of lead content in air and canned foods. From Table 10, it can be seen that the amount of lead ingested in water under baseline conditions is about 15% of the total intake in children and about 31% of the total in adults. Table 11 lists the sources and amounts of additional lead exposure which may occur in some cases. Most of these additional exposures derive from air or dust; few are from food or water. Several studies have found that, except in unusual cases, an average American consumes about 100 μg of lead per day, of which about 80 to 90 μg is from food, air and dust. This intake exceeds both the ADI in children (19 μg/day) and the ADI in adults (48 μg/day). In this situation, the ADI from water may be taken to be 15%

TABLE 10
Baseline Lead Exposure in Humans

	2-year-old child		Adult male	
Source	**μg/day**	**% Total**	**μg/day**	**% Total**
Air	0.5	1	1.0	2
Food	18.9	40	35.8	59
Dust	21.0	44	4.5	7
Water	6.9	15	18.9	31
Total	47.3	100	60.2	100

Adapted from USEPA (1984).

TABLE 11
Summary of Additional Human Exposures to Lead

Source	Total Lead consumed (μg/day)	
	Child	Adult
Baseline exposure (Air, food, water, dust)	47	60
Additional exposure due to:		
Urban atmosphere	91	28
Family gardens	48	120
Lead paints	110	17
Residence near smelter	2,200	250
Smoking	—	30
Wine consumption	—	100
Occupational	—	1,100
Total	47-2,496	60-1,705

Adapted from USEPA (1984).

of the total ADI, which is the value for baseline exposure through water in children (see Table 10). The AADI value is then calculated as follows:

AADI:

$$\frac{(0.15)(19\ \mu g/day)}{(1\ L/day)} = 3\ \mu g/L$$

where:

19 μg/day = ADI which protects both children and adults.
1 L/day = assumed water consumption by a 10-kg child.
0.15 = assumed fraction of total ADI which may be contributed by water.

CARCINOGENIC EFFECTS

Fifteen studies of cytogenetic abnormalities in persons exposed to lead have been summarized (IARC, 1980). Positive results were reported in nine of these studies, whereas six reported negative results. For example, O'Riordan and Evans (1974) reported no significant chromosomal damage in male workers with PbB values from 40 to 120 μg/dL. However, Forni et al. (1976) found the incidence of abnormal metaphases doubled ($P < 0.05$) in workers exposed to air lead levels of more than 0.8 mg/m^3 for one month; the incidence increased further for two months and remained elevated for up to seven months. Blood lead levels increased from an initial mean of 34 μg/dL to 45 μg/dL in the first month, although this change was not statistically significant. Erythrocyte ALAD activity was reduced almost 50% during the first month

TABLE 12
Summary of Studies on the Incidence of Tumors in Experimental Animals Exposed to Lead Compounds

Species	Pb Compound	Dose and mode[a]	Incidence (and type) of neoplasms	Reference
Rat	Pb phosphate	120–680 mg (total dose s.c.)	19/29 (renal tumors)	Zollinger (1953)
Rat	Pb acetate	1% (in diet)	15/16 (kidney tumors) 14/16 (renal carcinomas)	Boyland et al. (1962)
Rat	Pb subacetate	0.1% and 1.0% (in diet)	11/32 (renal tumors) 13/24 (renal tumors)	Van Esch et al. (1962)
Rat	Pb phosphate	1.3 g (total dosage s.c.)	29/80 (renal tumors)	Balo et al. (1965)
Rat	Pb subacetate	0.5 – 1% (in diet)	14/24 (renal tumors)	Hass et al. (1967)
Rat	Pb subacetate	1% (in diet)	31/40 (renal tumors)	Mao and Molnar (1967)
Rat	Pb acetate	3 mg/day for 2 months; 4 mg/day for 16 months (p.o.)	72/126 (renal tumors) 23/94 males (testicular [Leydig cell] tumors)	Zawirska and Medraś (1968)
Hamster	Pb subacetate	1.0% (in 0.5% diet)	No significant incidence of renal neoplasms	Van Esch and Kroes (1969)
Mouse	Pb subacetate	0.1% and 1.0% (in diet)	7/25 (renal carcinomas) at 0.1%; substantial death at 1.0%	Van Esch and Kroes (1969)
Rat	Pb nitrate	25 g/L in drinking water	No significant incidence of tumors	Schroeder et al. (1970)
Rat	Pb acetate	3 mg/day (p.o.)	89/94 (renal, pituitary, cerebral gliomas, adrenal, thyroid, prostatic, mammary tumors)	Zawirska and Medraś, 1972
Rat	Pb acetate	0, 10, 50, 100, 1,000, 2,000 ppm (in diet) for 2 yr	No tumors 0–100 ppm; 5/50 (renal tumors) at 500 ppm; 10/20 at 1,000 ppm; 16/20 males, 7/20 females at 2,000 ppm	Azar et al. (1973)
Rat	Pb chromate	10 mg (p.o.) 2 times each month	5/47 (1 lymphoma, 4 leukemias)	Furst et al. (1976)
		10 mg/monthly for 9 months; then 3 monthly injections of 5 mg	1/50 (fibrosarcoma)	

Adapted from USEPA (1984).

TABLE 13
Mortality and Kidney Tumors in Rats Fed Lead Acetate for Two Years

Pb in diet (ppm)	Average daily dose (mg/kg/day)[a]	No. of rats of each sex	% Mortality		% Kidney tumors	
			Male	Female	Male	Female
5	0.3	100	37	34	0	0
18	0.9	50	36	30	0	0
62	3.1	50	36	28	0	0
141	7.1	50	36	28	0	0
548	27.4	50	52	36	10	0
3	0.2	20	50	35	0	0
1,130	56.5	20	50	50	50	0
2,102	105.1	20	80	35	80	35

[a]Assuming consumption of 50 g food/kg body weight.
[b]Includes rats that either died or were sacrificed *in extremis*.
Adapted from Azar et al. (1973).

of exposure ($P < 0.05$), and decreased further in subsequent months. The reason for these conflicting findings is not clear. Deknudt et al. (1977) suggested that ancillary factors (e.g., the level of calcium intake) may be critical.

A number of studies of the carcinogenic potential of various lead salts in animals are summarized in Table 12. The most common observation was increased frequency of renal tumors, although evidence of tumors in other tissues has been noted. The doses of salt producing these effects were quite high, generally 0.1% to 1% in the diet (equivalent to about 50 to 500 mg Pb/kg/day, assuming consumption of 50 g food/kg body weight).

The study most useful in establishing a quantitative relationship between lead ingestion and frequency of renal tumors is the report by Azar et al. (1973). The data from this report are summarized in Table 13. A dose-dependent increase in renal tumor frequency in male rats was observed over the range of 500 to 2,000 ppm Pb (as lead acetate) in the diet. It should be noted that these doses are associated with moderate to severe non-carcinogenic effects in rats.

Several epidemiological studies of industrial workers, for whom the potential for lead exposure is typically greater than for a "normal population," have been conducted to evaluate the role of lead in the induction of human neoplasia (Cooper, 1976; Cooper, 1981; Cooper and Gaffey, 1975; Chrusciel, 1975; Dingwall-Fordyce and Lane, 1963; Lane, 1964; McMichael and Johnson, 1982; Neal et al., 1941; Nelson et al., 1982). In general, these studies made no attempt to consider the types of lead compounds to which workers were exposed, or to determine probable routes of exposure. While a number of these studies found an association between lead exposure and the frequency of various cancer types, no study was sufficiently free of confounding factors to permit a clear conclusion.

The International Agency for Research on Cancer (IARC) has performed an assessment of the degree of evidence for the carcinogenicity of lead and lead compounds in humans and experimental animals (WHO, 1982). This assessment concluded that lead and lead compounds are Group 3 compounds (sufficient evidence for carcinogenicity of some lead salts in animals, but inadequate evidence for carcinogenicity in humans).

QUANTIFICATION OF CARCINOGENIC RISK

Although some lead salts are clearly associated with increased renal tumor frequency in animals, no quantitative estimate of excess cancer risk was located. The data in Table 12 indicate that relatively high lead intake levels are required to produce increased tumor rates, and it seems probable that the non-carcinogenic effects of lead occur at lower levels.

EXISTING GUIDELINES AND STANDARDS

The current MCL for lead in drinking water is 50 μg/L (USEPA, 1976). This is based on the recognition that although water is not normally a major route of lead exposure, it nevertheless "would seem wise at this time to continue to limit the lead in water as low a level as practical" (USEPA, 1976). The WHO European standard for lead is 100 μg/L, as is the USSR standard.

The National Academy of Sciences (NAS, 1977) concluded that "no-observed-adverse-health-effect level cannot be set with assurance at any value greater than 0.025 mg/L" (25 μg/L), noting that: "the present limit of 50 μg/L may not, in view of other sources of environmental exposure, provide a sufficient margin of safety, particularly for fetuses and young growing children. Although further studies will be necessary to arrive at a reasonable limit, it is suggested that the limit be lowered."

SPECIAL CONSIDERATIONS

High-Risk Subpopulations. Numerous studies based on neurobehavioral, morphological or biochemical endpoints all suggest that the order of human susceptibility to the neurotoxic effects of lead is: young > adult; female > male. Animal studies also have pointed to the perinatal period of ontogeny as a time of particular sensitivity to lead. The precise boundaries of a critical period for lead exposure are not yet clear, and may vary depending on the species and function or endpoint that is being assessed. Nevertheless, there is general agreement that human infants and toddlers below the age of three years are at special risk because of increased opportunity for exposure through pica and increased rates of lead absorption.

In addition, women of child-bearing age are considered a high risk subpopulation, since the ratio of fetal/maternal PbB values is near 1:1. For these reasons, the AADI is set to protect all groups, including the fetus *in utero,* infants and children.

TABLE 14
Summary of Quantification of Toxicological Effects

	Drinking water concentration (μg/L)	
	10-kg child	**70-kg adult**
One-Day Health Advisory	25[a]	140[b]
Ten-Day Health Advisory	25[a]	140[b]
AADI (100% from water)	19	___[c]
AADI (15% from water)	3	___[c]

[a]Based on studies by Cory-Slechta and Thompson (1979).
[b]Based on studies by Govoni et al. (1979, 1980), Memo et al. (1980a, 1981) and Grant et al. (1980).
[c]Calculated value is higher than for the child, so the lower value (for the child) is adopted in order to protect both groups.

Interactions. A number of compounds are known to influence the toxicity of orally-ingested lead. In most cases, this is because the absorption of lead from the diet is affected by the compounds. For example, studies in animals and humans have shown that low dietary levels of calcium, phosphate, copper or zinc all tend to cause increased gastrointestinal absorption and retention of lead. Increased levels of vitamin D also cause increased lead absorption. Increased ingestion of dietary lipids (especially polyunsaturates) causes increased absorption, as do certain milk components (particularly lactose). The relationship between dietary protein levels and lead absorption is not clear, since both decreased and increased protein intake cause increased lead absorption. The dietary deficiencies which cause increased lead absorption are most prevalent in children, and especially in poor children.

Beneficial Effects. No beneficial effects of lead in mammals are recognized.

Other Factors. A large fraction of the lead in the environment which poses an exposure risk to humans is anthropogenic in origin. Of particular concern is lead in air, which is inhaled directly, or which settles and is subsequently ingested as dust. Another major concern is lead which enters food from soldered containers. While lead ingestion through water may be important in some cases, it normally does not represent the principal route of human exposure.

SUMMARY

The recommended HA and AADI values for lead are summarized in Table 14.

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ENVIRONMENTAL POLLUTION AND CRIME

Roger D. Masters*

Before discussing the role of environmental pollution in the complex interactions of "Biology, Behavior, and the Criminal Law," it is useful to identify three common misperceptions concerning discussions of law and biology. First, "biology" is *not* identical with genetics. The life sciences study the development and evolution of organisms in their environments; hence, as the research reported below illustrates, this field often provides unique insights into the *environmental* factors in human behavior. Second, "biological" characteristics are not immutable. The life sciences are not deterministic, and as modern medicine proves daily, discovering the complex relationships between inherited, developmental, and environmental factors often greatly *increases* human control over outcomes. Finally, biological approaches to human behavior need not entail the abandonment of legal and moral responsibility, especially for criminal actions. As neuroscientists have shown, what one thinks can influence the function and even neurochemistry of the brain.[1]

It follows, as James Q. Wilson has eloquently argued in his recent Godkin Lectures at Harvard University, that judgment of a criminal's behavior in a court of law is and should remain sharply different from scientific explanations of that behavior.[2] Knowledge of the factors influencing behavior gives humans increased ability to control outcomes. Because life-styles and behavioral choices are essential factors in vulnerability to environmental toxins like those discussed in the following analysis, this approach effectively *enhances* the legal and moral responsibility of the individual.[3] In legal terms,

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1. *See generally* PAUL M. CHURCHLAND, THE ENGINE OF REASON, THE SEAT OF THE SOUL (1995) (stating that the central nervous system is a recursive, massively parallel information processing system in which feedforward as well as feedback neural networks constantly reconstruct sensory input and memory; as a result, moral and law abiding behavior needs to be seen as a *skill*, not as the deduction of a specific case from a general rule).

2. *See* JAMES Q. WILSON, MORAL JUDGMENT 7 (1997) (asserting that the tendency to allow social scientific explanations of behavior to generate exculpatory evidence is undermining our criminal law, with serious implications for the public support of our legal system).

3. *See* Roger D. Masters, *Conclusions for Public Policy*, *in* THE NEUROTRANSMITTER REVOLUTION: SEROTONIN, SOCIAL BEHAVIOR AND THE LAW 227-42 (Roger D. Masters & Michael T. McQuire eds., 1993) (surveying the role of serotonin and other neurotransmitters in modulating human social behavior; and arguing that these biological explanations need not, and should not, have an

nothing in the biological study of human behavior justifies the abandonment of the traditional concept of mens rea.

I. THE BIOLOGY OF VIOLENCE

Although sociologists and criminologists often debate the origins and character of crime, from a biological perspective individual acts of violent behavior among humans are abnormal (in the sense of rare). Among other primates, aggressive or threatening behavior is statistically infrequent. For example, in twenty-five years of observing chimpanzees, Jane Goodall noted overtly aggressive acts in only 1.6% of observation hours among males and a mere 0.6% for females; more importantly, chimpanzee threat displays usually end without physical violence.[4] For other primates as well, while threats are part of the normal primate behavioral repertoire, they are usually inhibited and do not result in wounds or physical harm.[5]

It should therefore not be surprising that humans typically respond to a broad diversity of social situations, including instances when others show signs of threat or anger, without engaging in violent behavior. Although criminal violence is abnormal in this purely statistical sense, feelings of anger and hostility are part of our innate emotional and behavioral repertoire. Thus, from a biological perspective, violent crime appears to reflect the failure of normal systems of inhibition.[6]

The strongest evidence for this view comes from contemporary neuroscience. As David Goldman explained earlier in this symposium, mammalian social behavior is modulated in part by a complex system of neurotransmitters which can regulate the response strength of excitatory or inhibitory circuits in the brain. Serotonin, one of the best known of these regulatory chemicals—due to the popularity of Prozac, which increases serotonin's functional activity—has frequently been shown to play an inhibitory role in humans, as well as other mammals. Consistent with the view that violence often requires a failure of normal inhibitory mechanisms, deficits in serotonin have been associated with more frequent violent behavior not only in humans, but in nonhuman primates.

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exculpatory effect in criminal law; on the contrary, knowledge of these explanations should make possible enhanced responsibility for violations of social norms and laws) [hereinafter THE NEUROTRANSMITTER REVOLUTION].

4. *See* JANE GOODALL, THE CHIMPANZEES OF GOMBE 314 (1986) (surveying 25 years of observation of chimpanzee social behavior at Gombe).

5. *See generally* FRANS DE WAAL, GOOD NATURED (1996) (discussing the role of social cooperation as well as competition among chimpanzees and other primates).

6. *See generally* KONRAD LORENZ, ON AGGRESSION (1966) (emphasizing the natural origins of threat behavior and the essential role of inhibition in animal and human social behavior).

The mechanisms associated with this process are the result of hominid evolution as a group-living primate, interacting with others in face-to-face groups with normally less than two hundred members. Like other primates, we react instantaneously to facial displays and other nonverbal cues of the emotions and likely behavior of others. Among these displays are cues of anger, threat, and possible attack which humans from virtually every known culture distinguish accurately.

We perceive anger, and instantaneously respond emotionally to it; but then monitor our own feelings in the light of social norms and cultural expectations. If a parent sees a child about to hit another child, for example, it is not difficult to recognize the threat and intervene before the actual hitting takes place. These reactions are part of a broader system of emotional expression and social display behavior that is universal and innate to our species, but shaped and modified in each culture.[7]

For over fifteen years, I have been studying the role of this system of nonverbal displays in voter responses to political leaders, which provides an excellent example of "normal" or routine human social behavior toward a known person. Perceptions of the behavior of others are in fact filtered through many individual traits, including personality (some individuals are fearful and highly sensitive to threat, others relatively insensitive), attitudes toward the individual observed, and cultural expectations. Whatever these perceptions, experiments confirm that feelings elicited by the behavior of others are organized into two neuroanatomically and functionally distinct systems, usually called positive (hedonic) and negative (agonic or competitive). As a result, the experience of seeing another person's facial displays of emotion unconsciously activates the facial muscles underlying our own displays of emotion and triggers congruent feelings of positive and negative emotion.[8]

Because the neuroanatomical system underlying these responses is innately wired into the brain, we constantly monitor the intentions of others and respond emotionally and cognitively to them. In this process, both the positive (cooperative) and negative (competitive) circuits function simultaneously, so that our conscious responses reflect the net outcome of diverse cues and feelings. Contrary to the views of theorists in the tradition

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7. *See generally* ROGER D. MASTERS, THE NATURE OF POLITICS (1989) (noting the expressive and social functions of human facial displays and their relation to both innate or natural and cultural or cognitive elements); A. Michael Warnecke et al., *The Roots of Nationalism: Nonverbal Behavior and Xenophobia*, 13 ETHOLOGY & SOCIOBIOLOGY 267-82 (1992) (presenting experimental data of differences in responses to nonverbal cues of foreigners).

8. *See generally* ROGER D. MASTERS, BEYOND RELATIVISM (1993) (explaining that nonverbal displays of emotion can be understood as "innate ideas," communicating information about the social behavior and feelings of others that plays a necessary and ubiquitous role in normal social life).

of Rousseau, therefore, aggressive or competitive emotional responses are as natural to humans as positive or bonding responses.

Human morality and obedience to law are based in part on this complex system of emotions.[9] Positive emotional feelings are associated not only with physical pleasures but with events and ideas that confirm firmly held beliefs in expected social behavior. When one sees people doing proper things, it makes one feel good. When one sees them violating the rules, it makes one angry. While these emotional responses are triggered when our own interests are threatened, normally they are also aroused when, as bystanders, we see innocent people being harmed and social rules flouted.

These findings have a direct relevance to understanding human crime. It is not the case, as some have thought, that violence occurs because the criminal has imagined killing someone, whereas normal human beings never have such thoughts. Recent studies show that in a given year, as many as 70% of males and a majority of females have imagined in detail killing another person.[10] While cultural and social circumstances can increase or decrease the extent of imagined aggressiveness toward those who act unfairly toward us or thwart our desires, we all have feelings of aggression that we normally inhibit. And although the fear of punishment is involved in the capacity to inhibit aggressive impulses, so too is the anticipated reward of conforming to legitimate and expected social norms. Humans are, by nature, social animals.

From this perspective, violent offenders seem to have some systematic deficit in the way the central nervous system processes the displays of social and emotional behavior of others. Although the failure to inhibit anger and threatening feelings may be a common factor in violent crime, individual offenders almost certainly differ in the mechanisms that weaken this capacity to inhibit aggressive behavior. For a small proportion of the population, known as "primary psychopaths" (and often associated with incorrigible repeat offenders), the basic link between the perception or expression of social displays and emotions does not function normally. In these individuals, symbolized by Kevin Bacon's violent offender character in the film *The River Wild*,[11] feelings of bonding and sympathy are rarely, if ever, aroused, and nonverbal behavior is used, under cognitive control, for "cold-blooded" selfishness.[12] Most criminals, however, probably are capable of normal

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9. *See generally* MARGARET GRUTER, LAW AND THE MIND (1993) (asserting that understanding the legal process requires an understanding of the evolutionary analysis of the way humans behave, including emotional and nonverbal social factors long ignored by many legal theorists).

10. *See* Douglas T. Kenrick & Virgil Sheets, *Homicidal Fantasies*, 14 ETHOLOGY & SOCIOBIOLOGY 231 (1993).

11. THE RIVER WILD (Universal 1994).

12. *See generally* ROBERT HARE, WITHOUT CONSCIENCE (1993) (noting that the roots of

emotional responses, but have deficits in the positive emotional responses to bonding and normal law-abiding behavior, or in the inhibitory circuits that prevent angry emotions from erupting into physical violence, or both.

Recently, this view has been confirmed by an experimental study of Vermont prison inmates conducted by a former student of mine, Baldwin Way, now in the Department of Neuroscience at UCLA.[13] Preliminary data from this study provides evidence that violent offenders differ from non-violent criminals as well as law-abiding citizens used as controls, in the responses to the facial displays and nonverbal cues underlying normal social behavior.

In these experiments, viewers were asked to describe the facial display behavior of known leaders in a video excerpt shown without the sound, using separate rating scales for how happy, comforting, angry, and fearful the leader was. Over the previous fifteen years of experiments, we have found that these ratings provide an accurate assessment of the facial displays of others (as is confirmed by scientific ratings and cross-cultural studies of the same behavior). When the research was extended to prison inmates, we found no statistically significant differences between the "net description" by violent offenders, nonviolent prison inmates, and controls who had been subjects in similar experiments in a university setting.[14] When seeing a neutral image of a leader, people who commit violent acts do not seem to *see* more or less aggressiveness than normal people.

In over fifteen years of experiments, such descriptive scores after watching a leader have been strongly correlated with viewers' emotional responses during the excerpt, measured as the "net warmth" of positive emotions (happiness and comfort) minus the negative feelings (anger and fear). However, when the violent offenders looked at videotapes of the President's neutral displays, they did not report feeling the level of positive emotion that would be predicted from their perceptions.[15] At this level, there was a statistically significant difference between violent offenders, and either nonviolent prison inmates or law-abiding controls.

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behavioral characteristics of "primary psychopaths" lie in abnormal brain processing of emotion).

13. We thank John Gorzyck, Commissioner of Corrections, and John Perry, Director of Research for the Department of Corrections of the State of Vermont, for their assistance in making possible this study, which was funded by the Rockefeller Center for the Social Sciences at Dartmouth College.

14. *See infra* Figure 1. The "net description" is the overall rating of positive cues of happiness and comfort, minus negative cues of anger and fear. For the methods in this experiment, see Baldwin M. Way & Roger D. Masters, *Political Attitudes: Interactions of Cognition and Affect*, 10 MOTIVATION & EMOTION 219-36 (1996) (examining the effects of subliminal cues of emotion on the perception, emotional response, and attitudes to President Clinton). For the prison study, we present data for all experimental conditions combined.

15. *See infra* Figure 2.

Violent criminals do not seem to experience the normal emotions that lead to bonding with significant social others. Because this lack of positive feeling toward an authority figure was not observed among the nonviolent offenders in our prison sample, we may have discovered a common trait associated with the weakness of inhibiting aggressive behavior that I have suggested is a proximate mechanism in violent crime.

There are doubtless many possible reasons for an individual to exhibit abnormal emotional responses, poor social bonding, and weak inhibition of aggressive impulses. Developmental experiences, brain damage due to child abuse or disease, and socioeconomic deprivation are among the many "risk factors" that might combine to prevent the typical emotional responses to others. Our experimental data shows, however, that when the same violent offenders see an aggressive excerpt of the President, their positive emotional responses are congruent with the level of their descriptions of the excerpt. Hence the failure to bond to a neutral image of the national leader does not seem to be due to the total absence of emotional responsiveness. The neurons of violent offenders seem to be connected, but there is something different about the functional way that their brains operate.

Such findings are consistent with the view that there may be a "final common pathway" of abnormal social disinhibition that characterizes the diverse etiologies leading to criminal violence. Whatever the initial reasons for becoming a violent criminal, these risk factors must impinge on one of the many functional processes by which the human brain perceives and responds to social situations.

II. ENVIRONMENTAL FACTORS IN CRIMINAL VIOLENCE

According to conventional approaches, criminal behavior is a function of the conscious thoughts and intentions of the criminal in response to the social environment. Critics often jump to the conclusion that a biological perspective implies a reduction of these conscious choices to some form of molecular determinism within the brain. This need not follow, however. To the contrary, the complex causal pathways merely presume that the effects of either individual choices or social environments must impinge on the central nervous system if they are to explain the eruption of violent behavior.

It is easy to give a concrete illustration of the problem of analyzing the environmental factors in criminal violence. According to one widely shared view, television violence is one of the main explanations for the high rates of violent crime in the United States. While exposure to television violence might be a risk factor for crime, the usual argument meets two powerful objections. First, millions of Americans see violence nightly on television:

why do only some individuals commit such acts?[16] Second, rates of violent crime vary sharply from one place in the United States to another.[17] Since television violence is largely a constant, if it causes crime, why do individuals and environments differ so greatly in response to it?

Much the same challenge can be addressed to many other conventional explanations for crime. For example, poverty and unemployment are often said to be major reasons for criminal behavior. Yet not all poor or unemployed people are criminals, and some relatively poor counties and regions have remarkably low crime rates. When sociologists insist on a radical dichotomy between "nature" and "nurture," they often attribute behavior to environmental factors that are too broadly defined to account for the actual phenomena observed.

Paradoxically, however, these same risk factors may indeed play an important role for individuals with abnormalities in brain function that undermine normal inhibitory mechanisms. In particular, biochemical imbalances may be associated with many of the factors that have been widely associated with criminal violence, permitting us to see how *combinations* of these risk factors can trigger violence even when the same individual or social phenomena do not lead to crime.

For example, a very large proportion of violent offenders were under the influence of alcohol at the time of their criminal behavior. Although the disinhibitory effects of alcohol are well known to anyone who has relaxed during a cocktail hour, most of those who consume alcoholic beverages are not violent. Low levels of serotonin have also been associated with weakened inhibition, but the effects of this chemical condition range from psychological depression or obsessive-compulsive disorder, to an increased vulnerability to accident and suicide, as well as to increased frequencies of aggressive behavior.[18] As Markku Linnoila and his colleagues at the National Institute of Alcoholism and Alcohol Abuse have shown, however, the *combination* of alcoholism, low serotoninergic function, and a third biochemical condition, low glucose uptake, are highly predictive of impulsive violence or arson.[19]

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16. In 1991, the average rate for all violent crimes—homicide, aggravated assault, sexual assault, and armed robbery—in a county in the United States was 320 per 100,000 persons. Search of USA COUNTIES ON CD-ROM, based on STATE AND METROPOLITAN AREA DATA BOOK, 1982, 1986, & 1991 (Washington D.C.: U.S. Census Bureau, 1994).

17. In some counties, the violent crime rate is under 200 per 100,000 persons, whereas in urban centers like Baltimore the rate can be over ten times higher. *See id.*

18. *See generally* PETER KRAMER, LISTENING TO PROZAC (1993) (describing clinical evidence of the effects—sometimes remarkable—of psychoactive drugs like Prozac in treating a wide variety of psychiatric symptoms and personal problems). *See also* THE NEUROTRANSMITTER REVOLUTION, *supra* note 3.

19. *See* Markku Linnoila et al., *Serotonin and Violent Behavior, in* THE NEUROTRANSMITTER REVOLUTION, *supra* note 3, at 61 (explaining that biochemical and behavioral markers provide highly

Other factors usually traced to the social or cultural environment also have their effects only insofar as they are mediated through changes in normal brain function. For example, social stress is an independent factor in regulating neurotransmitters. Individuals under prolonged social stress reduce the production of a number of neurotransmitters including serotonin and dopamine. Their immune systems also function less effectively, increasing the frequency of disease. While many non-scientists think of social stress as an environmental factor, its effects are also felt at the level of brain biochemistry and neuronal function.

Indeed, this probably explains why hard drug use spans diverse social groups—including not only ghetto youths in disintegrating socio-cultural environments, but also high status, highly-stressed professionals in the business and entertainment worlds. Cocaine, for example, is a nonselective dopamine re-uptake inhibitor. Like those who take Prozac (a selective serotonin re-uptake inhibitor), therefore, illicit drug users may be attempting to normalize brain chemistry imbalanced for diverse reasons often including social stress.

Similarly, social status changes brain chemistry. When a low status monkey (or human) gains dominance, the perception of submissive behavior in others leads to an increase in serotonin levels. When a teacher stands in front of a class, or a judge enters a courtroom, the perception of subordination has the same effect as taking Prozac. Because uncertain social status or isolation lowers serotoninergic levels, it is not hard to see how gang membership can entail rewards at the biochemical as well as the conscious and behavioral levels.[20]

Among the many factors that can impinge on the neurochemical or functional efficacy of the human brain, many arise from the combined effects of dietary insufficiencies, substance abuse, and environmental pollution. At the individual level, violent offenders are more likely to have absorbed toxic metals like lead or cadmium and manganese in their blood and brain. In seven of seven studies of toxic elements in the head hair of prison inmates, and as Professor Deborah Denno[21] explained in this symposium, the absorption of lead in blood, and the presence of anemia (which facilitates the uptake of lead in the brain) are two of the most accurate predictors of the likelihood of seven-

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accurate *prediction* for recidivism for impulsive violence—violent crimes in which the name of the victim is unknown to the attacker—or arson; testing serotonin levels in blood and glucose uptake permits a discriminant analysis in which there are less than 10% of either false positives or false negatives when predicting, at sentencing for a first offense, a repeat crime after release from prison).

20. *See* Michael Raleigh & Michael T. McGuire, *Serotonin, Aggression, and Violence in Vervet Monkeys*, *in* THE NEUROTRANSMITTER REVOLUTION, *supra* note 3, at 129.

21. *See* Deborah W. Denno, *Gender Differences in Biological and Sociological Predictors of Crimes*, 22 VT. L. REV. 305 (1997).

year-old inner city residents becoming juvenile delinquents or adult violent criminals.

Could it be that environmental pollution is among the environmental risk factors for violent crime? This question, which makes sense when criminal violence is analyzed from a biological perspective, is rarely addressed by traditional criminologists. The hypothesis explored below shows, furthermore, that far from oversimplifying the causes of crime, the search for intervening biological mechanisms suggests additional environmental risk factors associated with violence that have unfortunately been ignored by criminologists. Among these, I will argue, absorption of neurotoxic metals is perhaps the most important missing link in our theories of violence.

III. NEUROTOXICITY AND VIOLENCE

Some heavy metals, such as lead, cadmium, manganese, and mercury, are highly neurotoxic. If these elements cross the blood-brain barrier and are absorbed in neurons, crucial changes in brain structure and function result. In particular, toxins like lead and manganese reduce the functional level of essential neurotransmitters, including serotonin, dopamine, acetylcholine, and gamma aminobutyric acid (GABA). Normally, humans excrete these toxins without absorbing them in the brain. When vital elements such as calcium, iron, or zinc are missing from the diet, however, the neurotoxic metals—which often have a similar electric charge—are absorbed in their place. Exposure to toxic elements, poor diet, and substance abuse therefore have interacting effects on the brain function and social behavior.

> Changed brain chemistry can alter behaviour, and changed behaviour can alter brain chemistry: the interaction is two way. It therefore follows that behaviour, cognition, social interactions, and other expressions of brain function are subject not only to the social environment but also to certain aspects of the chemical environment. The relevant chemical factors include (a) neurotoxic pollutants in general, of which lead is evidently now the most serious in its impact, (b) certain common nutrient deficiencies, particularly of zinc, and (c) neurotoxins of voluntary abuse, of which ethanol is still probably producing the most widespread social damage.[22]

22. Derek Bryce-Smith, *Environmental Chemical Influences on Behaviour and Mentation*, 15 CHEM. SOC. REV. 122 (1986) (citations omitted).

This approach is especially relevant to violent crime because neurotoxic elements can alter neurotransmitter function by disinhibiting aggressive impulses under stress. In addition to lead, manganese is a neurotoxic metal that has been associated with violence. Among the dietary deficiencies, increasing neuronal uptake of toxic metals are deficits in calcium and other essential vitamins, as well as zinc. Since there are geographic variations in the incidence of pollution, alcoholism, and populations with dietary deficiencies, the neurotoxicity hypothesis can be used to predict differences in rates of violence, using county-level data that are likely to reflect the influence of environmental pollution. Data testing this prediction shows that exposures to lead and manganese, especially when combined with alcoholism, are highly correlated with increased frequency of violent crime.

A. Lead Toxicity

Behavioral and cognitive deficits caused by lead, noted in antiquity by Hippocrates, have been the subject of widespread scientific analysis.[23] Although legislation prohibiting lead in gasoline additives and paint reduced lead levels in blood by seventy-eight percent between 1976-1978 and 1988-1991,[24] toxic release of lead and lead compounds remains a serious problem due to aging water systems and industrial pollution. Exposure to subclinical doses of this neurotoxic metal can be a major hazard for three principal reasons: (1) children absorb up to fifty percent of the lead they ingest, compared to eight percent for adults; (2) prolonged exposure to even very low doses of lead can cause neuronal damage during early development, resulting in lasting cognitive and behavioral deficits; and (3) the highest levels of lead uptake are reported for the population groups most likely to commit violent crimes. Because the consequences of exposure depend on lifestyle factors such as alcoholic consumption, smoking, and diet, moreover, averages for a population will tend to mask the risks arising from combinations of co-factors.

Prolonged exposure to low levels of lead can influence dopaminergic, cholinergic, and glutamatergic neurotransmitter functioning, producing learning deficits that include impairment of passive avoidance learning—the capacity to be deterred by future punishment. Attention Deficit Disorder (ADD) and Attention Deficit Hyperactivity Disorder (ADHD), conditions

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23. *See generally* HUMAN LEAD EXPOSURE (Herbert L. Needleman ed., 1989) (offering an excellent survey of the nature and effects of lead toxicity). For more extensive documentation, see ROGER D. MASTERS ET AL., *Environmental Pollution, Neurotoxicity, and Criminal Violence*, in ASPECTS OF ENVIRONMENTAL TOXICOLOGY 13-48 (J. Rose ed., forthcoming 1998) (reviewing evidence for the neurotoxicology hypothesis and supporting data).

24. *See* J. Pirkle et al., *The Decline in Blood Lead Levels in the United States*, 272 J. AMER. MED. ASSN. 284 (1993).

frequently associated with juvenile delinquency, have also been traced to prolonged exposure to lead. Analyses of the head hair of violent offenders often show significantly higher levels of lead, sometimes combined with higher levels of another toxic metal such as cadmium, when compared to nonviolent controls. That subclinical plumbism has behavioral effects is, moreover, confirmed by measures of educational success, such as the correlation between lead levels in tooth enamel and lowered IQ scores.[25]

B. Manganese Toxicity

Manganese has also been associated with subclinical behavioral disturbances due to its effects on neurotransmitter function.[26] Although industrial manganism can be traced to direct exposure to large quantities of manganese dioxide or manganese nitrate, chronic exposure to low levels of manganese is probably more relevant to loss of impulse control and outbursts of violent behavior, especially under stress.

Exposure to manganese lowers levels of serotonin in the brain, while paradoxically increasing serotonin levels in blood and body tissue; manganese also degrades dopamine and reduces levels of essential minerals in brain cells. While the mechanism of these effects is not clearly understood, some of these effects may be due to altered levels of monoamine oxidase (MAOA). However produced, low levels of serotonin in the brain are associated with mood disturbances, poor impulse control, and increases in aggressive behavior—effects that have increasingly been treated with Prozac and other psychotrophic medications that enhance serotoninergic function.

Vitamin and mineral deficiencies play a central role in manganese uptake. In laboratory studies, where environmental exposure alone does not lead to toxicity, animals with deficits in calcium intake show significant manganese retention. Other aspects of diet also influence the absorption of manganese. For example, laboratory animals raised on infant formula have cellular retention of manganese that is five times greater than controls raised on human mother's milk. The combination of calcium insufficiency and manganese toxicity could therefore be described to the general public as "reverse Prozac."

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25. *See* Herbert L. Needleman et al., *The Long-Term Effects of Exposure to Low Doses of Lead in Childhood*, 322 NEW ENG. J. MED. 83, 83-88 (1990).

26. *See* John Donaldson, *The Physiopathologic Significance of Manganese in Brain: Its Relation to Schizophrenia and Neurodegenerative Disorders*, 8 NEUROTOXICOLOGY 451, 456-62 (1987) (listing technical evidence of neurotoxic effects of manganese). For fuller documentation, see Way & Masters, *supra* note 14.

C. Brain Biochemistry and Synergy

Other elements have effects on brain biochemistry that can either increase or reduce probabilities of violent behavior. Lead and cadmium apparently interact, with significantly higher levels of cadmium found in samples of those violent offenders with higher lead in head hair analyses. Further, relatively low levels of zinc have been associated with increased lead uptake. Conversely, lithium reduces the levels of manganese and other toxic metals in the brain, and is lower in hair samples of violent offenders than in hair samples from controls. At the population level, homicide rates in Texas were inversely related to levels of lithium in the water supply. Other studies of violent or violence-prone individuals have shown abnormal levels or ratios of a variety of elements or toxic metals.[27]

Ethanol in alcoholic beverages may be the most important substance that interacts with toxic metals. In laboratory animals, the effects of a toxic element (cadmium) and of ethanol were "significantly altered," especially because "animals exposed to alcohol alone have a significantly greater accumulation of cadmium in liver and intestine and a tendency for greater accumulation in other organs as compared to their normal counterparts. . . . This . . . is likely due to the increased permeability of membrane in the presence of ethanol."[28] In humans, ethanol interacts with the physiological effects of lead on blood pressure and other physiological processes. Alcohol reduces behavioral inhibition by enhancing the effects of other toxins that reduce the levels of functional activity of inhibitory neurotransmitters (as manganese does for serotonin) or damage inhibitory neuronal structures (as lead does for interneurons). Not surprisingly, consumption of alcohol is associated with a very high proportion of violent crimes.[29]

While suggestive, these studies do not provide convincing evidence that the synergy between manganese, lead, and other substances contributes to differential rates of criminal behavior. The data implicating neurotoxicity at the individual level can, however, be verified by contrasting rates of violent crime in geographical areas with and without releases of toxic metals as well as with lower or higher rates of alcoholism. To be sure, there are many other possible risk factors explaining violence. Moreover, Environmental

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27. *See generally* Gerhard N. Schrauzer et al., *Lithium in Scalp Hair of Adults, Students, and Violent Criminals*, 34 BIOLOGICAL TRACE ELEMENT RES. 161-76 (1992) (noting that individuals with higher lithium levels have lower levels of criminal behavior).

28. Geeta Sharma et al., *Effect of Ethanol on Cadmium Uptake and Metabolism of Zinc and Copper in Rats Exposed to Cadmium*, 121 J. NUTRITION 87, 90 (1991).

29. For a review of the literature, see generally Roger D. Masters et al., *Brain Biochemistry and Social Status: The Neurotoxicity Hypothesis*, *in* INTELLIGENCE, POLITICAL INEQUALITY, AND PUBLIC POLICY 141 (Elliott White ed., 1997).

Protection Agency (EPA) reports of toxic release do not report all sources of pollution. But, as Felton Earls puts it, "an adequate theory must explain why rates of violence differ by age, gender, and size of community, and whether it is environmental conditions that cause individuals to behave violently (social causation), or whether persons prone to behave violently create environments characterized by high rates of violence (social selection)."[30]

IV. EMPIRICAL EVIDENCE LINKING POLLUTION AND CRIME

We have tested the hypothesis that neurotoxicity is among the factors related to violent crime by using ecological data on the distribution of environmental pollutants to predict the rates of criminal violence. The EPA's Toxic Release Inventory (TRI) for lead and manganese was correlated with FBI crime reports from 1,165 counties in 16 states with a total population of over 130 million.[31] This approach cannot measure the effect of the individual's childhood environment. However, the realities of economic stratification suggest that the county of current residence has a high probability of being ecologically similar to the childhood environment. If so, for most of the population, present exposure to toxic metals should also be a rough measure of environmental exposure during development.

A. The Effects of Lead and Manganese

The distribution of manganese and lead pollution is highly skewed: over seventy-five percent of counties have no release of either manganese and its compounds, or lead and its compounds recorded in the TRI. Correlations between pollution from these two metals and several behavioral outcomes—crime, alcoholism and dropping out of high school—are significant but non-linear. Because toxicity occurs at levels of micrograms per gram, even an average level of toxic release (2,540.9 pounds/year of manganese and 4,101.5 pounds/year of lead) could adversely effect a substantial population.

When counties are dichotomized for the presence or absence of lead and manganese toxic releases, the two-way analysis of variance (ANOVA) shows that pollution from each of these neurotoxic metals is significantly associated with increased rates of violent crime: in the 166 counties with pollution from both sources, rates of violent offenses were 520 per 100,000, as compared to

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30. Felton J. Earls, *Violence and Today's Youth*, 4 FUTURE OF CHILDREN 4, 11 (1994).

31. The states were: Alabama, Arizona, California, Florida, Georgia, Illinois, Massachusetts, Mississippi, Minnesota, New Hampshire, New York, Pennsylvania, Texas, Vermont, Washington, and Wisconsin. Search of USA COUNTIES ON CD-ROM, *supra* note 16.

the sample average of 350 per 100,000. The effects are highly significant for both manganese toxic releases ($p = .0039$, $F1,1137 = 8.36$) and lead toxic releases ($p = .0001$, $F1,1137 = 31.22$), with no statistically significant interaction between them.[32]

These effects are confirmed by focusing on counties whose rates of violence are either well above average or unusually low.[33] Among the 310 counties with relatively high rates of the FBI's four categories of violent crime (more than 400/100,000 per year), releases of both manganese and lead are significantly higher than in the 255 low-crime counties with rates under 100/100,000 per year.[34] Other statistical measures, such as analysis of those counties in the top decile of toxic releases of either lead or manganese, and the independent effects of toxic releases of the element of each metal and its compounds, are consistent with the hypothesis.

These correlations between environmental pollution and crime do not seem to be statistical artifacts. Multiple regression models, predicting the level of pollution in each county on the basis of socioeconomic and ethnic variables—e.g., population composition, per capita income, unemployment rates—explain less than five percent of the variance for either lead or manganese releases. Although few rural counties (those with population densities below twenty per square mile) have reports of either lead or manganese releases, the geographic distribution of pollution from these sources seems to be a distinct variable which criminologists have not adequately studied.

B. The Effects of Alcoholism

Because consumption of alcohol is frequently associated with violence, if alterations in brain chemistry increase the risk of violent aggression, one might expect interactions at the population level among environmental pollution, rates of alcoholism, and violent behavior. To test this extension of the hypothesis, deaths from all forms of alcoholism were dichotomized at the sample average and used as a measure of the varying degree of alcohol usage in each county. The three way ANOVA—presented as Figure 5, *infra*—shows that when this factor is combined with exposure to pollution from lead and manganese, all three factors are highly significant (lead $p = .0001$, $F1,1111 = 48.114$; manganese $p = .0001$, $F1,1111 = 16.836$; alcoholism $p = .0001$, $F1,1111 = 70.217$), with significant interactions between

32. *See infra* Figure 3.
33. *See infra* Figure 4.
34. Search of USA COUNTIES ON CD-ROM, *supra* note 16.

manganese toxic release and alcoholism ($p = .0001$, $F1,1111 = 15.512$) and between lead toxic release and alcoholism ($p = .0347$, $F1,1111 = 4.473$).

Students of human behavior have rarely explored such complex interactions or synergies between environmental toxins and consumption. In laboratory experiments, as noted above, exposure to a toxic metal like manganese has effects that vary depending on diet. The effects of lead, manganese, and alcohol seem to have a similar synergy. In counties with no reported releases of lead or manganese and below average rates of alcoholism, rates of violent crime are below average (250/100,000). In contrast, the 57 counties of our sample with toxic releases from both metals and above average alcoholism rates have three times as much violent crime, with a county rate of 790/100,000. Although epidemiologists are increasingly aware of the importance of such synergies or interactions, most conventional models in criminology have looked at each variable independently.

C. The Effects of Other Risk Factors

To ascertain how these effects might relate to other risk factors associated with violence, a multiple regression was computed using twenty-two socioeconomic and demographic variables to predict rates of violent crime. Although such multiple-regression equations are notoriously sensitive to the variables included and the linearity of effects, these results are consistent with the hypothesis that both alcoholism and dietary insufficiencies are risk factors that interact with environmental pollution. Since about three-fourths of counties studied had no releases of lead or of manganese and the effects of pollution are nonlinear, it is not surprising that levels of toxic releases are not independent predictors of violent crime in the regression model.[35] It is interesting, however, that two of the variables with the highest standardized coefficients are alcoholism, which is associated with *higher* crime rates, and per capita expenditure on welfare, which is associated with *lower* rates of violent crime (perhaps because of the contribution of food stamps to improved diet among the poor). Independent contributions of white, black, and hispanic poverty, controlling for rates of unemployment, urbanism, and the overall wealth of a county, also suggest that violent behavior is more likely for those with dietary insufficiencies in calcium and other essential

35. That this is an artifact of the size of the sample is confirmed by the comparable statistics from a global sample of 2899 counties from the 48 contiguous states, in which both multiple regression and structural co-variate analyses confirm the independent contribution of environmental neurotoxic releases as a risk factor in crime. *See* MASTERS ET AL., *supra* note 23, at 34, 36; *see also* Masters et al., *supra* note 29, at 164-71.

elements, which lead to increased uptake of toxic substances in the environment.

Because leaded paint and aging water systems, especially in decaying urban areas, can be a significant source of lead and manganese, the data presented here probably underestimate the effects of toxic metals. A study now underway is analyzing towns and cities in Massachusetts to assess the effects of lead in public water supplies on a number of behavioral measures.[36] Preliminary findings show that higher average levels of lead correlate with lower standardized educational test results, increased high school drop-out rates, increased numbers of single-parent families, and increased crime rates.[37] Indeed, controlling for other socioeconomic and demographic variables, lead seems to be one of the strongest factors in predicting rates of both violent and property crimes.

Because calcium, zinc or other vitamin and mineral insufficiencies are associated with uptake of lead and manganese, differential vulnerability to toxic chemicals must be considered in explaining high rates of violent crime, particularly among population groups with poor diets and low levels of breast feeding. As a number of studies have shown, blacks are differentially vulnerable to the uptake of toxic metals such as lead.[38] Indeed, it would seem that *all* of the effects analyzed in the *Bell Curve*,[39] especially with regard to black-white differences in intelligence and crime, can be traced to differential exposure to, and differential uptake of, neurotoxic metals.[40]

V. PRELIMINARY IMPLICATIONS FOR THE LAW

This biological perspective on violent behavior diverges from contemporary sociological and legal conceptions of crime and criminals in a number of ways. On the one hand, it is unlikely that any single risk factor for violence will provide a satisfactory explanation of criminal behavior; rather, multiple factors, including the offender's chosen lifestyle, interact to explain any one instance of criminal violence. If this is so, there is no need to alter traditional notions of mens rea, and hence of legal responsibility, due to the

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36. This work, being conducted with Myron Coplan of Intellequity, located in Natick, Massachusetts, utilizes a database developed by Prof. Adrian Bailey of the Department of Geography of Dartmouth College, and his colleagues, extending it to include rates of crime and levels of lead in public water supplies.

37. *See* Masters et al., *supra* note 29.

38. *See infra* Figure 6.

39. RICHARD J. HERRNSTEIN & CHARLES MURRAY, THE BELL CURVE: INTELLIGENCE AND CLASS STRUCTURE IN AMERICAN LIFE (1994).

40. For evidence of the differential vulnerability of blacks to neurotoxins, see Masters et al., *supra* note 30, at 164-71.

discovery of objective factors that modify human brain chemistry and central nervous system function. Because diet, consumption of alcohol or psychoactive drugs, and preferred patterns of social behavior can influence the brain's capacity to inhibit aggressive impulses, knowledge of risk factors influencing neurochemistry and emotional responsiveness should not be viewed as disculpatory. The so-called "Twinkie defense"—which in any event has never been broadly accepted—would be both imprudent and unnecessary as a legal application of the biological findings described in this symposium.[41]

On the other hand, however, both crime prevention and effective sentencing need to consider a broader range of risk factors than has hitherto been customary. In particular, insofar as there are differences in the factors leading to violent crime, offenders guilty of the same offense should probably receive different punishments or probationary conditions. Such a trend will, however, pose serious problems for the law because case disposition would increasingly have to be based on the actor and not the act. In place of mandatory sentencing and the notion that all those convicted of a given offense deserve identical punishments, it would become essential to find out what made each violent offender different from nonviolent offenders.

For example, at the minimum, it will be necessary to distinguish between episodic or low risk criminals and repeat offenders (like those described by Professor Denno), who commit a large number of crimes even though they are a small proportion of the population.[42] Rehabilitation seems least promising for those who have been called "primary psychopaths" or for whom there is a physiological deficit in integrating emotion and cognition (like Phineas Gage, whose abnormal social behavior was described at this symposium by Oliver Goodenough).[43] In contrast, criminal offenders with reversible neurochemical deficits may occasionally commit violent acts but be more likely to be influenced by measures of prevention and treatment.

This distinction may be particularly relevant to what has been described as the epidemic of violence in American society. Rates of violent crime vary enormously from one city or county to another. Since the population frequency of primary psychopathy has been viewed as roughly constant across many social and cultural circumstances, irreversible genetic or neuroanatomical deficits are not likely to explain the most salient dimensions

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41. *See generally* WILSON, *supra* note 2 (arguing that the tendency to allow social scientific explanations of behavior to generate disculpatory evidence is undermining our criminal law, with serious implications for the public support for our legal system).

42. *See* Denno, *supra* note 21, at 311.

43. *See* Oliver R. Goodenough, *Biology, Behavior, and Criminal Law: Seeking a Responsible Approach to an Inevitable Interchange*, 22 VT. L. REV. 263 (1997).

of violent crime in American society. Paradoxically, biology can suggest some environmental risk factors that might distinguish repeat offenders from the episodic offenders.

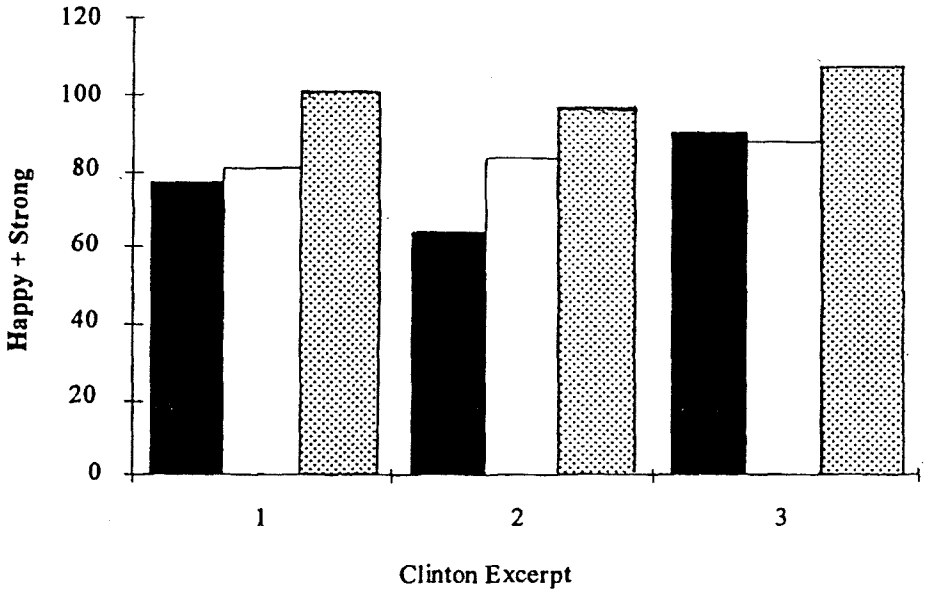
Figure 1

MEDIAN DESCRIPTIVE RATINGS — VERMONT PRISON INMATES

VIOLENT OFFENDERS (BLACK BARS, n = 17)
NONVIOLENT OFFENDERS (WHITE BARS, n = 15)
NONPRISONEER CONTROLS (GREY BARS, n = 89)

VIEWING NEUTRAL EXCERPTS OF CLINTON

Median Positive Description



Median Negative Description

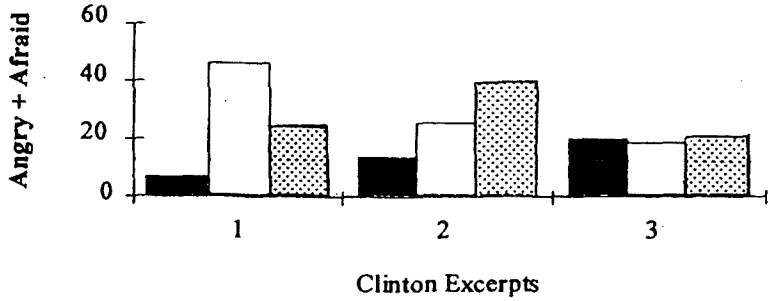


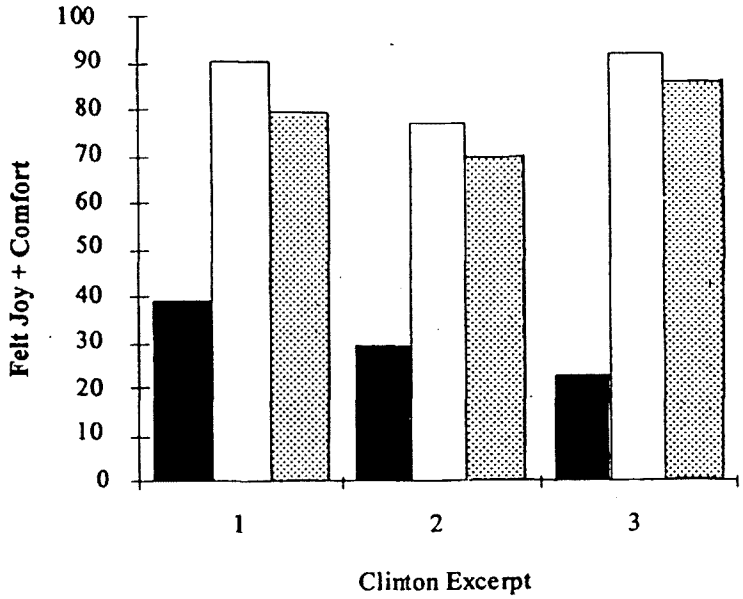
Figure 2

MEDIAN EMOTIONAL RESPONSES - VERMONT PRISON INMATES

VIOLENT OFFENDERS (BLACK BARS, n = 17)
NONVIOLENT OFFENDERS (WHITE BARS, n = 15)
NONPRISONER CONTROLS (GREY BARS, n = 89)

VIEWING NEUTRAL EXCERPTS OF CLINTON

Median Positive Emotion



Median Negative Emotion

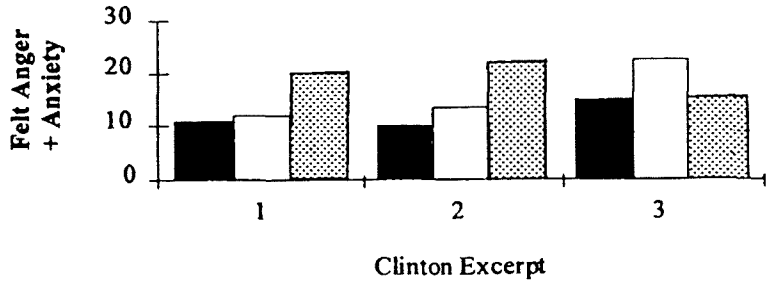
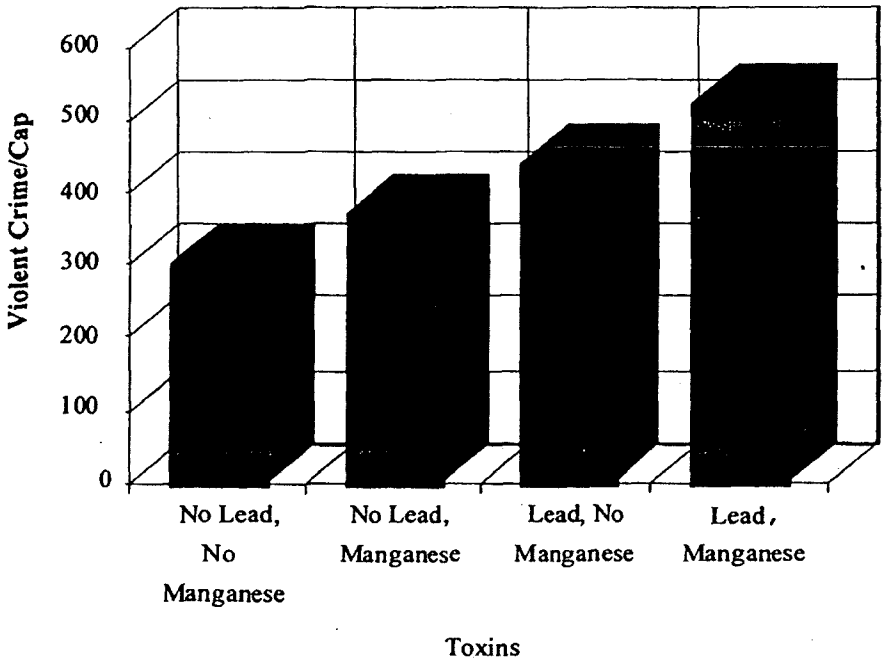


Figure 3

Toxic Release of Lead and Manganese as a Predictor of Rates of Violent Crime, by County

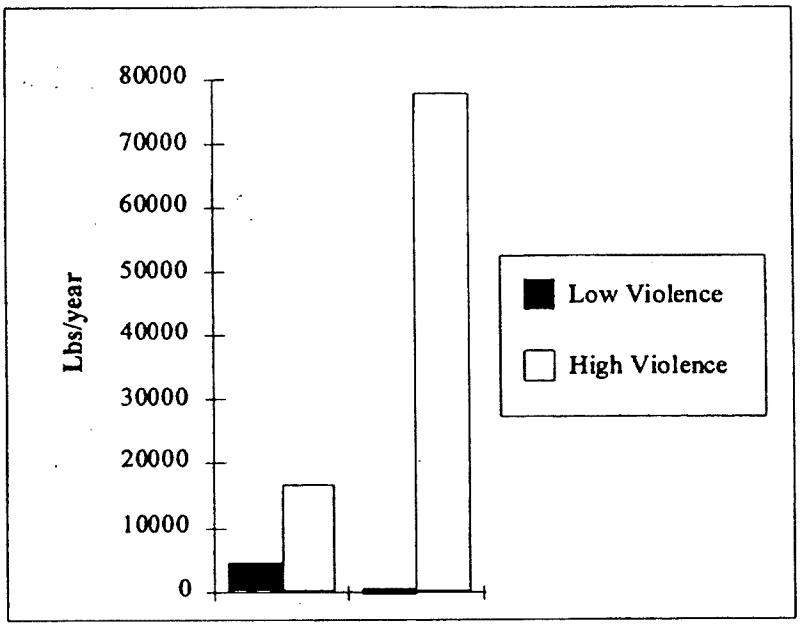
(16 State Sample, 1165 Counties)



Source: FBI Crime statistics, 1991 (total rate of homicide, aggravated assault, sexual assault, and robbery). Total releases of toxic metals: Environmental Protection Agency, Toxic Release Inventory, 1991. Parentheses: number of counties. Data from 1141 counties (24 counties deleted from sample for missing data). Significance: total manganese release: $p = .0039$, $F_{1,1137} = 8.36$; total lead release: $p = .0001$, $F_{1,1137} = 31.22$; interaction between manganese and lead, n.s.

Figure 4
Annual Toxic Release Inventory of Manganese and Lead in Counties with Low and High Crime Rates

(16 State Sample, 1165 counties)



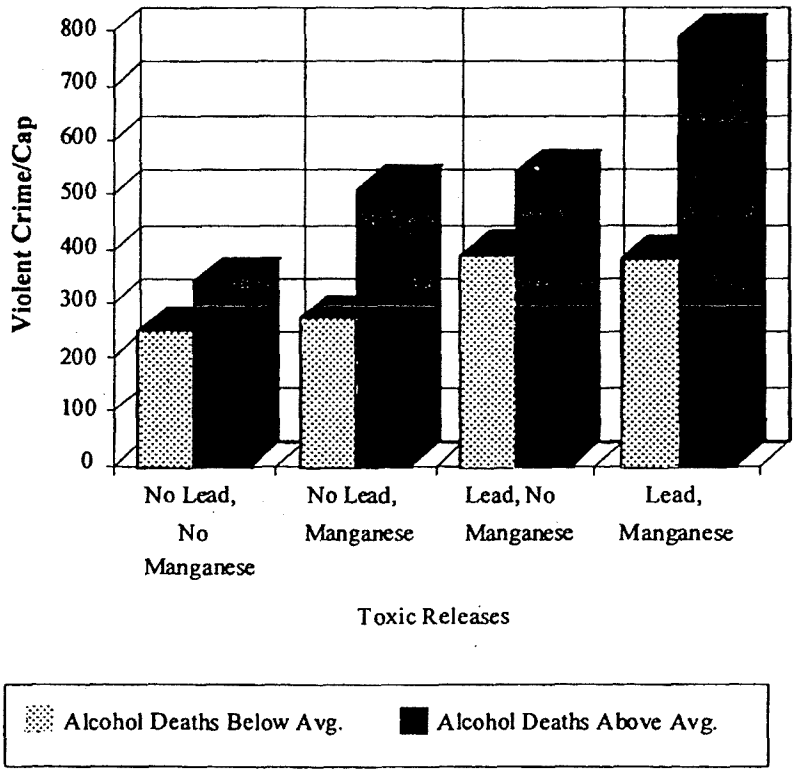
Low violence counties (n = 255): < 100/100,000 per year
High violence counties (n = 310): > 400/100,000 per year
Statistical significance: Manganese, p = .0094; Lead, p =.0027
(1 tailed Chi-square)

Note: The effects shown above can be confirmed by "contingency" analysis. Approximately three-fourths of counties in the total sample had no reported release of either manganese or lead. Among counties within top 10% in lead toxicity for the entire sample, with releases of over 8594 pounds per year, 48 had above average rates of violent crime (greater than 400/100,000) and only 1 had a rate below 100/100,000. Of the 47 counties in the top 10% of manganese toxicity (5879 pounds per year), 32 had above average rates of violence and only 15 had rates below 100/100,000. Similar effects could be seen for low levels of toxic release (less than 8594 pound of lead or 5879 pounds of manganese). Counties with no reported release of lead or manganese were about equally divided between high and low violence rates. This statistical measure of the correlation between toxic metals and violent crime is also highly significant (lead, p = .0001; manganese, p = .0008)

Figure 5

Association of Manganese and Lead Pollution and Rates of Alcoholism with Violent Crime

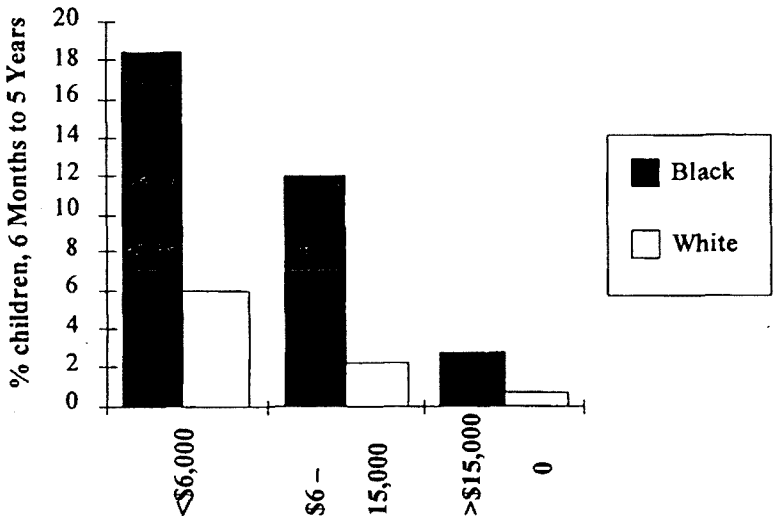
(16 State Sample, 1165 Counties)



NOTES: Three-way ANOVA, data from 1119 counties (22 counties from Table 3 deleted for missing data on alcoholism). Rates of Death from Alcoholism dichotomized at average (42.4/100,000 per year). Significance of main effects: Presence or Absence of Manganese Pollution (TRI): $p = .0001$, $F_{1,1111} = 16.836$; Lead Pollution (TRI): $p = .0001$, $F_{1,1111} = 48.114$; Alcoholism: p. .0001, $F_{1,1111} = 70.217$. Significance of interactions: Manganese and alcoholism: $p = .0001$, $F_{1,1111} = 15.512$; Lead and Alcoholism: $p = .0347$, $F_{1,1111} = 4.473$.

Figure 6

Percent Children with PbB > 30ug/dL, by Family Income



Source: Jane S. Lin-Fu, "Modern History of Lead Poisoning," in Herbert Needleman, ed., *Human Lead Exposure* (Boca Raton, FL: CRC Press, 1992), Table 2, p. 37; data from NHANES II Survey (1976-1980).

2.
ENVIRONMENTAL POLLUTION, NEUROTOXICITY, AND CRIMINAL VIOLENCE

ROGER D.MASTERS*, BRIAN HONE† and ANIL DOSHI‡

"the Opinion of this mischievous Effect from Lead is at least above Sixty Years old; and you will observe with Concern how long a useful Truth may be known and exist, before it is generally receiv'd and practised on."

Benjamin Franklin[1]

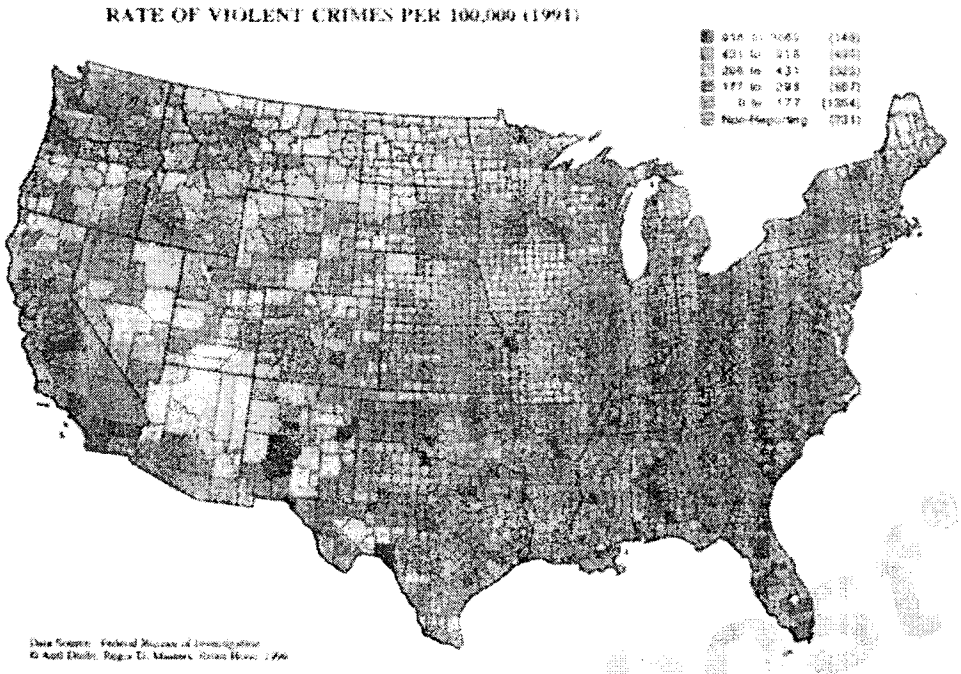
"Regarding violence in our society as purely a sociologic matter, or one of law enforcement, has led to unmitigated failure. It is time to test further whether violence can be amenable to medical/public health interventions."

Dr. C.Everett Koop and Dr. George Lundberg[2]

I. INTRODUCTION

This chapter will explore the hypothesis that uptake of neurotoxic metals may be among the many factors contributing to the unusually high and widely varying rates of violent crime in the United States. The hypothesis rests on findings that loss of impulse control and increased aggressive behavior can be related to abnormalities of brain chemistry caused by a complex interaction of insufficiencies of essential vitamins and minerals, toxic uptake, alcoholism, and social stress. After reviewing evidence at the level of individual neurochemistry, ecological data will be presented to show that, controlling for standard socio-economic and demographic variables, environmental releases of lead and manganese predict geographical differences in rates of violent crime. Although this approach to criminal violence might seem at first unduly reductionist, analysis of the complex interactions between brain biochemistry, environment, and behavior explains otherwise puzzling variations in crime rates and suggests potentially effective approaches to crime prevention.

To provide an adequate account of violence in contemporary industrial societies, it will be useful to take an epidemiological approach to geographical and historical variations in crime rates that are not well explained—and often not examined—in conventional analyses[3]. This is particularly appropriate in the United States, where violence is, in the words of two leading physicians, "a public health emergency, largely unresponsive to methods thus far used in its control"[2]. Despite a recent levelling of rates of violent crime, the United States still faces what the Center for Disease Control has called an "epidemic" of violence. Although prevailing theories of violent crime properly implicate a host of social, economic, and psychological variables[4,5], these factors alone do not adequately explain why American counties have rates of violent crime that vary from less than 100 to over 3000 per 100,000 population (Map 1).



Map 1

Although much is known about the behavioral effects of abnormal brain biochemistry, this information has hitherto not been brought together to account for these variations in rates of criminal violence. In part this is because most discussions of environmental toxins focus on such health risks as cancer. There is, however, increasing awareness that neurotoxins such as lead and manganese also have subclinical effects on brain biochemistry leading to learning disabilities, poor impulse control, and an increased risk of aggressiveness. An analysis of such relationships between environmental pollution and violence must rest on a complex, multi-causal analysis of human behavior. In this view, neurotoxicity is only one cause among many, at most functioning as a catalyst which, in addition to poverty, social stress, alcohol or drug abuse, individual character, and other social factors, increases the likelihood that an individual will commit a violent crime (below, Section II).

As in other epidemiological studies, five distinct relationships need to be established to confirm the hypothesized contribution of environmental neurotoxicity to high rates of violence: (1) correlation, (2) prediction, (3) function, (4) transmission, and (5) ecological verification.

(1) **Correlation** *associating toxic uptake with criminal violence at the individual level.* To be credible, the first condition of the neurotoxicity hypothesis must be evidence that individuals who engage in criminal violence are more likely to have absorbed a toxic chemical than comparable controls. It has long been known

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that serious behavioral and cognitive deficits are caused by exposure to lead, especially during infancy and childhood. Subclinical lead poisoning has been correlated with learning disabilities, attention deficit disorder, and other psychological conditions sometimes associated with deviant behavior. Similar effects have been found for manganese. In seven populations of criminal offenders whose bodily uptake of toxic metals has been studied, lead, manganese, cadmium, or other toxic metals have been significantly elevated in the violent offenders compared to nonviolent criminals or controls (below, Section IIIA).

(2) **Prediction** *showing that young children with higher levels of toxic uptake are more likely, later in life, to engage in aggressive or violent behavior.* Correlational studies need to be confirmed by long-term, prospective research showing that toxicity allows prediction of future violent behavior. Such research only seems to have been conducted with regard to lead. In two studies of this neurotoxin, using different methods, lead uptake at age 7 was significantly predictive of juvenile delinquency or increased aggression in teen-age years and early adulthood (below, Section IIIB)

(3) **Functional effects** *of the neurotoxin that could account for loss of impulse control and increased violence.* Lead has both negative consequences for neuroanatomical development and functional effects degrading catecholamines and other basic neurotransmitters. Of particular importance is the deleterious effect of lead on glia, the brain cells that play an essential role in inhibition and detoxification. Manganese has the effect of lowering levels of serotonin and dopamine, neurotransmitters associated with impulse control and planning. Other neurotoxins may contribute to violent behavior, since cadmium, aluminum, and other metals have also been found to have deleterious effects on the brain. These biochemical factors interact in complex ways: for example, because lead degrades the detoxification capacity of the brain, exposure to lead pollution will enhance the effects of alcohol, drugs, or other toxins. Combinations of several toxic elements are probably synergistic rather than additive, with the extent of brain dysfunction also depending on diet, allergies, social status, stress, and individual experience (below, Section IV).

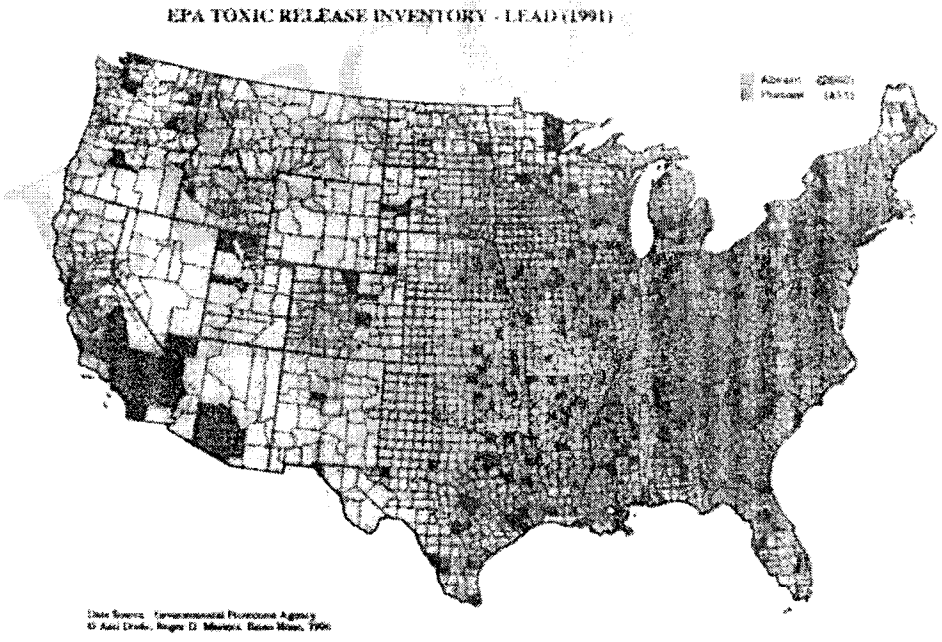
(4) **Transmission** *by known pathways must deliver neurotoxic elements to individuals in quantities associated with violent behavior.* Despite the prohibition of leaded gasoline and paint, environmental pollution of lead and other neurotoxins remains a serious problem. In four American cities, high traffic corridors contain soil residues of leaded gasoline that cause unhealthy levels of lead absorption in children. In the state of Massachusetts, controlling for other socio-economic factors, individuals absorb significantly higher amounts of lead in towns where industrial factories are located than in other localities. In addition to contemporary effects of industrial releases of neurotoxins, residues of particulate matter remain in soils for long periods, contaminating dirt with which children play. Other pathways include lead and manganese in aging public water systems or pipes within residential units and peeling leaded paint. In addition to such environmental exposure, dietary sources of manganese may be important. Infants absorb manganese in high levels from baby formula, and some crops absorb manganese from soil and fertilizers. Susceptibility to neuronal uptake of toxic metals is greatly increased for individuals with a diet low in calcium, zinc, and other essential vitamins. Lead, manganese, and other toxic elements thus probably have a disproportionate effect on the poor, since the combination of dietary insufficiency and environmental pollution has effects not observed when only one of the two is present (below, Section V).

(5) **Ecological measures** *of environmental pollution, controlling for other variables, should correlate with higher rates of violent crime.* Although the foregoing data are consistent with the possibility that neurotoxic metals could cause loss of impulse control, antisocial behavior, and violence (as well as learning disabilities, memory deficits, or physical disability), the role of environmental pollution needs to be more directly tested. Geographical data from counties in the United States provide a valuable test since ecological differences in environmental pollution should predict otherwise unexplained variations in rates of criminal violence. For this purpose, a dataset of all counties in the United States was constructed, integrating the

U.S. Environmental Protection Agency's Toxic Release Inventory (TRI), for lead and for manganese, crime reports from the Federal Bureau of Investigation, rates of alcoholism from the Department of Health and Human Services, and socio-economic and demographic data from the Census Bureau. Controlling for such conventional factors as income, population density, and ethnic composition, environmental pollution had an independent effect on rates of violent crime (measured as total homicide, sexual abuse, aggressive assault, and robbery). When all counties are dichotomized into presence or absence of industrial lead pollution (Map 2) and presence or absence of industrial manganese releases (Map 3), and higher and lower than average rate of alcoholism (Map 4), counties with all three factors of neurotoxicity have rates of violent crime over 3 times that of the national average (Fig. 1, see below, Section VI).

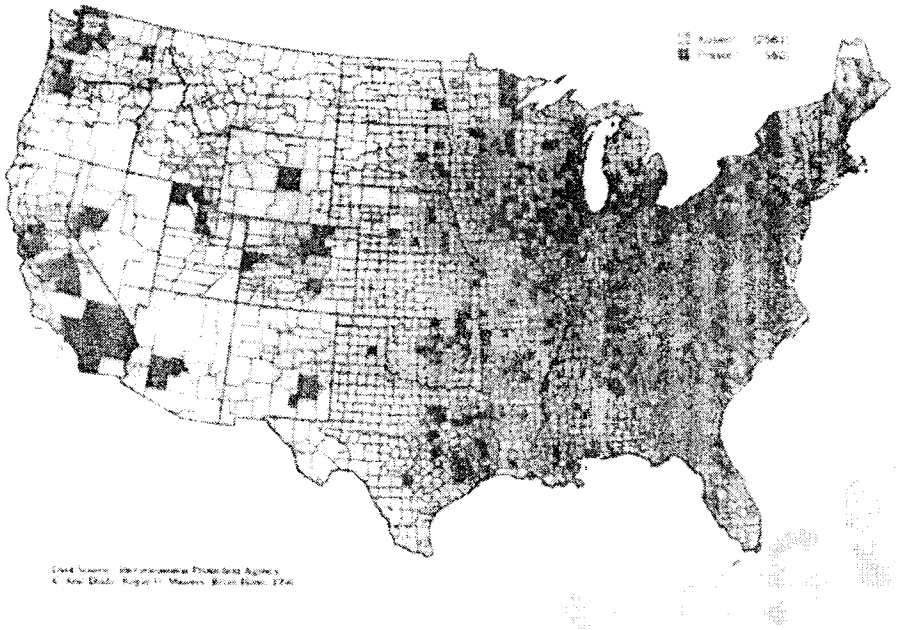
Neurotoxicity is obviously only one of many factors contributing to violence, but it may be especially important in explaining why rates of crime have differed so widely by geographical region and by ethnic group. Local rates of pollution are largely independent of such variables as unemployment rates, high school dropouts, and police per capita. Both multiple regression analysis and a structural co-variate model indicate that, controlling for socio-economic and demographic factors, environmental pathways of neurotoxic metals significantly contribute to rates of violent crime.

This exploration of relationships between brain biochemistry, diet, neurotoxic metals and violent behavior has obvious relevance to public policy. Crime prevention and improved educational performance may be greatly enhanced by parent-training in breast-feeding and proper diet. Vitamin and mineral supplementation, which some studies suggest may even increase IQ, could be particularly important in improved school performance and cognitive development. If releases of neurotoxic metals are associated with rates of crime, reducing environmental pollution takes on higher priority. Such a finding could also aid the criminal justice system by improving predictions of recidivism, which are currently little better than



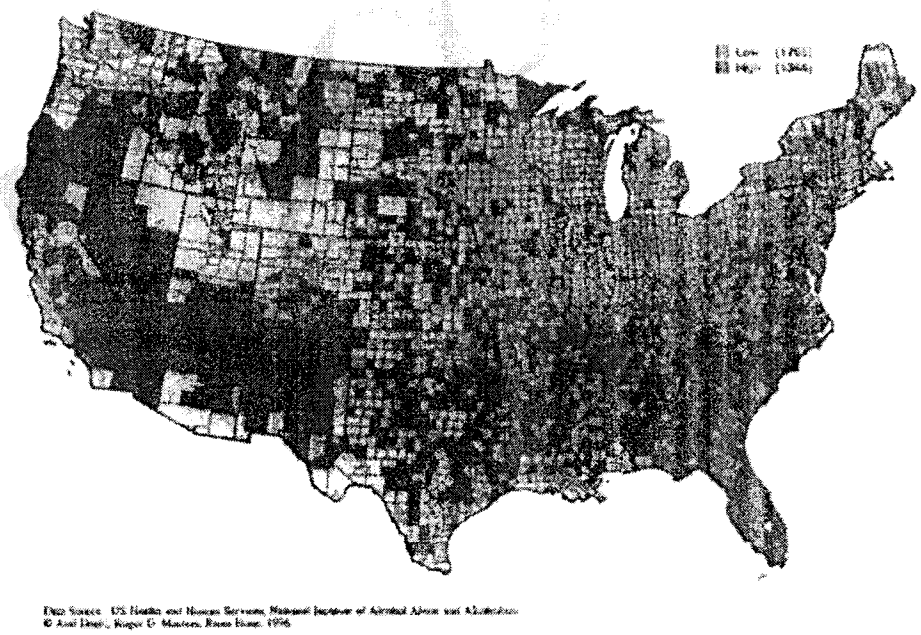
Map. 2

EPA TOXIC RELEASE INVENTORY - MANGANESE (1991)



Map. 3

RATE OF ALCOHOL RELATED DEATHS



Map. 4

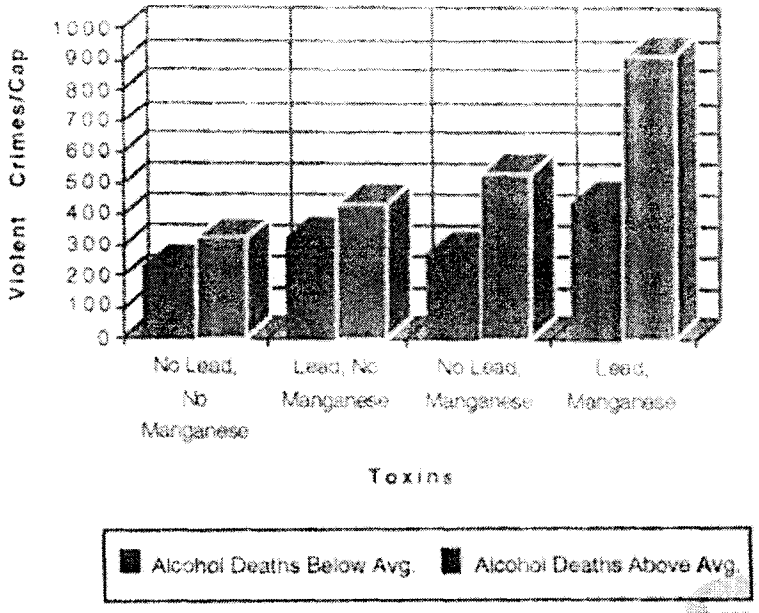


Figure 1 Association of manganese and lead pollution and rates of alcoholism with violent crime (50 state sample, 2899 Counties). *Notes:* Three-way ANOVA, data from all 2899 counties in U.S. reporting 1991 crime statistics to FBI. Rates of Death from Alcoholism dichotomized at national average (47.2/ 10,000). Significance of main effects: Presence or Absence of Manganese Pollution (TRI): t ratio= 11.32, p<.0001, $F_{1,2898}$=128.25; Lead Pollution (TRI): *t* ratio=9.66, *p*<0.0001, $F_{1,2898}$=93.22; Alcoholism: *t* ratio=11.99, *p*<0.0001, $F_{1,2898}$=143.64. Significance of interactions: Manganese and alcoholism: *t* ratio=6.86, *p*<0.0001, $F_{1,2898}$=47.04; Lead and Alcoholism: *t* ratio=3.00, *p*<.0027, $F_{1,2898}$=9.01; Lead and Manganese: *t* ratio=3.91, *p*<0.0001, $F_{1,2898}$=15.30; Lead, Manganese and Alcoholism: *p*<0.0169, *t* ratio=2.39, $F_{1,2898}$=5.72.

chance, and by pointing to the use of vitamin and mineral normalization to improve rehabilitation (below, Section VII). Before major policy changes are proposed, however, it is essential to confirm the hypothesized relationships with further cross-cultural and experimental studies. This caution is especially necessary because the suggestion that violent crime might be associated with abnormalities in brain biochemistry has been highly controversial.[6]

II.
NEUROTOXICITY, BRAIN BIOCHEMISTRY, AND BEHAVIOR

The study of possible links between environmental pollution and crime requires a focus on biochemical toxins that change brain structure and function, often in complex interactions with diet, alcohol or drug use, stress, and other ecological or cultural factors. This approach assumes that neurotoxicity is only one cause among many, at most functioning as a catalyst which, in addition to poverty, individual character, and other social factors, sometimes increases the risk of violent crime[7]. To clarify the relationship between these multiple levels of analysis, the pathways linking toxic exposure to behavior will be discussed in general before describing the specific effects attributed to lead and manganese. This is particularly important

because discussions of environmental toxins have often focused on such health risks as cancer rather than on brain biochemistry and behavior.

Exposure to toxic elements, poor diet, and substance abuse have interacting effects on brain function and social behavior: "Changed brain chemistry can alter behaviour, and changed behaviour can alter brain chemistry: the interaction is two way. It therefore follows that behaviour, cognition, social interactions, and other expressions of brain function are subject not only to the social environment but also to certain aspects of the chemical environment. The relevant chemical factors include (a) neurotoxic pollutants in general, of which lead is evidently now the most serious in its impact, (b) certain common nutrient deficiencies, particularly of zinc, and (c) neurotoxins of voluntary abuse, of which ethanol is still probably producing the most widespread social damage"[8].

Among the biochemical factors that influence brain function, metal ions such calcium, potassium, and zinc play an important role[9]. As in other human cells, neuronal cell surfaces must have an equilibrium of positive ionic charges ("cations") and negative ionic charges ("anions"). Because neurons form an electrochemical system of communication, deficits in calcium and zinc can lead to neuronal uptake of toxic cations like lead and manganese; the resulting abnormalities of bioinorganic chemistry can be harmful when concentrations of key elements are either too low or too high[10].

Toxic elements can have both direct and indirect effects on behavior. On the one hand, neurotoxicity may result in abnormal neuronal development or destruction of neurons and brain structures; on the other, neurotoxins can interfere with normal brain biochemistry, degrading the level of necessary neurotransmitters and regulatory substances. In many cases, these effects depend on ratios or interactions between potentially toxic metals rather than absolute values of any single element[11,12]. Of particular importance may be synergistic interactions between elements whose toxicity is greatly multiplied when they are combined.

- **Direct or "frank" toxicity.** In some cases, uptake of neurotoxic elements leads to clinically diagnosed conditions. For example, prolonged exposure to manganese dust from mines or factories leads to the condition known as "industrial manganism," with symptoms like those of Parkinson's Disease[13]. Extreme concentrations of manganese have also been associated violence in environments with mining operations or industrial exposure, as on Groote Eylandt off Australia[14,15]. Exposure to copper during neonatal development has been associated with abnormal structures of the hippocampus, a key brain structure for learning. And, of course, the lasting effects of fetal alcohol syndrome and lead poisoning on normal brain development are well documented[16].
- **Indirect or subclinical toxicity.** Changes in brain biochemistry that are at first not obviously associated with environmental pollution can also have significant effects on behavior. Neurotoxic elements can lower levels of basic neurotransmitters, disturbing normal brain function by reducing inhibition and thereby contributing an additional "risk factor" for violence. For example, neuronal uptake of manganese has the effect of reducing neuronal levels of serotonin while increasing serotonin concentrations elsewhere in the body (Fig. 2).

The effects of abnormal levels of neurotoxic metals on neurotransmitter function and behavior depend on many factors. Dietary deficits in calcium, zinc, and essential vitamins or minerals can result in greater absorption of lead, manganese, and other toxic metals from water supplies or food and uptake of such neurotoxic elements inbrain cells. For example, laboratory animals whose diet included excess manganese did not absorb it when calcium levels were normal, whereas manganese uptake became significant when their diet was deficient in calcium (Fig. 3)[17]. As will be noted below, this effect could be associated with social

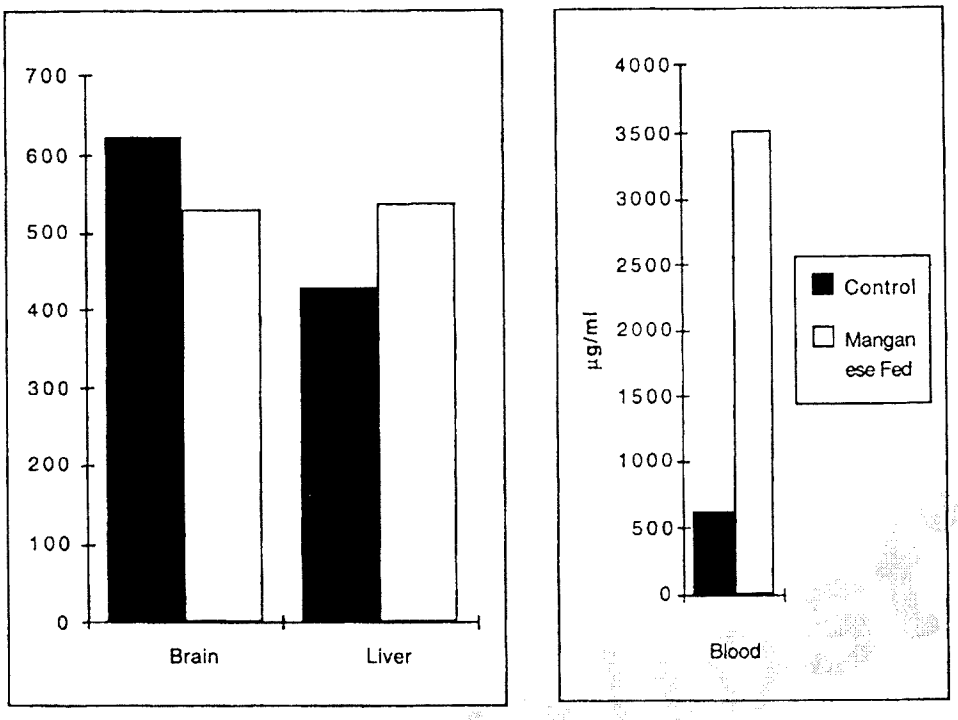


Figure 2 Effects of mangnese feeding on serotonin levels. Serotonin levels in brain and liver: mg/g tissue, wet weight; in blood: mg/ml. Significance: effects on brain and blood, $p<0.05$; on liver, n.s. *Source:* M.Kimura, N.Yagi and Y.Itokawa, "Effect of subacute manganese feeding on serotonin metabolism in the rat," *J. Toxicology and Environmental Health* **4**, 701–707 (1978).

class both because diets are likely to be poorly balanced and because toxic substances are more likely to be present in environments marked by industrial pollution, aging water systems, and housing with lead paint.

Because individuals differ in the level and activity of neurotransmitters like serotonin[18–20], the effects of neurotoxic elements that degrade neurotransmitter function are also likely to interact with personality. Equally important is social status. Serotonin levels are usually higher among dominant individuals than subordinates, a difference that occurs as a consequence of higher status in humans as well as nonhuman primates[21]. Conversely, chronic stress (which is often associated with marginal social status or personal insecurity) increases cortisol levels in the brain and thereby lowers functional levels of catecholamines such as serotonin and dopamine[22,23].

Additional biochemical imbalances have been associated with hypoglycemia, a biochemical deficiency in glucose, particularly when combined with alcohol or drugs[24,25]. Alcohol or drug consumption possibly represent crude efforts at self-medication triggered by these imbalances. Criminal violence could thus be traced to the interactive effects of personality, dietary imbalance, poverty and social disintegration, social marginality, substance abuse, and toxic elements in the environment[26].

The traditional concept of causality adopted by most social scientists is thus not appropriate for the analysis of the ways brain biochemistry and behavior areinfluenced by the interactions of poverty and social stress, lifestyle, substance abuse, personality, and environmental toxins. Instead, it is necessary to conceive of multiple risk factors, some originating in the social environment, some from the physical environment,

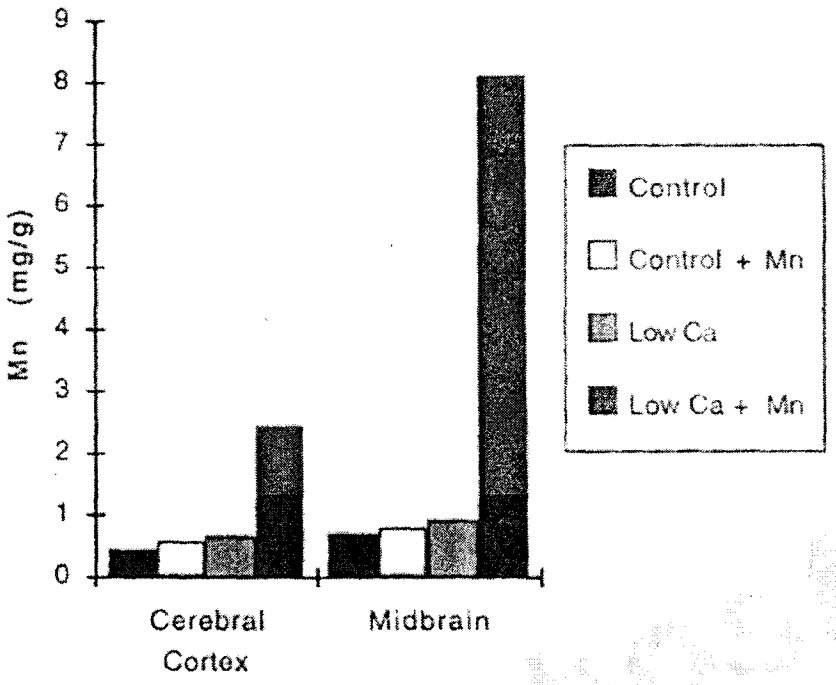


Figure 3 Calcium deficiency and manganese uptake. Source: V.A.Murphy, J.M.Rosenberg, Q.R.Smith, and S.I.Rapoport. "Elevation of brain manganese in calcium-deficient rats," *Neuro Toxicology,* **12,** 255–264 (1991). *Note:* Control: normal diet (7 g Ca/kg and 0.055 g Mn/kg food with 1.92g/l sodium acetate trihydrate in water); Control+Mn: manganese supplement only (normal diet plus 1.79g/l Mn (II) acetate tetrahydrate) in water, 0.4 g Mn/l); Low Ca: calcium deficiency (0.1 g Ca/kg and 1055 g Mn/kg food with Na acetate in water); Low Ca+Mn: manganese supplement and calcium deficiency combined. In Lo Ca+Mn group, levels of manganese were within range of values in figure: cerebral cortex=2.45mg/g; Caudate nucleus=5.48 mg/g; Hippocampus=3.64mg/g; Hypothalamus=6.32 mg/g; Thalamus=4.44mg/g; Midbrain Colliculi=8.10mg/g; Cerebellum=4.27mg/g; Pons-Medulla=6.27mg/g; Spinal Cord=3.20mg/g.

and some from the genetics and individual life history of individuals. While complex, this approach avoids a puzzle in the typical explanations of violence as the product of social factors. Sociological approaches to crime lack a theoretical framework that explains why factors like violence on TV or availability of handguns and drugs trigger violent behavior in only a small proportion of the population as well as why this proportion should vary from one place to another. Part of the missing linkage may concern the way physical as well as social environments and lifestyles effect brain chemistry and behavior.

III.
NEUROTOXICITY AND VIOLENCE AT THE INDIVIDUAL LEVEL

If neurotoxicity is to be linked to criminal violence, the first requisite is evidence that individuals who have absorbed toxic elements from the environment are significantly more likely to engage in aggressive or violent behavior. As in the study of epidemiology in public health, such evidence takes two forms:

(1) **correlational studies,** in which violent offenders are compared to nonviolent criminals and law-abiding controls at a specific time; and
(2) **prospective studies,** in which individuals are followed from childhood to maturity, with measures of toxic uptake in childhood used to predict later behavior.

Studies using both approaches have found that individuals who have absorbed toxic metals such as lead and manganese are significantly "at risk" to engage in violent or aggressive behavior.

Various methods have been used to assess the uptake of neurotoxic elements by an individual. Although one frequent technique for assessing neurotoxicity is the analysis of head hair[27,28], this method is sometimes questioned on grounds of contamination by shampoos or hair treatments. Other measures, such as blood, teeth, bone, and saliva, also have their limitations, however, and all have been used in studies cited here correlating toxic uptake with behavioral abnormalities. While the bodily tissue or fluid used as a measure may influence results in some cases, the specific assay used in any one study cannot be used to deny the general finding that uptake of toxic elements has deleterious behavioral consequences for humans.

Section A.
Correlational Studies

A review of the literature indicates seven different samples of prison inmates whose levels of toxic metals were studied. In all seven groups, either lead and cadmium or manganese were significantly higher in head hair of violent offenders than in nonviolent inmates or controls (Table I)[12,29–32]. In addition, silicon was significantly elevated in two samples of violent offenders, and, in one group, mercury was abnormally high. Equally interesting is the fact that lithium, which has been found to detoxify manganese, was abnormally low in two of the seven samples. Other correlational studies are consistent with the hypothesis that violent offenders are more likely to have abnormal brain biochemistry than non-violent criminals or law abiding citizens[33,34].

Section B.
Prospective Studies

Claims that neurotoxicity has behavioral effects, like supposed discoveries of the cause of any new disease, are properly subject to the objection that correlational studies are in themselves not conclusive[35]. To meet this objection, prospective research provides critical evidence that neurotoxicity in childhood predicts aggressive behavior and crime among juveniles and young adults. Because such prospective studies measure levels of neurotoxicity years before violent behavior is observed, they provide even stronger evidence that individual absorption of toxic elements is a major risk factor for violence. In the largest and longest of these studies, a longitudinal biosocial study of 1000 black residents of Philadelphia from birth to 22[8], both lead intoxication and anemia at age 7 were significant predictors of the number of juvenile offenses, seriousness of juvenile offenses and number of adult offenses for males (Table II)[36]. Another study found lead absorption in bones in childhood to predict aggressive behavior later in life[37].

Other approaches to the prediction of violent behavior also implicate abnormal brain biochemistry as a potential factor. Among these is the study of recidivism among criminals found guilty of impulsive homicide and arson. In this subset of violent offenders, a combination of hypoglycemia, low levels of serotonin, and alcoholism were among factors that effectively identify repeat offenders at the time of first sentencing[38,39].

Table I Element levels in hair of violent offenders: *Ratio of Violent/Control*

Element	Pihl and Ervin[5]	Marlow *et al.*[6]	Gottschalk *et al. #1*[8]	Gottschalk *et al. #2*[8]	Gottschalk *et al. #3*[8]	Schauss *et al.*[7]	Schrauzer *et al.*[13]
Lead	2.31***	2.38*	1.66	NS	NS	Sig.**	NS
Cadmium	1.50***	2.06*	0.68	NS	NS	Sig.**	NS
Manganese	1.04	1.50	7.33***	3.39***	2.15**	0.78	2.28**
Chromium	NS	1.14	NS	NS	NS	NS	NS
Sodium	1.33	1.41	NS	NS	NS	NS	NS
Mercury	NS	1.41*	NS	NS	NS	NS	NS
Silicon	NS	1.26*	NS	NS	NS	NS	NS
Copper	0.95	2.07*	NS	NS	NS	Sig.**	NS
Cobalt	0.91	1.39	NS	NS	NS	NS	NS
Lithium	NS	0.58*	NS	NS	NS	NS	0.28***
Calcium	0.87	NS	NS	NS	NS	NS	1.14
Magnesium	0.73	NS	NS	NS	NS	NS	0.64
Potassium	1.33	NS	NS	NS	NS	NS	NS
Iron	0.95	NS	NS	NS	NS	NS	NS
Zinc	0.97	NS	NS	NS	NS	NS	NS

*$p < 0.05$, **$p < 0.01$, ***$p < 0.0005$, NS = Not Significant.

Note: Comparable data on learning disabled children reveals significantly higher levels of lead (ratio 5.75*), cadmium (ratio 1.59*), maganese (ratio 1.43*), chromium (ratio 2.78*) and sodium (ratio 1.78*), and lower levels of cobalt (ratio 0.70*) and lithium (ratio 0.55*). *Source:* Bryce-Smith D. "Lead Induced Disorder of Mentation in Children." *Nutrition and Health.* 1983; 1:179–194.

Table II Factors predicting juvenile delinquency, males in Philadelphia biosocial study (n=487)

Independent variables	Ages	Number of juvenile offenses, ages 7–17	Seriousness of junvenile offenses, ages 7–17
Pregnancy and delivery conditions	Birth	NS	NS
Mother's education	Birth	NS	NS
Father's education	Birth	NS	NS
Family income	Birth	NS	NS
Time father unemployed	Birth	0.179***	0.115**
Hand preference	1	NS	NS
Stanford-Binet	4	NS	NS
Hand preference	4	NS	NS
Eye preference	4	NS	0.059**
Foot preference	4	NS	NS
Neurological abnormalities	7	NS	NS
Abnormal movements	7	NS	NS
Abnormal vision	7	NS	NS
Lead Intoxication	7	0.149***	0.143**
Anemia	7	NS	0.044*
Intellectual status	7	NS	NS
Speech	7	0.045*	NS
Foster parents	7	NS	NS
Father absence	Birth–7	NS	NS
Household moves	Birth–7	0.092*	0.112
Persons supported	7	NS	0.09**
Family income	7	NS	NS
r^2		0.157	0.128

*p<0 .05, **p<0.01, ***p<0.0005, NS=Not Significant. *Source:* Denno D.W.Gender, Crime, and the Criminal Law Defenses. *Journal of Criminal Law & Criminology.* 1994; 85:80–180 (Table II).

IV.
NEUROTOXICITY, LOSS OF IMPULSE CONTROL AND VIOLENCE

To be plausible, the neurotoxicity hypothesis requires the identification of precise biochemical mechanisms underlying correlations between individual concentrations of toxic metals and violent behavior. Correlational or prospective data do not in themselves explain how or why individuals who have absorbed a toxic chemical might be more likely to engage in violent behavior or crime than others. Empirical evidence that neurotoxins have functional effects on brain structure and behavior, undermining impulse control and triggering violence, is therefore of great importance. Such effects, however, are different for each toxin.

Section A.
Lead Toxicity, Behavior, and Violence

Behavioral and cognitive deficits caused by lead, noted in antiquity by Hippocrates and two centuries ago by Benjamin Franklin, have been the subject of widespread scientific analysis[40–44]. Such subtle effects have often been ignored in the light of more obvious health defects produced by very large doses of lead poisoning, such as those from industrial pollution or peeling paint in aging buildings. Less attention has been paid to the effects of lead upon the brain, despite strong evidence that lead absorption lowers IQ

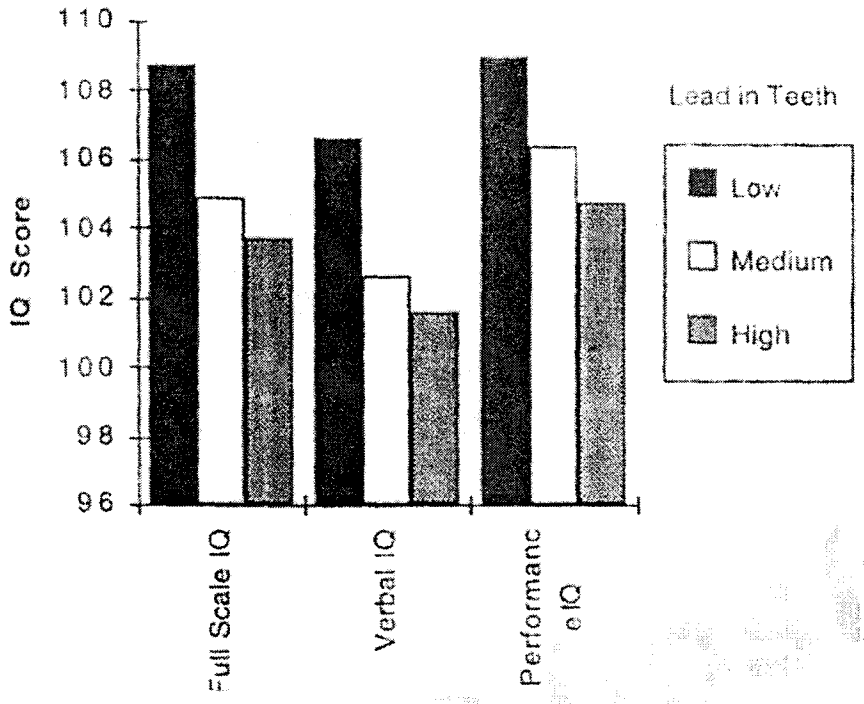


Figure 4 Effects of lead absorption on IQ. *Source:* D.Bryce-Smith, "Environments chemical influences on behaviour and mentation," John Jeyes Lecture—1984, *Chemical Society Review,* **15,** 93–123 (1985).

(Fig. 4) and otherwise disturbs cognitive development and social behavior. Because exposure of infants to lead has effects lasting through puberty and beyond, the long-term developmental damage from lead can interact with the short term behavioral consequences of neurotoxic metals.

Although legislation prohibiting lead in gasoline additives and paint reduced lead levels in blood by 78% between 1976–1978 and 1988–1991[45], toxic release of lead and lead compounds remains a serious problem due to aging water systems and industrial pollution[46]. Exposure to subclinical doses of this neurotoxic metal can be a major hazard for four principal reasons: (1) children absorb up to 50% of lead they ingest, compared to 8% for adults;[47] (2) prolonged exposure to even very low doses of lead can cause neuronal damage during early development, resulting in lasting cognitive and behavioral deficits;[7,48] (3) current lead levels have direct effects on neurotransmitter function, influencing cognition and reducing impulse control [2,17,22–26,33], and (4) highest levels of lead uptake are reported for the demographic groups most likely to commit violent crimes[49,50]. Because the consequences of exposure depend on lifestyle factors such as alcoholic consumption, smoking, and diet, moreover, averages for a population will tend to mask the risks arising from combinations of co-factors[51].

Prolonged exposure of low levels of lead can influence dopaminergic, cholinergic, and glutamatergic neurotransmitter functioning, producing learning deficits that include impairment of passive avoidance learning—i.e., the capacity to be deterred by future punishment[5,13]. Attention deficit disorder (ADD) and attention deficit hyperactivity disorder (ADHD), conditions frequently associated with juvenile delinquency, have also been traced to prolonged exposure to lead[5,13,15,52,53]. Because lead interferes with

the detoxification functions of glial cells[54], moreover, it can enhance the effects of other toxic substances. Apart from its effects on such health concerns as hypertension[55], therefore, it should not be surprising that lead uptake at age seven is predictive of both juvenile and adult crime (Table II)[8].

Successful medical treatment of learning and behavioral deficits due to early exposure to lead provides even more convincing evidence of the effects of subclinical toxicity: "the involvement of lead as a cause of some types of mental retardation is clearly demonstrated by the observation made by a number of different workers that reducing the burden of lead in the child with chelating agents, usually penicillamine, brings about a substantial IQ increase (typically of 7 IQ points) in about two children out of three. Similar detoxification has been applied to children exhibiting hyperactive behaviour problems"[56]. Particularly given the contributions of attention deficit disorder and school failure to violence, it should hardly be surprising that head hair analyses of criminals often show abnormally high levels of lead (Table I).

Section B.
Manganese Toxicity and Violent Behavior

Manganese is a cation whose valence can change among several states (Mn^{2+}, Mn^{3+}, Mn^{4+}); whatever its toxicity elsewhere in the body, within the brain manganese can lower the levels of essential neurotransmitters[57]. Once present in abnormal levels, manganese can therefore produce subtle behavioral effects in the absence of clinical signs of disease. Because other toxic metals may have similar effects, disturbances in behavioral inhibition could follow from interactions between heavy metals rather from amounts of manganese as an isolated factor. Of particular relevance, for example, is the finding that occupational exposure to lead also has the effect of significantly increasing blood levels of manganese[58].

Manganese has been associated with subclinical behavioral disturbances due to its effects on catecholamine function[59–62]. Although industrial manganism can be traced to direct exposure to large quantities of manganese dioxide or manganese nitrate,[63–65] chronic exposure to low levels of manganese is probably more relevant to loss of impulse control and outbursts of violent behavior, especially under stress. Exposure to manganese lowers levels of serotonin in the brain, while paradoxically increasing serotonin levels in blood and body tissue (Fig. 2)[66]; manganese also degrades dopamine and reduces levels of essential minerals in brain cells[67–69]. While the mechanism is not clearly understood, some of these effects may be due to altered levels of monoamine oxidase (MAO A)[29,32,38]. However produced, low levels of serotonin in the brain are associated with mood disturbances, poor impulse control, and increases in aggressive behavior—effects that have increasingly been treated with Prozac and other psychotropic medications which enhance serotoninergic function[70,71].

Vitamin and mineral deficiencies play a central role in lead and manganese uptake. In laboratory studies, for example, mere environmental exposure to manganese does not lead to toxicity, whereas animals with deficits in calcium intake show significant manganese retention[72]. Other aspects of diet also influence the absorption of manganese. In particular, laboratory animals raised on infant formula have cellular retention of manganese that is five times greater than controls raised on human mother's milk (Fig. 5)[73]. Full-term human infants raised on infant formula had manganese retention of 2.8 μg/kg, over five times the level (0.43 μg/kg) in similar infants who were breast fed[74]. The combination of calcium insufficiency andmanganese toxicity could therefore be described to the general public as "reverse Prozac." Similar interactions between diet, lifestyle, and toxic uptake have been reported for lead (see Section IIIA above).

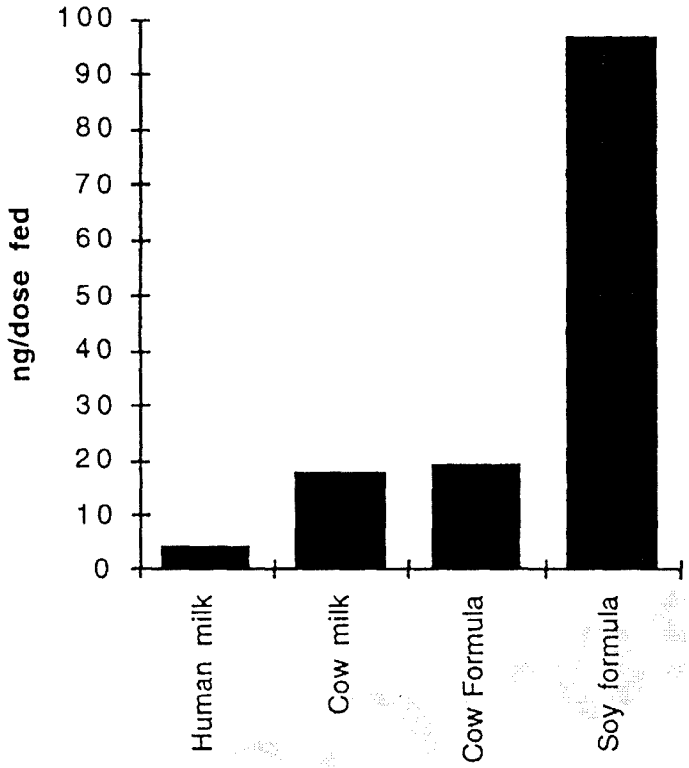


Figure 5 Manganese retention from milks and formulas. Source: Carl L.Keen, Janet G.Bell and Bo Lönnerdal, "The effect of age on manganese uptake and retention from milk and infant formulas in Rats," *J. Nutrition,* **116,** 395–402 (1986). *Note:* although the *rate* of absorbing manganese in brain tissue is actually higher in human mother's milk (1.4% of dose) than either cow milk formula (0.9% of dose) or soy formula (1.0% of dose), the total *amount* of manganese in mother's milk (0.01mg/ml), is 4 times lower than in cow milk (0.04 mg/1), 5 times lower than cow milk formula (0. 05mg/ml), and 30 times lower than soy formula (0.30). "As the concentration of manganese in most infant formulas exceeds that found in human milk, it is evident that the *amount* absorbed and retained from cow milk and the infant formulas can far exceed that from human milk." (p. 400).

Section C.
Other Neurotoxic Metals and Brain Biochemistry

Because a number of metals may disturb normal brain function[75–77], there are other, often unsuspected correlations in addition to those already noted for lead and manganese. Analysis of individuals with learning disabilities or records of criminal violence sometimes reveal imbalances in heavy metals, some of which—such as chromium, cadmium and sodium—seem to be associated with violent behavior, while other metals such as lithium and cobalt seem to reduce aggressive or inappropriate impulses (Table II)[78]. Dietary deficiencies in calcium and vitamin D may also play the key role in the uptake of these elements and minerals.

Conversely lithium lowers the levels of manganese and other toxic metals in the brain—and is lower in hair samples of violent offenders than controls (Table II)[31,34]; at the population level, moreover, homicide levels in Texas were found to be inversely related to levels of lithium in the water supply[26]. Not

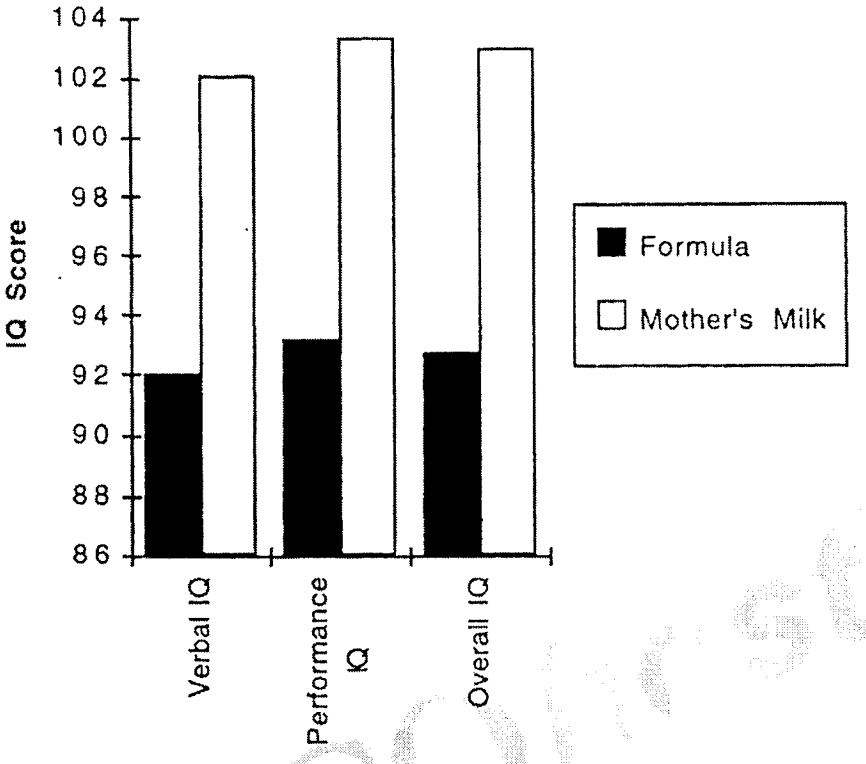


Figure 6 Breast milk and subsequent IQ (age 7.5–8 years). *Source:* A.Lucas, R.Morley, T.J.Cole, G.Lister, and C.Leeson-Payne, "Breast milk and subsequent intelligence quotient in children born preterm," *Lancet,* **339,** 261-264 (1992). *Note:* Preterm infants babies under 1850 g at birth divided into mothers who chose formula (n=90) and those who chose to nurse with their own milk (n=210). Of latter group in above chart, 17 were unable to provide milk; although included in the nursing group, their infants had IQ scores at age 7.5–8 comparable to infants raised on formula (overall IQ=94.8, compared to 92.8 for formula and 103.7 for successful breast-fed infants). Controlling for social class, mother's education, female sex, and days of ventilation, mother's milk had a net contribution of 8.3 points to higher IQ.

surprisingly, lithium has been used successfully to prevent violence in mental patients as well as prison inmates[79].

Other interactions between social behavior, brain biochemistry and violence are also important. As many observers have noted, alcohol and drug use is highly correlated with loss of impulse control and with violence[80]. For some offenders, this may be associated with hypoglycemia and low levels of serotonin[40]. The culture of juvenile gangs provides another factor, creating needs for alcohol and drugs as a condition of belonging to a meaningful peer group[81].

Evidence for an interaction between diet, neurotoxicity, and violence might also be derived from long-term studies of policies that have provided nutritional supplements to poor and disadvantaged populations. Programs like AFDC or WIC that have the potential to improve the diet and parenting-skills of poor, unwed mothers, may be especially important because they can reduce prenatal and infant deficiencies in essential vitamins and minerals. Use of infant formula instead of mother's milk, which as noted leads to increased uptake of manganese, has been significantly associated with lower IQ scores for premature infants (Fig. 6) [82]; similar though somewhat smaller differences were found for full-term infants[83]. More direct measures

of manganese toxicity have also been linked to learning disabilities[84]. Many studies confirm that higher rates of school completion and employment as well as lower rates of criminal violence are among the multiple benefits flowing from concerted early intervention programs that include dietary improvement[85].

Section D.
Alcoholism, Brain Biochemistry and Synergy

Ethanol in alcoholic beverages is a toxin that has serious developmental and functional effects[86]. In addition, ethanol greatly increases the deleterious effects of other toxic metals. In laboratory animals, the effects of a toxic element (cadmium) were "significantly altered" when combined with ethanol, especially because "animals exposed to ethanol absorb much more cadmium than their unexposed counter-parts"[87]. In humans, ethanol interacts with the physiological effects of lead on blood pressure and other physiological processes[88,89] as well as with manganese[90]. As a result, the combination of alcohol consumption and poor diets, often found in marginal young males, puts them at particular risk. Given the interaction between alcohol and various imbalances in brain biochemistry, there should be little surprise that consumption of alcohol has often been associated with a high proportion of violent crimes[3,4,91,92]; consistent with individual data, the geographical distribution of alcoholism resembles that of violent crime (cf. Maps 1 and 4). But not everyone who consumes alcoholic beverages becomes violent. That multiplicative interaction— or synergy —between neurotoxic elements and ethanol could be an important part of the explanation is supported by the county data on rates of violent crime (see Fig. 1 and Section VI below).

Although heroin, cocaine, and other psychoactive drugs have often been implicated in violent crime, somewhat less is known about the precise causal effects of the brain biochemistry involved. In any event, drug users often also consume alcohol, making a differential assessment of behavioral effects difficult. In some cases, moreover, the role of hard drugs has been associated with conflict over the "turf" associated with illegal markets rather than the disinhibiting effects of the drug itself[93]. Because we have not located reliable geographical statistics for drug use comparable to those for alcohol, data analysis will focus on the latter as a general measure of the role of substance abuse in violence.

Anthropologists have also noticed a correlation between violence and hypoglycemia, the tendency for lower than average uptake of glucose[94]. Associated with abnormally high levels of insulin, this condition is frequently associated with alcoholism, though it can be independent of it[34].

V.
ENVIRONMENTAL PATHWAYS OF TOXIC ELEMENTS

In exploring the neurotoxicity hypothesis, it is not sufficient to show that abnormal brain chemistry could be a contributory "risk factor" for violent behavior. From an epidemiological perspective, it is also necessary to show that environmental pathways have the effect of transmitting toxic elements to individuals in quantities associated with violent behavior. Among the multiple pathways of exposure to lead, manganese, and other neurotoxic metals, some have now been studied in sufficient detail to demonstrate the linkage between environmental sources and individual absorption (especially with regard to lead and manganese).

Despite the belief that direct exposure leaded gasoline and house-paint have been the primary pathways for lead absorption, other sources now seem equally if not more important[95]. Perhaps the most comprehensive data concern residues of lead in the soil along highly travelled automobile corridors in urban areas. Although legislation prohibiting lead in gasoline additives and paint reduced lead levels in blood by 78% between 1976–1978 and 1988–1991[96], toxic elements can remain in soils for prolonged periods, especially

in the absence of extensive forest cover facilitating absorption in plant material[97]. Lead-contaminated soils have been demonstrated to cluster in urban neighborhoods along heavily traveled urban roadways in Baltimore[98], Minneapolis[99–102], and New Orleans[103,104]; these studies show, moreover, that the contact of children's hands with contaminated soils in play-grounds and backyards is directly responsible for their absorption of lead[105,106].

Industrial plants are a second source of pollution with toxic metals. In a survey of all towns in Massachusetts, controlling for other variables, individual measures of lead uptake were correlated with the presence of old industrial factories, suggesting that both long-lasting residues and current releases of neurotoxic metals in industry can be effectively transmitted to children. In this study, the average age of housing stock—associated with the presence of peeling lead paint—was also correlated with high lead levels among children[107].

Like lead, manganese is another toxic metal often used in industrial processing. In addition, public water supplies may also be sources of these neurotoxic metals, since aging cast iron pipes contain both lead and manganese that can leach into residues. Early in this century, cast iron was the most typical material for pipes and conduits; #20 Grey cast iron normally contains 0.4–0.6% manganese along with 2.25% silicone, 3. 5% carbon, 0.4% Phosphorous, and 0.1% sulfur[108]. Aging or rusting pipes could therefore leach toxins like lead and manganese into water supplies, particularly in the decaying inner cities now inhabited by the poor. For example, the superintendent of the Edgartown, Massachusetts water department recently described renovations in the town's water system: "When we dismantled the standpipe, we found two and a half feet *(sic)* of a substance with the consistency of pudding"[109]. Other sources of toxicity occur in water systems within buildings. Even in newer, multi-story construction, water supplies in the upper floors may be particularly high in lead: in a recently constructed science building at Dartmouth College, where the first three floors had lead levels around the EPA water standard of 15mg/dL, the fourth floor faucet had levels between 100 and 200mg/dL[110].

A further source of manganese toxicity arises from diet and lifestyle. Because laboratory studies indicate that cellular uptake of manganese from formula based on cows' milk is five times greater than from mother's milk and 20 times greater for soy formula (Fig. 5)[111]. The practice of bottle-feeding infants greatly increases the infant's exposure to toxicity[112]. Bottle-feeding will have even worse effects if water polluted with manganese or lead is used to mix the formula. For those with diets deficient in calcium, zinc, vitamin D, and other necessary vitamins and minerals, rates of bottle feeding are likely to be higher and its effects on the infant more damaging.

Consumption of alcohol and psychoactive drugs also delivers toxic substances to the brain that tend to disinhibit behavior. These effects can have highly complex interactions with the uptake of neurotoxic elements like lead and manganese. On the one hand, alcohol and cocaine provide short-term bursts of neurotransmitters like serotonin and dopamine, whose activity is reduced by lead and manganese; hence the desire to use alcohol and drugs may be increased by neurotoxic exposure as well as by social stress. On the other hand, drugs like cocaine are often processed with chemicals like potassium permanganate and, over the long run, tend to reduce endogenous production of catecholamines. Hence environment-lifestyle interactions may generate positive feedback loops in which the presence of neurotoxins trigger synergistic reactions of the sort noted previously.

As the foregoing suggests, all individuals are not equally vulnerable to ecological pathways of toxins. The human body, and especially the brain, has evolved defense mechanisms against neurotoxic metals. For example, under normal circumstances, virtually all ingested manganese is excreted, which is hardly surprising insofar as manganese is naturally the 12th most abundant element in soil. Because neuronal

uptake occurs for individuals with vitamin and mineral deficiencies, however, factors in the social environment have the effect of facilitating the transmission of neurotoxicity.

America's poor often suffer from dietary deficiencies, including deficits in calcium and vitamin D (the rate limiting factor for cellular binding of calcium). For example, the 1976–1980 National Health and Nutrition Examination Survey, based on a national probability sample, found that black teenage males consume on average only two-thirds as much calcium (887mg/day) as do whites (1332mg/day); a follow-up survey revealed that calcium intake among Hispanics (1195 for Mexican Americans, 1255 for Puerto Ricans, and 1185 for Cubans) was also below the white average.[113] The calcium needs of pregnant or breast feeding women or adolescents (1200mg/day) are higher than average—and even higher for pregnant teenagers (1600 mg/day). This presents a specific problem for pregnant minority women: for example, non-Hispanic black women between the ages of 18 and 39 get only 467 mg/day of calcium as compared to 642 mg/day among white women of the same age[114]. Given the increased uptake of neurotoxic metals associated with calcium deficiencies in laboratory studies, calcium deficits among the poor may have particularly deleterious effects during infant development and childhood.

Finally, poor mothers typically do not breast-feed their infants. "By 1986–87, 73 percent of infants born to mothers with more than 12 years of education were breastfed compared with 49 percent of infants born to mothers with 12 years of education and 31 percent of mothers with less than 12 years of education"; this difference interacts with ethnicity since "white infants were nearly three times as likely to be breastfed as were black infants"[115]. The effects of manganese toxicity associated with infant formula are thus greatest for the poor, for ethnic minorities, and for those with little education.

Factors in the social environment are likely to interact with environmental pollution from industrial sources and the urban infrastructure. While it may be difficult to isolate how much toxicity is transmitted by each of the interacting pathways, an ecological approach to behavioral deregulation and criminal violence seems reasonable. If the neurotoxicity hypothesis is correct, it should be directly confirmed by geographic differences in rates of crime that are associated with the known pathways of toxic elements. Using multivariate analysis, moreover, it should be possible to assess how various socio-economic and demographic factors compare with neurotoxicity as risk factors for violence.

VI.
THE ECOLOGY OF VIOLENCE: ENVIRONMENTAL POLLUTION AND RATES OF VIOLENT CRIME

County-level reports of toxic releases and other ecological factors can be used to study violence, much as they have been proposed as a way to measure the impact of ambient pollution on human health[116]. To test the hypothesis that neurotoxicity is among the co-factors related to violent crime, we used ecological data on the distribution of environmental pollutants and alcoholism to predict the rates of criminal violence. The Environmental Protection Agency's Toxic Release Inventory (TRI) for lead and manganese was correlated with 1991 FBI crime reports from all counties (n=3141) in the US; counties with no reported incidence of either violent or property crimes were dropped from the analysis (n=242, of which 231 are in the continental US shown on Map 1).

Although county-level reports in toxic release inventory measure current levels of pollution, they are likely to indicate the existence of industrial plants that have left lasting residues of toxicity in soil and on buildings. The report of environmental releases of lead or manganese can thus be viewed as a proxy variable for industrial activities resulting in environmental pollution with any neurotoxic substance. To be sure, our data concern current residence rather than an individual's childhood environment, and hence do not

provide prospective measures like those cited above. Given barriers to geographic mobility into neighborhoods of different social classes, however, it is not implausible that the county of current residence is similar to environments during infancy, especially for poor and minority populations; if so, for many of those most at risk, present exposure to toxic metals should also be a rough measure of environments during development.

The distribution of manganese and lead pollution is highly skewed: over 80% of counties have no reported release of either manganese and its compounds, or lead and its compounds. The EPA's recorded releases of these toxic metals are not predicted by other demographic or socio-economic variables normally associated with crime: a multiple regression model using 19 factors (population density, ethnic composition, ethnic poverty, unemployment, income, police per capita, public sewers, public water supplies, rate of infant death, age of housing stock, welfare, education, alcoholism, and other toxic release) predicts less than 5% of the variance of reported levels of lead (r^2=0.031) or manganese (r^2=0.045).

Because environmental releases of these toxic metals have non-linear effects on behavior, EPA reports were dichotomized as the presence or absence of reported lead or manganese toxicity. To assess whether toxicity might exacerbate the effects of alcoholism, rates of death from all causes associated with alcoholism were dichotomized at the national mean (47.2 per 100,000). The three way ANOVA (Fig. 1) shows that all three variables (death from alcoholism as well as lead and manganese pollution) are highly significant predictors of rates of violent crime (lead: p<0.0001, t ratio=9.66, $F_{1,2898}$=93.22; manganese, p<0.0001, t ratio= 11.32, $F_{1,2898}$=128.25; alcoholism, p<0.0001, t ratio=11.99, $F_{1,2898}$=143.64), with significant two-way interactions between manganese and alcoholism (p<0.0001, t ratio=6.86, $F_{1,2898}$=47.04), lead and alcoholism (p<0.0027, t ratio=3.00, $F_{1,2898}$=9.01), lead and manganese (p<0.0001, t ratio=3.91, $F_{1,2898}$=15.30), and a significant three-way interaction between alcoholism, lead, and manganese (p<0.0169, t ratio=2.39, $F_{1,2898}$=5.72).

In counties with no reported releases of lead or manganese and below average deaths from alcoholism, rates of violent crime are below average (216 per 100,000 compared to the national mean of 298). In contrast, the 52 counties with toxic releases from both metals and above average rates of alcoholism have almost four times as much violent crime (920 per 100,000). Although epidemiologists are increasingly aware of the importance of such synergistic interactions, most conventional models of violent crime have looked at individual variables rather than complex effects of ecological and lifestyle factors on brain chemistry and behavior[2,3,9].

The correlations between environmental pollution and crime interact significantly with population density. For example, in counties with no reported pollution from either lead or manganese and below average alcoholism, population density makes little difference in crime rates (which are 170 per 100,000 in the 677 counties with below average density, and 265 in the 565 counties with above average density). In contrast, where lead and manganese pollution are accompanied by high rates of alcoholism, densely populated counties (n=48) report 970 violent crimes per 100,000, or three times the national average, while the four low density counties with similar neurotoxicity have only 138 violent crimes per 100,000. Since the stress involved in urban living has neurochemical correlates that exacerbate the effects of neurotoxicity [28,40,45], toxic pollution and rates of alcoholism seem to be hitherto unsuspected risk factors that contribute to geographic differences in violence.

Multivariate Analysis To ascertain how these effects might relate to other risk factors associated with violence, a multiple regression was computed, controlling for 10 socio-economic and demographic variables in addition to toxic variables and their interactions (Table IIIa). Consistent with a multi-causal model, twelve of these variables had significant effects, predicting over a third of the variance (adjusted $r2$=0.369). Controlling for other factors (population density, median grade of education, police per capita,

Table III Multiple regression model for 50 states

Factors	IIIa: All Violent Crimes	IIIb: Aggravated Assault	IIIc: Property Crimes	IIId: Welfare Recip/Cap	IIIe: Median Grade Completed
Population Density (log)	82.42**** (20.24)	38.77**** (12.26)	490.91**** (22.39)	0.00574**** (24.56)	−0.08903**** (−8.23)
Per-capita Income	−0.007**** (−2.74)	−0.0059*** (−2.81)	NS	NS	0.00017**** (25.23)
Unemployment	NS	2.27* (1.71)	16.05* (1.74)	0.00021** (2.10)	−0.03665**** (−8.06)
%Black Poverty	40.06** (2.33)	27.62** (2.07)	NS	NS	−0.13322*** (−2.88)
%Hispanic Poverty	62.11*** (2.79)	41.23** (2.39)	541.5**** (4.52)	NS	NS
Police/Capita	153423**** (16.56)	89318**** (12.42)	821385**** (16.47)	4.97007**** (9.34)	NS
Infant Death Rate	(1.813)*** (2.78)	1.173** (2.32)	NS	0.000065* (1.74)	NS
%Housing pre-1950	−526.75**** (−13.43)	−447.34**** (−14.69)	−3722**** (−17.63)	0.007379*** (3.28)	0.93843**** (9.02)
Public Water/Cap	225.34**** (4.07)	197.4**** (4.59)	2618**** (8.78)	NS	1.00734**** (6.82)
Median Grade Completed	24.68*** (3.50)	16.82*** (3.07)	586.7**** (15.46)	0.001109** (2.74)	N/A
Lead Present/ Absent	40.80**** (4.67)	18.49* (2.73)	134.5*** (2.86)	0.003491**** (6.95)	NS
Manganese Present/Abs.	58.71**** (6.68)	39.26**** (5.75)	155.3**** (3.28)	0.004482**** (8.88)	0.05672** (2.40)
Alcohol Death Rate	101.62**** (11.55)	62.46**** (9.14)	188.4*** (3.97)	0.001186** (2.35)	−0.10258**** (−4.35)
Alcohol * Lead	21.48** (2.54)	NS	NS	0.001626*** (3.34)	0.05316** (2.34)
Alcohol * Manganese	55.40**** (6.54)	36.79**** (5.59)	117.6** (2.58)	0.001578*** (3.24)	0.04051* (1.78)
Lead * Manganese	34.89**** (4.11)	15.57** (2.36)	NS	0.003126**** (6.41)	NS
Alcohol * Lead * Manganese	19.21** (2.27)	NS	NS	0.001764*** (3.63)	NS
adj r^2	0.369	0.259	0.467	0.369	0.415
F	97.45	58.60	145.12	97.17	125.02
DF	17,2783	17,2783	17,2783	17,2783	16,2784
p	0.0000	0.0000	0.0000	0.0000	0.0000

****p<0.0001, ***p<0.005, **p<0.005, *p<0.10.

Note: Cell entries are unstandardized coefficients (*t* ratio in parentheses).

percent blacks and percent hispanics in poverty, per capita income, unemployment rate, percent households on public water supplies, rates of infant death, and percent housing built before 1950), lead and

manganese toxicity and rates of death from alcoholism are significant predictors of violent crime, with significant two-way and three-way interactions among the three measures of toxicity.

Some findings from the multiple regression model are contrary to conventional wisdom. Unemployment is not associated with overall rates of violence, and the higher the median grade of school completed, the *higher* the crime rate (suggesting that educational failure *per se* is not a source of violent behavior). Conversely, sociological theories of crime do not predict that the percent of households on public water supplies could be a significant risk factor of crime. Since aging pipes in public water systems often contain lead and manganese[117–120], this finding provides additional support for the neurotoxicity hypothesis. Finally, age of housing stock—often assumed to measure infants' exposure to pica from leaded paint—is *negatively* associated with violence, suggesting that water supplies and environmental pollution are more likely than paint to serve as pathways for lead[121].

To insure that the findings were not an artifact of inaccurate statistics, these results were checked by using the same factors to predict rates of aggravated assault (a category of violent crime not confounded by reporting errors attributed to sexual assault, yet more frequent and hence more reliable in rural areas than homicide), property crime (to measure non-violent deviance), per capita welfare loads (an index of social disintegration), and median grade of education completed (Table IIIb-e). Findings are consistent with the hypothesis that environmental pollution is a risk factor in violence, in the disintegration of stable families, and—to a lesser extent—in educational failure.

These results are also consistent with the evidence that exposure to environmental pollutants does not effect all people in the same way. Since deficiencies in calcium, zinc or other vitamins and minerals are associated with uptake of lead and manganese, population groups with poor diets and low levels of breast feeding should have greater vulnerability to toxic chemicals[5,12,14,17,38,57,122–126]. Comparison of the regression equations predicting violent and property crime (Table IIIa-c) is consistent with this explanation: while population density, police per capita, and age of housing have similar effects on both types of criminal behavior, lower per capita income and black poverty—which presumably are associated with higher risks of dietary insufficiency—are not correlated with property crime as they are for total violent crime or aggravated assault.

As would be predicted by the neurotoxicity hypothesis, percent of households on public water supplies—a probable pathway of toxicity—is correlated with increased crime of all kinds, but not with percent of population on welfare. Moreover, neither ethnic poverty nor alcoholism is significantly associated with percent of population on welfare, suggesting that these factors associated with crime do not have a direct impact on familial and social disintegration.

Structural Co-Variate Model Although multiple regression and analysis of variance are widely used by social scientists to assess the effects of diverse variables on phenomena such as crime, these statistical methods have been criticized. The statistics reported in Table IIIa-e are mathematically based on the debatable assumption that the predictive factors are "independent;" in multiple regression models where the predictive variables are interdependent, results are highly unreliable. In a survey of geographic analyses of homicide rates, Land, McCall, and Cohen demonstrated the importance of this objection by showing how apparently parallel statistical studies implicated quite diverse risk factors to explain variations between cities, metropolitan areas, or states[127].

To circumvent this analytical problem, Land, McCall and Cohen introduced a more complex methodology. First, they computed a principal components analysis of the set of predictive variables, thereby identifying underlying dimensions or factors that might account for interrelated variables (such as *per capita* income and rates of unemployment); each geographical entity was then given a "factor score" for each of these underlying factors or dimensions—and it is these scores that are used to predict crime rates.

Table IV Principal components analysis and structural covariate model

IVa Principal components analysis			IVb Structural co-variate model		
	21 variables		Estimate	*t*-ratio	Std. error probability
Wealth			0.0155	5.85	0.00266 < 0.0001
PC income	0.77				
Med. grade	0.62				
White poor	−0.89				
Unemployment	−0.51				
(Hispanic poor)*	(−0.43)				
Eigenvalue		3.6			
% Variance		17.30%			
Old housing/White			−567.19	−21.11	26.8969 < 0.0001
Housing pre 1950	0.82				
White %	0.62				
Hispanic %	−0.51				
Eigenvalue		2.8			
% Variance		13.30%			
Public goods			496.648	16.7	29.738 < 0.0001
Sewers/Capita	0.89				
Public H2o/Cap	0.83				
Police/Cap	0.74				
Eigenvalue		2.3.			
% Variance		11.20%			
Rural			−48.071	−18.56	2.59 < 0.0001
Pop. Dens./Sq. Mi.	−0.61				
AFDC $/Capita	−0.77				
AFDC #/Capita	−0.76				
Eigenvalue		1.6			
% Variance		7.50%			
Alcoholism			−3.62	−9.32	0.388 < 0.0001
Deaths--Alcoholism	−0.83				
All alcohol related	−0.76				
Eigenvalue		1.4			
% Variance		6.60%			
Black population			150.183	5.53	27.173 < 0.0001
% Black	0.73				
Black poverty	0.51				
(Infant death)*	0.46				
Eigenvalue		110.00%			
% Variance		5.10%			
Industrial pollution**			−31.698	−5.34	5.936 < 0.0001
Lead TRI	0.77				
Manganese TRI	0.71				
Eigenvalue		1			
% Variance		5.00%			
Intercept in co-variate model			6.461	0.16	40.787 ns
Adjusted r-square in co-variate model					0.411

* Variables poorly predicted by factor structure (commonality <0.25)

** Entered in structural co-variate model as a dichotomous variable counties either with or without pollution from either lead or manganese. (Negative sign means less crime where no pollution.) A parallel model with continuous variation on this co-variate showed little difference, with seven factors all highly significant and explaining comparable variance (adjusted r-square=0.407).

Using this method, called a "structural co-variate model," Land, McCall, and Cohen found stable findings for homicide rates in cities and states, with three "structural indexes/covariables—population structure, resource deprivation/affluence, and percentage of the male population divorced" showing "statistically significant relationships to the homicide rate...across all time periods and levels of analysis"[128]. In this study, "population structure" represents the average population density and population size of the geographic units; "resource deprivation/affluence" reflects income, poverty, economic inequality, blacks as a percentage of population and percentage children under 18 not living with both parents. Many of the variables used in our study were not included by Land, McCall, and Cohen, and their results also showed that cities in the "south" had, controlling for all factors, consistently higher homicide rates than elsewhere in the US.

To insure that our findings are not vitiated by the statistical questions raised by Land, McCall, and Cohen, our set of 21 predictor variables was factor analyzed (Table IVa). Given the larger variability introduced by studying counties, it was not surprising that we found seven distinct factors: wealth and inequality, comparable to their "resource deprivation/affluence" (reflecting per capita income, median grade of education completed, percent white poverty, and—more weakly—unemployment); urban-rural, comparable to their "population structure" (reflecting population density, welfare load per capita, and welfare expenditures per capita); old housing-white population (reflecting % housing pre-1950, % white, and negatively % hispanic); public goods (reflecting sewers per capita, households on public water supplies per capita and police per capita), rates of alcoholism (reflecting two measures of alcohol disease and death), black population (reflecting % black population and black poverty), and industrial pollution (reflecting the toxic release inventories for lead and manganese). It is worth stressing that this approach confirms that the geographical distribution of our measures of neurotoxicity are independent of conventional socio-economic and demographic variation.

Following the procedure suggested by McCall, Land, and Cohen, we then computed the multiple regression using this structural co-variate model (Table IVb). As will be seen, the principal components or factors reflecting environmental pollution and alcoholism—the direct measures of neurotoxicity—remain highly significant. Moreover, this approach probably understates the effects of environmental pollution. On the one hand, the toxicity of soils in the highly traveled automobile corridors studied by Mielke and his collaborators is a characteristic of urban, high density counties (and hence is including in the rural-urban co-variate); on the other, aging public water supplies are included in the public goods co-variate. That these pathways of toxicity are more important than aging house-paint is confirmed by the negative sign for the co-variant for % white and housing pre-1950.

VII.
CONCLUSIONS AND POLICY IMPLICATIONS

In these findings, urbanism, ethnicity and toxicity emerge as important correlates of violent crime. If so, the traditional approaches to crime in the United States need to be reconsidered from an ecological point of view. The environment seems to increase the probability that some individuals lose capacities for impulse control, leading to increased rates of crime and reinforcing patterns of school dropout, family disintegration, and unemployment.

Apart from studies cited above, the principal geographic explanation of violence in the literature is Nisbett and Cohen's analysis of the Southern "culture of honor"[129,130]. Although this hypothesis is not inconsistent with the present findings, the data presented by Nisbett and Cohen focus on differential rates of violence in small cities and towns as well as broad regional patterns of culture. Moreover, their attribution

of an upland herding "culture of violence" to "Scotch-Irish" immigration could well introduce co-variation with rates of alcoholism, a trait that may have a genetic component[131]. In any event, regional cultural attitudes, state legislation, and differences in homicide rates between cities under 200,000 in "hills and high plains" and in "moist plains"[117] do not explain many of the county-level variations we have observed and analyzed.

The neurotoxicity hypothesis may also help to explain historical trends that have otherwise puzzled many experts. Evidence that rates of violent crime in New York and other cities have fallen about 10% (*New York Times,* 23 July 1995, Section 4, pp. 1, 4; *New York Times,* 13 August 1995, pp. 1, 18) may be linked to changes in diet and neurotoxicity. Lead consumption in the U.S. which was over 0.201×10^6 mt per year in 1977–1978, declined to 0.119×10^6 mt per year in 1982, and to pre-1943 levels of 0.028×10^6mt per year in 1988–1989, with corresponding declines in deposition in air and even in forest soils[97]. At the same time, calcium consumption, which had risen to historically high levels during World War II, declined from 1945 through the 1960s before beginning a modest increase. Because there is a lag of 15–20 years between the effects of lead uptake on neonatal or infant development and the onset of criminal behavior in juvenile or early adult years, increased rates of violence between 1955 and 1975 may have reflected the combination of greater emissions of lead and reduced calcium intake. Further research is therefore needed to assess whether the recent declines in violence might be in part related to the maturation of the first generations of youth to benefit from the prohibition of leaded gasoline and other initiatives to reduce environmental pollution.

Cross-national data are also be relevant. It is sometimes claimed, for example, that the neurotoxicity hypothesis is contradicted by low rates of violence in Canada, where MMT—a manganese based gasoline additive—has been used since leaded gasoline became illegal. In fact, the statistics may actually suggest the contrary. Although homicide and robbery rates in Canada are lower than in the United States (perhaps in part due to gun control and other public policies), when all four types of crime classified as violent by the FBI (homicide, aggravated assault, sexual assault, and robbery) are combined, rates are actually higher in Canada than the U.S. (Fig. 7). This effect is clearly related to rates of aggravated assault, the most frequent kind of interpersonal violence in both countries. More important for present purposes, since the introduction of MMT, the rate of all violent crimes has increased at a faster rate in Canada than in the U.S. Since factors such as guns and ethnicity that are often associated with high crime rates in the U.S. are absent in Canada, the increasing difference between the two countries contradicts the assertion that the Canadian experience with MMT is evidence against a relationship between manganese and violent behavior.

If confirmed, the neurotoxicity hypothesis would have obvious implications for public policy. Crime prevention and improved educational performance may be greatly enhanced by parent-training in proper diet. Studies indicate that breast feeding increases IQ in both preterm, low birthweight infants[132] and full-term infants[133]. Vitamin supplements may be useful in improving some aspects of school performance and cognitive development, at least for those with poor diets[5,134–138]. Pre-school programs, which in some cases have been found to reduce rates of crime[139,140], need to be evaluated in terms of their effects on diet rather than by conventional educational assessments; indeed, the short-term nature of the gains in IQ reported in follow-up studies of Head Start may reflect the essential role of nutritional supplementation in remedial education[141,142]. If releases of neurotoxic metals are associated with rates of crime, as this study suggests, reducing environmental pollution takes on higher priority. In our criminal justice system, because existing means of predicting recidivism are little better than chance, the assay of toxic metals in head hair might provide an inexpensive marker for potential violence in probation decisions[35]. With adequate identification of the precise biochemical imbalances associated with violent crime, vitamin and mineral normalization could contribute to improved rehabilitation[13,35,40].

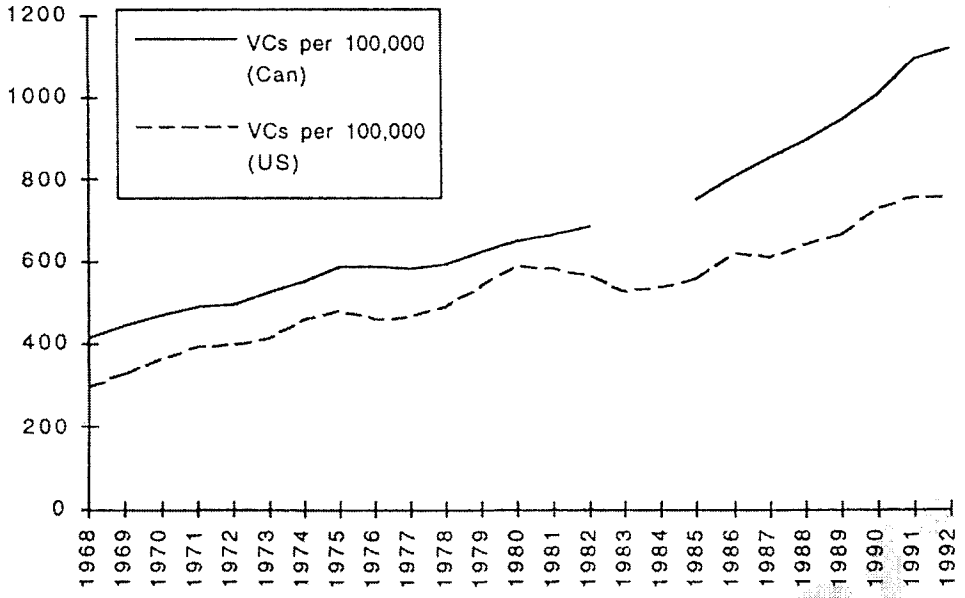


Figure 7 Violent crime in Canada and the United States, 1968–1992 (Annual rate for homicide, aggravated assault, sexual assault, and robbery). *Source:* US Crime statistics: *Statistical abstract of the US:;* Canadian Crime Statistics, *Statistics Canada: Canadian Cime Statistic Annual.* No data reported for Canada, 1983–1984. *Note:* While rates of homicide are consistently lower in Canada than in the U.S. (presumably as a result of such factors as gun control legislation and urbanization), the phenomena of violent crime are most closely correlated with and better measured by aggravated assault, whose rates closely parallel the overall rates shown here. Cf. F.Zimring, "Firearms, violence, and public policy," *Scientific American* **265,** 48–57 (1991); A.A.Leenaars, D.Lester, "Effects of gun control on homicide in Canada," *Psychological Reports,* **75,** 81–82 (1994). While cross-cultural differences in reporting categories and criteria might explain the higher rates in Canada, they would not explain the differing trends since 1985.

One issue of immediate importance is posed by MMT, the manganese-based gasoline additive used in Canada. The introduction of MMT into the United States has long been prohibited under an EPA finding that it violated the Clean Air Act[143]. The recent reversal of this decision by a Federal Appellate Court (*Wall Street Journal,* October 25, 1995) increases the importance of the present findings[144]. While the manufacturers have claimed that "no adverse health effects have been observed" from the use of MMT in Canada, traditional studies use standards of occupational exposure that do not consider interactions between manganese, lead, alcoholism, and dietary insufficiencies, and focus on health rather than on behavioral outcomes such as learning disabilities and crime that are sensitive to subclinical levels of toxicity[145]. In animal models, ingestion of MMT alters catecholamine function[146]. And in one study comparing Canadian blue collar workers and garage mechanics, higher atmospheric exposure to manganese was associated with higher levels of the metal in garage mechanics head hair[147]. This study is, however, far from conclusive: the contribution of atmospheric manganese was estimated as 1% of total absorption, and the same investigators found these garage mechanics to be exposed to significantly higher levels of manganese in their household tap water[148]. At most, therefore, it can be said that more study is needed, including consideration of occupational effects of MMT vapor and skin contact.

Given the extraordinary level of violence that persists in urban America and the failure of traditional policies to meet it, further confirmation of the hypothesized relationships between dietary deficiencies,

neurotoxic uptake, and violence is urgently needed. Of particular importance will be direct studies correlating environmental exposure, diet, neurotoxic uptake, and behavior. Equally important will be experimental studies of the effects of vitamin and mineral normalization for violent offenders. Although changes in the criminal justice system should not be implemented without further evidence, the neurotoxicity hypothesis may require important changes in the way Americans think about crime and its prevention.

APPENDIX ON METHODOLOGY

Our analysis is based on a dataset constructed from multiple sources; all data are by county, a reasonable geographic unit for measuring the effects of industrial pollution and geographic variability in crime rates[114]. Counties without data for FBI crime reports were dropped from the analysis. Variables, with definition and source, are as follows:

Violent Crime. Data for 1991: the sum of (1) Murder and nonnegligent manslaughter ("the willful (nonnegligent) killing of one human being by another"); (2) Forcible rape ("carnal knowledge of a female forcibly and against her will. Assaults or attempts to commit rape by force are also included; however, statutory rape (without force) and other sex offenses are excluded"); (3) Robbery ("taking or attempting to take anything of value from the care, custody, or control of a person or persons by force or threat of force or violence and/or by putting the victim in fear"); and (4) Aggravated assault ("unlawful attack by one person upon another for the purpose of inflicting severe or aggravated bodily injury"). Source: Federal Bureau of Investigation[149].

Property Crime. Data for 1991: the sum of (1) burglary ("the unlawful entry of a structure to commit a felony or theft"). (2) larceny-theft ("the unlawful taking, carrying, leading, or riding away of property from the possession or constructive possession of another [including] shoplifting, pocket-picking, purse snatching, thefts from motor vehicles, thefts of motor vehicle parts and accessories, bicycle thefts, etc. in which no use of force, violence or fraud occurs. This crime category does not include embezzlement, 'con' games, forgery, worthless checks, and motor vehicle theft). (3) Motor vehicle theft ("the theft or attempted theft of a motor vehicle"). Source: Federal Bureau of Investigation, Uniform Crime Reporting (UCR) Program[80].

Toxic Release Inventory. Total in pounds per year for 1991, computed separately for lead, lead compounds, manganese, and manganese compounds (sum of midpoint of range for "point air release, land release, chemicals transferred to an off-site facility, chemicals transferred to publicly owned treatment works, underground injection releases, discharges to water"). Source: U.S. Environmental Protection Agency, Toxic Release Inventory, 1987–1993[150].

Population. Total population in county as recorded in the FBI database. Source: Federal Bureau of Investigation, Uniform Crime Reporting (UCR) Program[80].

Population Density. Definition. "Persons per square mile is the average number of inhabitants per square mile of land area." Source: U.S. Bureau of Census, Population Division, July 1, 1992[80].

Houses with Water Supply from a Public System or Private Company. Households served by a common source supplying water to five or more units...supplied by a city, county, water district, water company, etc. or a well which supplies water to five or more housing units[80].

Houses on Public Sewer Systems. Households served by "a public sewer...operated by a government body or by a private organization. Source: U.S. Bureau of the Census, Census of Government, Public Employment"[80].

Housing Built before 1950. Percentage of households living in units constructed before 1950. (Comparable data also analyzed for housing units built before 1939.) Source: U.S. Bureau of the Census, Census of Government, Public Employment[80].

Police Officers. Police protection employees of local government with arrest power, as "full time equivalent." Source: U.S. Bureau of the Census, Census of Government, Public Employment[80].

School Drop-outs. Educational Attainment—Persons 25 years and over completing less than 9th grade. Source: U.S. Bureau of the Census, Census of Population and Housing[80].

High School Drop-outs. Educational Attainment—Persons 25 years and over completing 9th to 12th grade, no diploma. Source: U.S. Bureau of the Census, Census of Population and Housing[80].

Educational Achievement—BA or Higher Education. "Persons 25 or older— Bachelor's, graduate, or professional degree." Source: U.S. Bureau of the Census, Census of Population and Housing[80].

Per capita Money Income. Aggregate money income ("wages and salaries, non-farm self-employment; farm self-employment; Social Security; public assistance; and all other regularly received income such as veteran's payments, pensions, unemployment compensation, and alimony") divided by total resident population. Source: U.S. Bureau of the Census, Census of Population and Housing[80].

Unemployment Rate. Number of unemployed as a percent of the civilian labor force ("all civilians 16 year old and over classified as employed or unemployed"). Source: U.S. Bureau of Labor Statistics[80].

White Population. U.S. Census Bureau classification based on guidelines in Federal Statistical Directive #15, OMB ("Includes persons who indicated their race as 'White' or reported entries such as Canadian, German, Italian, Lebanese, Near Easterner, Arab or Polish"). Source: U.S. Bureau of the Census, Population Division, July 1, 1992[80].

Black population. U.S. Census Bureau classification ("Includes persons who indicated their race as 'Black or Negro' or reported entries such as African American, Afro-American, Black Puerto Rican, Jamaican, Nigerian, West Indian, or Haitian"). Source: U.S. Bureau of the Census, Population Division, July 1, 1992[80].

Hispanic Population. U.S. Census Bureau classification ("Includes persons who classified themselves in one of the specific Hispanic origin categories listed on the [self-identification] questionnaire—'Mexican,' 'Puerto Rican,' or 'Cuban'—as well as those who indicated that they were of 'other Spanish/Hispanic' origin.... Persons of Hispanic origin may be of any race"). Source: U.S. Bureau of the Census, Population Division, July 1, 1992[80].

Persons below Poverty Level (by racial or ethnic category). U.S. Census Bureau definition of poverty, computed separately for whites, blacks, and hispanics ("poverty thresholds are computed on a national basis only... Poverty status is derived on a sample basis"). Source: U.S. Bureau of the Census, Census of Population and Housing[80].

Aid to Families with Dependent Children (AFDC)—Number Served. Total recipients, month of February ("Federal grants to help defray State costs of providing financial assistance to needy children who are under age 18 (or under age 21 and attending school); living in the home of a parent or other relative; and deprived of parental support or care because of the death, continued absence from the home, or physical or mental incapacity of a parent—or if a State elects, the unemployment of a father").Source: U.S. Social Security Administration[80].

Aid to Families with Dependent Children (AFDC)—Payments. Total payments, in dollars, for February. Source: U.S. Social Security Administration[80].

Women, Children and Youth Program (ACYF)—Number Served. Total number of children enrolled in WIC program[151].

Woman, Children and Youth Program (ACYF)—Payments. Average monthly expenditures, ACYF program[82].

Infant Deaths per 1000 Live Births. "infant death rates represent the number of deaths of infants under 1 year of age per 1,000 live births. They exclude fetal deaths." Source: U.S. National Center for Health Statistics, Vital Statistics of the United States[80].

All Alcohol Related Causes of Death. "Any death certificate with explicit mention of alcohol, diseases often caused by alcohol or accident often caused by alcohol. If, for example, 42% of auto deaths involve alcohol, 42% of traffic deaths are included." Source: U.S. Department of Health and Human Services, National Institute on Alcohol Abuse and Alcoholism[152].

Causes of Death with Explicit Mention of Alcohol. "Alcoholic psychoses, alcohol dependent syndrome; nondependent abuse of alcohol; alcoholic polyneuropathy, cariomyopathy, gastritis, fatty liver, hepatitis (acute), cirrhosis of the liver, liver damage unspecified; excess blood alcohol content, accidental poisoning by ethyl alcohol, not elsewhere specified." Source: U.S. Department of Health and Human Services, National Institute on Alcohol Abuse and Alcoholism[80].

Parameters for analyses of variance (ANOVA) and multiple regression analyses are noted in Tables and Figures. Earlier work with samples of counties in eight states[153] and sixteen states[154] showed similar patterns of correlation between toxic metals, alcoholism, and violent crime. While more complex statistical manipulations are possible (e.g. path analysis), inferential statistics must in any event be supplemented by direct observation of individual diet and neurotoxic uptake in polluted versus non-polluted environments. Such tests of the neurotoxicity hypothesis are urgently needed to assess causal factors in learning disabilities and health risks as well as probabilities of engaging in violent behavior[155].

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114. *Nutrition Monitoring in the U.S., Chartbook I: Selected Findings from the National Nutrition Monitoring and Related Research Program* (B.Ervin and D.Reed, eds.) (Public Health Service, Hyattsville, MD, 1993) Figure 57. These ethnic differences have persisted despite recent increases in calcium intake. Hence the 1988–91 National Heath and Nutrition Examination Survey (K.Alaimo, M.A.McDowell, R.R.Briefel, A.M.Bischof, C.R.Caughman, C.M.Loria and C.L.Johnson, *Dietary Intake of Vitamins, Minerals, and Fiber of Persons Ages 2 Months and Over in the United States* U.S. Department of Health and Human Services, Center for Disease Control, Washington, 1994.) revealed that black males 16–19 years old had only 78% of the calcium intake (1076mg/day) of comparable white males (1373mg/day). Since there is usually a delay of at least 15 years between infant environment and the onset of criminal careers, it would be valuable to explore the possibility that changes in diet and environmental toxicity might be related to the recently reported declines in rates of violent crime reported in some cities (see note 10 above).
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Fast facts on the economic benefits of reducing violent crime

Reducing current rates of murder, rape, assault, and robbery would produce a wide range of savings and other benefits for Americans as individuals, property owners, and taxpayers. In 2010 these four main types of violent crime cost Americans more than $42 billion in direct costs, including the associated costs of police, courts and correctional institutions, out-of-pocket-medical expenses borne by victims, and lost earnings by both victims and perpetrators who are arrested and convicted. These costs totaled $137 per American in 2010, the last year for which complete data are available.

Violent crimes also inflict other, more intangible costs, including the pain and suffering of victims, a reduced quality of life for everyone, and lower investment levels and property values. While these intangible costs are difficult to measure with precision, analysts agree that they greatly exceed the direct costs. Here are the fast facts enumerating the economic benefits of reducing violent crime:

- Across five cities with the necessary data for our analysis, we found that a 10 percent reduction in homicides should lead to a 0.83 percent increase in housing values the following year, and a 25 percent reduction in homicides should produce a 2.1 percent increase in housing prices over the next year. Applying these results to all residential housing in the metropolitan areas of our sample of eight American cities, we find that:

- A 10 percent reduction in homicides should increase the value of residential real estate by $4.4 billion in the Boston metropolitan area, $3.2 billion in Philadelphia, $2.9 billion in Seattle, $2.4 billion in Dallas, $2.2 billion in the Chicago area, $800 million in Milwaukee, and $600 million in Jacksonville. At current property tax rates, the increase in housing values associated with a 10 percent reduction in homicides would substantially expand revenues from property taxes in all eight cities.

- A 25 percent reduction in homicides should be followed by increases in metropolitan area housing values totaling $11 billion in the Boston area, $8 billion in Philadelphia, $7.25 billion in Seattle, $6 billion in Dallas, $5.5 billion in the Chicago area, $2 billion in Milwaukee, and $1.5 billion in Jacksonville. At their current property tax rates, these increases in housing values would substantially expand the revenues from property taxes in all eight cities.

- The other, direct annual costs of violent crime in the eight cities total $3.7 billion per year, ranging from $89 million per year in Seattle and $198 million in Boston to $752 million per year in Houston, $736 million in Philadelphia, and $1.1 billion in Chicago. These direct costs average $320 per person per year across the eight cities, ranging from $144 in Seattle and $246 in Jacksonville to about $390 in Milwaukee and Chicago, and $472 in Philadelphia.

- The value of the more intangible pain and suffering borne by the victims of these violent crimes totals some $13.9 billion per year across the eight cities, ranging from $216 million per year in Seattle and $734 million in Boston, to nearly $3 billion per year in Philadelphia and $4.2 billion in Chicago. These annual, intangible costs average more than $1,200 per person across the eight cities, ranging from $350 per person per year in Seattle and nearly $980 in Jacksonville, to $1,486 per person per year in both Chicago and Milwaukee, and more than $1,900 in Philadelphia.

Successful efforts to reduce violent crime can generate significant savings for municipal budgets and large benefits for residents, apart from increases in their housing values. On the next page we list the economic savings the eight cities in our study would reap by reducing violent crime by 10 percent and by 25 percent.

Boston

- A 10 percent reduction could save $5 million per year, reduce direct costs to victims by more than $7 million per year, and avert more than $73 million in annual intangible costs to victims—reducing the total government costs by an average of $145 per resident per year.
- A 25 percent reduction could save more than $12 million per year, reduce the direct costs borne by victims by some $18 million per year, and avert more than $180 million in annual intangible costs—reducing total government costs by the equivalent of more than $360 per resident per year.
- This 25 percent savings could enable a mix of boosting city spending on housing and community development by up to 14.4 percent or reducing property taxes by up to 0.8 percent.

Chicago

- A 10 percent reduction could save $24 million per year, reduce the direct costs to victims by nearly $43 million per year, and avert more than $420 million in annual, intangible costs to victims—reducing total government costs by an average of $187 per resident per year.
- A 25 percent reduction could save $59 million per year, reduce the direct costs to victims by more than $107 million per year, and avert more than $1 billion in annual intangible costs—reducing total government costs by the equivalent of nearly $470 per resident per year.
- This 25 percent savings could enable a mix of reducing all local taxes by up to 2.5 percent or increasing city spending on community services by up to 66 percent.

Dallas

- A 10 percent reduction could save $7 million per year, reduce the direct costs to victims by nearly $15 million per year, and avert more than $140 million in annual, intangible costs to victims—reducing total government costs by an average of $138 per resident per year.
- A 25 percent reduction could save $19 million per year, reduce the direct costs to victims by more than $36 million per year, and avert more than $360 million in annual intangible costs—reducing total government costs by the equivalent of more than $450 per resident per year.
- This 25 percent savings could enable a mix of reducing property taxes by up to 4.3 percent or increasing the parks and recreation budget by up to 29 percent.

Houston

- A 10 percent reduction could save $17 million per year, reduce the direct costs to victims by nearly $27 million per year, and avert more than $265 million in annual, intangible costs to victims—reducing total government costs by an average of nearly $150 per resident per year.
- A 25 percent reduction could save more than $43 million per year, reduce direct costs to victims by $67 million per year, and avert more than $660 million in annual intangible costs—reducing total government costs by the equivalent of more than $370 per resident per year.
- This 25 percent savings could fund a mix of doubling city spending on health and human services or cutting property taxes by up to 5 percent.

Continued on next page

Jacksonville

- A 10 percent reduction could save $4 million per year, reduce the direct costs to victims by nearly $8 million per year, and avert more than $80 million in annual, intangible costs to victims—reducing total government costs by an average of $122 per resident per year.
- A 25 percent reduction could save nearly $12 million per year, reduce the direct costs to victims by nearly $20 million per year, and avert more than $200 million in annual intangible costs—reducing total government costs by the equivalent of more than $305 per resident per year.
- This 25 percent savings could enable a mix of cutting its property taxes by up to 2 percent or increasing local spending on economic development by up to 26 percent.

Milwaukee

- A 10 percent reduction could save nearly $5 million per year, reduce the direct costs to victims by more than $9 million per year, and avert some $90 million in annual, intangible costs to victims—reducing total government costs by an average of nearly $190 per resident per year.
- A 25 percent reduction could save more than $12 million per year, reduce the direct costs to victims by some $23 million per year, and avert $225 million in annual intangible costs—reducing total government costs in Milwaukee by the equivalent of nearly $470 per resident per year.
- This 25 percent savings could enable a mix of cutting property taxes by up to 4 percent or increasing spending on housing and community development by up to 71 percent.

Philadelphia

- A 10 percent reduction could save more than $17 million per year, reduce the direct costs to victims by nearly $30 million per year, and avert nearly $300 million in annual, intangible costs to victims—reducing total government costs by an average of nearly $240 per resident per year.
- A 25 percent reduction could save more than $43 million per year, reduce the direct costs to victims by nearly $75 million per year, and avert some $742 million in annual intangible costs—reducing total government costs by the equivalent of more than $595 per resident per year.
- This 25 percent savings could enable a mix of cutting local property taxes by up to 11 percent or doubling spending on homeless and housing assistance.

Seattle

- A 10 percent reduction could save more than $2 million per year, reduce the direct costs to victims by more than $2 million per year, and avert nearly $22 million in annual, intangible costs to victims—reducing total government costs by an average of nearly $50 per resident per year.
- A 25 percent reduction would save the city budget $6 million per year, reduce the direct costs to victims by more than $5 million per year, and avert some $54 million in annual intangible costs—reducing total costs in Seattle by the equivalent of $123 per resident per year.
- This 25 percent savings could enable a mix of cutting property taxes by up to 2.4 percent and increasing city spending on neighborhoods and development by up to 5.4 percent.

Contents

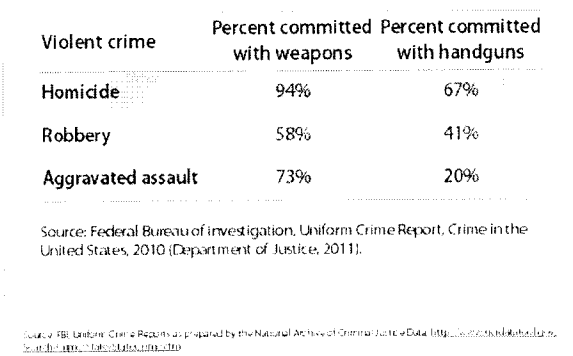
Introduction and summary

Violent crimes are costly. Murders, rapes, assaults, and robberies impose concrete economic costs on the victims who survive as well as the families of those who lose their lives, in the loss of earnings and their physical and emotional tolls. Violent crimes also impose large costs on communities through lower property values, higher insurance premiums, and reduced investment in high-crime areas. In addition, violent crimes impose significant costs on taxpayers, who bear the financial burden of maintaining the police personnel and operations, courts, jails, and prisons directed toward these crimes and their perpetrators.

Fortunately, the incidence of violent crimes in the United States has fallen sharply over the last 20 years. From 1960 to 1990 the rates of these crimes rose sharply as did their attendant costs. Over that period murder rates nearly doubled, rates of rape and robbery increased fourfold, and the rate of assault quintupled. Since the early 1990s, however, rates of most violent crimes have been cut nearly in half. (see Figure 1)

FIGURE 1
The good news: Falling crime rates

Violent crime in the United States, offenses per 100,000 population, 1960-2010



Violent crime	Percent committed with weapons	Percent committed with handguns
Homicide	94%	67%
Robbery	58%	41%
Aggravated assault	73%	20%

Source: Federal Bureau of Investigation, Uniform Crime Report, Crime in the United States, 2010 (Department of Justice, 2011).

Source: FBI Uniform Crime Reports as prepared by the National Archive of Criminal Justice Data. http://www.icpsr.umich.edu/NACJD/guides/ucr.html#Publications

Yet rates of most violent crimes in the United States remain high compared to the 1950s and 1960s and to other advanced societies today. The U.S. murder rate, for example, has fallen to a 50-year low, but that rate is still nearly three times the level in Canada and more than four times the level in the United Kingdom.[1] Among all of the world's developed countries, the United States today, on a per capita basis, ranks second in murders, fourth in rapes, and sixth in robberies.

The Bureau of Justice Statistics reports that the majority of all violent crimes involve the use of weapons, and in two-thirds of all homicides and 41 percent of all robberies, the weapon is a handgun. (see Table 1)

TABLE 1
Most violent crimes involve weapons

Use of weapons and guns in violent crimes, 2010

Violent crime	Percent committed with weapons	Percent committed with handguns
Homicide	94%	67%
Robbery	58%	41%
Aggravated assault	73%	20%

Source: Federal Bureau of Investigation, *Uniform Crime Report, Crime in the United States, 2010* (Department of Justice, 2011).

Moreover, from 2005 to 2010 the nationwide incidence of homicides declined by 12.5 percent, the number of robberies decreased by nearly 9 percent, and the number of aggravated assaults declined by 7 percent. The share of crimes committed with guns in all three categories, however, remained constant.

By most measures, violent crime continues to impose significant costs on Americans and their communities. The costs borne by the American public for this level of criminal activity are significant. Medical care for assault victims, for example, costs an estimated $4.3 billion per year.[2] We spend $74 billion per year on incarcerating 2.3 million criminals, including some 930,000 violent criminals.[3]

Moreover, the costs of the pain and suffering borne by the victims of violent crimes is several times greater than the more direct costs of those crimes. As a result, successful efforts to reduce violent crime can produce substantial economic benefits for individuals, communities, and taxpayers.

This report presents the findings and conclusions of a yearlong project to examine and analyze the costs of violent crimes in a sample of eight major American cities and estimate the savings and other benefits that would accompany significant

reductions in those crimes. This analysis draws on data pinpointing the incidence and location of murders, rapes, assaults, and robberies. The data were provided by the police departments of Boston, Chicago, Dallas, Houston, Jacksonville, Milwaukee, Philadelphia, and Seattle.

We examined a broad range of both direct and intangible costs associated with those violent crimes based on their incidence in each of the eight cities in 2010. The direct costs reported here are those borne by the residents and city governments of the eight cities, although additional costs are also borne by state and federal governments and the taxpayers who finance them. Finally, we calculated the benefits to those residents associated with substantial reductions in violent crime, including the impact on residential home values and a variety of savings to the city governments.

In today's tight fiscal and economic environment, the mayors and city councils of every city—along with state and the federal governments—are searching for ways to reduce their spending and expand their revenues. The common challenge is to achieve sustainable fiscal conditions without hobbling government's ability to provide the vital goods and services that most Americans expect, all without burdening businesses and families with onerous new taxes. This analysis provides another way available to many American municipalities: Secure budget savings, higher revenues, and personal income and wealth gains by reducing violent crime rates.

To calculate the extent of those savings and benefits, we analyze a broad range of direct costs associated with the violent crime in the eight cities sampled here. These direct costs start with local spending on policing, prosecuting, and incarcerating the perpetrators of those crimes. These costs also encompass out-of-pocket medical expenses borne by surviving victims of violent crime as well as the income those victims must forgo as a result of the crimes. These costs also include the lost incomes that would otherwise be earned by the perpetrators of violent crimes had they not been apprehended—as distasteful as it is to calculate the foregone income of rapists or armed robbers who are arrested, convicted, and incarcerated. These direct, annual costs range from $90 million per year in Seattle to around $200 million per year in Boston, Jacksonville, and Milwaukee, to more than $700 million in Philadelphia and nearly $1.1 billion for Chicago.

This report also examines certain intangible costs associated with violent crime, including the pain and suffering of the surviving victims of violent crime and the costs to the families of murder victims. Across the eight cities examined here, the total annual costs of violent crimes, including these intangible costs as well as the more direct ones, range from more than $300 million per year in Seattle to more than $900 million in Boston, to some $3.7 billion per year in Philadelphia and $5.3 billion for Chicago.

Based on this analysis we also estimate the budgetary savings that each of the eight cities should expect to achieve if their rates of violent crime declined by either 10 percent or 25 percent. These savings include lower expenditures on law enforcement and the justice system, as well as the additional revenues that each city could expect to collect from applying local taxes to the income earned by those who otherwise would have been victims or perpetrators of those crimes.

All told, the estimated savings for municipal budgets from a 25 percent reduction in violent crime range from $6 million per year in Seattle to $12 million per year in Boston and Milwaukee, to $42 million per year in Philadelphia and $59 million for Chicago. We also estimate the value of other benefits associated with reduced rates of violent crime, including lower out-of-pocket medical costs for those who otherwise would have been victims as well as their averted pain and suffering.

The largest economic benefits, however, arise from the impact of lower rates of violent crime on the housing values in the cities sampled here. To estimate this effect, we use data covering several years on the incidence of violent crimes by zip code in each city and changes in housing values in the same zip codes over the same period. Five of the eight cities were able to provide data by zip code covering at least six years. Our analysis of those data found that a reduced incidence of murders in a particular zip code is followed by a predictable and significant increase in housing values in the same zip code in the next year.

On average, a reduction in a given year of one homicide in a zip code causes a 1.5 percent increase in housing values in that same zip code the following year. We applied these findings to available data on the value of the housing stock in the metropolitan areas of all eight cities. The estimated increases in the value of the housing stock for the eight cities and their immediate metropolitan areas, following a 10 percent reduction in homicides, range from $600 million in Jacksonville and the surrounding area to $800 million in the Milwaukee area, to $3.2 billion in Philadelphia and the surrounding suburbs, and $4.4 billion in the Boston area. Unfortunately, inconsistent reporting of other types of violent crime—rapes, assaults, and robberies—preclude a reliable analysis of the impact on housing values of changes in the incidence of those crimes.

Methods to reduce violent crime

A full analysis of the ways communities reduce crime is beyond the scope of this report, but it is important to note that many strategies for reducing violent crime entail budgetary costs as well. We do not attempt to calculate those costs. Nevertheless, readers should be aware that scholars have conducted extensive research to identify which crime control and prevention strategies contributed most to the reductions in violent crimes in recent decades.

Problem-oriented policing is an evidence-based intervention for reducing violence.[4] An evaluation of the "pulling levers" strategy—policing that "focuses criminal justice and social service attention on a small number of chronically offending gang members"—found a 43 percent decline in assaultive gun violence events and a 66 percent reduction in gang-related homicides after the intervention.[5]

An economic analysis of underground gun markets in Chicago suggests that intervening in networks of black-market gun brokers may also offer promise in reducing illegal access to guns.[6] Police stings of gun dealers engaged in illegal gun sales were associated with a subsequent reduction in the supply of new guns to criminals in some but not all cities.[7]

Many empirical studies, for example, have examined which strategies have been most cost effective.[8] A 1997 meta-analysis commissioned by the U.S. Department of Justice identified a range of practices that have proved successful with various kinds of offenders. Family therapy and parent-training efforts have been quite effective for at-risk pre-adolescents while vocational training has worked well for certain groups of older, male ex-offenders. Additional police patrols also reduced the incidence of serious offenses in high-crime hotspots.[9]

Similarly, a 1998 RAND Corporation study analyzed the cost effectiveness of several approaches in California. It found that $1 million expended on graduation incentives reduced the number of yearly serious crimes by 258.[10] By contrast, $1 million for parent training led to 157 fewer crimes, $1 million on supervising delinquents led to 72 fewer serious crimes, and $1 million devoted to three-strike laws led to just 60 fewer serious offenses.[11]

As a crime-prevention strategy, longer and more certain prison sentences seem to reduce property crimes, but not violent crimes.[12] Yet incarceration does make it much easier to build up DNA databases and recent research has found that criminals included in DNA databases are less likely to commit new crimes as well as more likely to be apprehended when they do so.[13] According to one analysis a 50 percent increase in the size of the average DNA database could produce a 13.5 percent reduction in murders, a 27.2 percent reduction in rapes, and a 12.2 percent reduction in aggravated results.

Many social and economic policies designed for other purposes may also reduce the incidence of serious crimes. Programs to encourage young people to remain in school, for example, have proved to be one of the most cost-effective crime-reduction strategies.[14] Similarly, community-development efforts to increase business investment in at-risk neighborhoods have also been shown to reduce crime rates.[15] Finally, demographics play a role. Male youth are the population group most prone to commit serious crimes so as their share of the population grew with the initial baby boom and then fell with the subsequent baby bust, crime rates also increased and then subsided.[16]

The consequences of falling crime rates on real estate values, city budgets, and local residents

Housing values

First, we will examine and analyze how a reduction in the incidence of violent crimes in a city affects the value of housing in that city. To undertake this analysis we collected data on the incidence of violent crimes by geographic area for eight cities:

- Boston
- Chicago
- Dallas
- Houston
- Jacksonville
- Milwaukee
- Philadelphia
- Seattle

These cities provided data covering varying periods of time from 2000 on, ranging from 6 to 11 years. Police departments in five of the cities were able to provide complete data by zip code covering a sufficiently long period for statistical analysis—Chicago, Houston, Jacksonville, Milwaukee, and Philadelphia.

We also collected data on the value of residential real estate in each city by zip code for the same time periods covered by the crime data. We used those two datasets to analyze the consequences of the actual changes in violent crime rates on actual housing values, using so called dynamic panel regression models in conjunction with Granger causality testing. (See Appendix A, Table A-2, on page 54 for a detailed description of this methodology.) This analysis shows that, on average, a reduction in homicides of one incident in a zip code during a given year causes a 1.52 percent increase in home prices in that zip code the following year.

The impact on real estate values of lower rates of other violent crimes, however, is more difficult to establish. Our analysis did not establish a statistically significant relationship. In fact, it found that falling rates of violent crimes other than homicides were followed by falling housing prices, though not to a statistically significant degree. We discount these results, however, because they likely reflect persistent problems with these types of crime data. Unlike murders, other violent crimes are sharply underreported. According to the Justice Department National Crime Victimization Surveys, on average only 45 percent of rapes and 59 percent of assaults are reported to police.

Moreover, the rates at which those crimes are reported may shift from year to year in no stable relationship to the rates at which those crimes actually occur. Since this analysis depends on changes in crime rates in small geographic areas (zip codes), these random variations preclude reliable results.

The results from homicides are reliable, however, and the economic consequences of reduced rates of homicides can be very large. Here, we were able to roughly estimate the metropolitan statistical area-wide impact for seven of the eight sample cities (all but Houston). We estimate, for example, that a 10 percent reduction in homicides could increase the value of the housing stock of the Boston area by $4.4 billion in the following year. (see Table 5 on page 17)

Similarly, a 10 percent drop in homicides could increase the value of the housing stock by $3.2 billion in the Philadelphia metropolitan area, by $2.9 billion in the Seattle area, by $2.4 billion in the Dallas area, by $2.2 billion in the Chicago metropolitan area, by $800 million in and around Milwaukee, and by $600 million in the Jacksonville area. A 10 percent reduction in homicides, therefore, should generate large revenue gains from the property taxes applied to those values.

The housing stock data, however, cover metropolitan areas, which in each case encompass city and suburban jurisdictions with different property tax rates. Therefore, we cannot estimate the precise dimensions of these additional property tax revenues for the eight sampled cities.

City budgets and their residents

Next, we analyzed other benefits and savings for individuals, communities, and municipal budgets that come from reductions in violent crimes, using data on violent crimes from all eight cities examined in this report. This analysis begins with an accounting of the direct costs of violent crime:

- The medical costs borne by surviving victims of violent crime
- Municipal spending on police
- Courts and corrections
- The foregone productivity of murder victims, victims of other violent crimes while they recover, and of criminals while they remain in jail or prison

Across the eight cities, these direct costs arising from the four types of violent crimes total nearly $3.7 billion per year. These direct costs are equivalent to an average of $320 per resident per year for the eight cities, ranging from $144 per resident per year in Seattle to $472 per resident per year in Philadelphia. (see Table 2)

TABLE 2

The direct costs of violent crimes

Estimated direct costs of violent crimes by city, 2010 ($ millions)

City	Victims	Justice system	Productivity losses (criminals)	Total	Cost per resident
Boston	$72	$102	$24	$198	$308
Chicago	$426	$547	$132	$1,104	$390
Dallas	$145	$175	$43	$363	$278
Houston	$268	$393	$91	$752	$330
Jacksonville	$79	$100	$24	$202	$246
Milwaukee	$92	$115	$27	$235	$388
Philadelphia	$299	$351	$86	$736	$472
Seattle	$21	$56	$12	$89	$144

Source: Author's calculations, Federal Bureau of Investigation, Uniform Crime Reports, Department of Justice, National Crime Victimization Survey, 2005–2010, Department of Justice, Bureau of Justice Statistics, Criminal Costs, Expenditure, and Employment, and Department of Justice, Bureau of Justice Statistics, National Judicial Reporting Program

Given these costs, reducing the incidence of violent crime should produce substantial benefits. The direct savings for city governments associated with a 10 percent reduction in these four violent crimes would include commensurate reductions in spending on law enforcement, courts and corrections, and additional revenues from taxing the earnings of both would-be victims and would-be perpetrators of crimes that would not occur under this scenario.

A 10 percent decline in violent crime should produce direct savings to the governments of the eight cities ranging from $2 million per year in Seattle to $24 million per year in Chicago. Similarly, a 25 percent reduction in violent crime—half of the nationwide decline seen from 1990 to 2010 for rape, robbery, and assault—would mean annual savings for the eight city governments ranging from $6 million per year in Seattle to $59 million per year in Chicago.

Across all eight cities a 10 percent reduction in violent crime rates would produce combined direct savings of $82 million per year for the eight city governments, while a 25 percent reduction would produce $204 million. (see Table 3)

TABLE 3

Savings from reduced violent crime

Estimated budget costs from violent crime and budget savings from 10 percent and 25 percent reductions in those crimes, by city, 2010 ($ millions)

	Budgetary costs			Annual budget savings from reducing violent crimes	
City	Police, courts and corrections	Tax revenue	Total	10 Percent reduction	25 Percent reduction
Boston	$102	$6.8	$109	$11	$27
Chicago	$547	$18.2	$565	$56	$141
Dallas	$175	$5.6	$180	$18	$45
Houston	$393	$10.8	$404	$40	$101
Jacksonville	$100	$4.1	$104	$10	$26
Milwaukee	$115	$2.9	$118	$12	$30
Philadelphia	$351	$33.3	$384	$38	$96
Seattle	$56	$1.1	$57	$6	$14

Source: U.S. Census Bureau, 2010; author's calculations' Estimates of revenues lost as a result of a relationship between growth in the income of city residents and growth in the city's total tax revenue)

Such reductions in violent crime could release or create new resources for other municipal purposes. A 25 percent reduction would save the city of Boston, for example, sufficient funds and generate sufficient additional revenues to either fund a 1 percent cut in that city's property taxes, increase city spending on health and human services by 6.8 percent, or boost spending on housing and community development by 14.4 percent. Similarly, a 25 percent drop in Houston's violent crime rates would generate sufficient additional resources to double that city's budget for health and human services or fund a 5.1 percent cut in property taxes.

Researchers also have studied and estimated the less tangible, indirect costs arising from violent crime, especially the pain, suffering, and diminished quality of life that surviving victims of violent crime experience as well as effects on the families of murder victims. Unlike the more tangible, direct costs of violent crimes, there are no objective measures for these genuine losses. But scholars have developed a variety of methods to estimate the value of the pain, suffering, and reduced quality of life of people who are raped, violently assaulted, or robbed.

All of these approaches find that these intangible costs exceed the direct costs by an order of three to four. Nationwide, these intangible costs come to an estimated $156 billion per year. For the eight cities examined here, these intangible costs are estimated to total nearly $14 billion per year, ranging from $216 million per year in Seattle to $4.2 billion per year in Chicago.

Therefore, a 10 percent or 25 percent reduction in violent crime should proportionately reduce those indirect, intangible costs. A 25 percent reduction would save potential victims of violent crimes in Milwaukee, for example, pain, suffering, and diminished quality of life valued at $225 million per year, while a similar decline in violent crimes in Dallas would be worth $361 million in intangible benefits for those who otherwise would have been victims of violent crimes. (see Table 4 on next page)

TABLE 4

Direct and indirect costs of violent crimes

Estimated direct and indirect costs of violent crimes, by city, 2010 ($ millions)

	Direct costs				Intangible and total costs	
City	Victim	Justice system	Criminal	Total direct	Intangible	Total direct and intangible
Boston	$72	$102	$24	**$198**	$734	**$932**
Chicago	$426	$547	$132	**$1,104**	$4,206	**$5,310**
Dallas	$145	$175	$43	**$363**	$1,444	**$1,807**
Houston	$268	$393	$91	**$752**	$2,655	**$3,407**
Jacksonville	$78	$100	$24	**$202**	$802	**$1,004**
Milwaukee	$92	$115	$27	**$235**	$900	**$1,135**
Philadelphia	$299	$351	$86	**$736**	$2,970	**$3,705**
Seattle	$21	$56	$12	**$89**	$216	**$305**

Source: FBI, "Uniform Crime Reporting," as prepared by the National Archive of Criminal Justice Data, available here: http://www.icpsr.umich.edu/icpsrweb/NACJD/search/victim-offender/state-offense.jsp; Kathryn E. McCollister, Michael T. French, and Hai Fang, "The Cost of Crime to Society: New Crime-Specific Estimates for Policy and Program Evaluation," Drug and Alcohol Dependence 108 (1-2) (2010): 98–109.

We now turn to more detailed analyses of the consequences of falling violent crime rates on real estate values, city budgets, and local residents.

The impact of lower rates of violent crime on real estate values

The analysis in this section is based on data provided by the police departments of five cities on the incidence of violent crimes by geographic location, and data on residential home sales in those cities by zip code from DataQuick. As we will demonstrate, changes in homicide rates in particular across those five cities had clear and significant effects on the value of homes in the same areas. Homicides obviously exact an incalculable toll on the victims and significant costs on their families. But costs also are borne by everyone who lives nearby through the impact on property prices. Moreover, our analysis found that successful efforts to reduce homicides would generate substantial benefits not only for those who otherwise would be victims but also for the area as a whole.

These findings have important implications for policymakers because the equity that people hold in their homes accounts for much of Americans' wealth. Increases in home values driven by the exogenous factor of a falling homicide rate translate directly into increases in the wealth and financial security of the families who own those homes. Such increases in housing values also can lead to substantially higher local government revenues when property tax assessments catch up with the underlying increase in home values.

Data and methodology

This analysis focused on the five cities of Chicago, Houston, Jacksonville, Milwaukee, and Philadelphia, covering at most the 11 years between 2000 and 2011. The police departments in these cities provided violent crime data by zip code. We obtained median and mean residential property prices by zip code for the same cities tract from DataQuick, a public-records database company. DataQuick compiles real estate data from public sources such as county assessors' offices and county recorders' offices and provides estimates of prices for new, existing, attached, and detached home-sale transactions.[17]

The crime data we collected covered reports of homicides, rapes, robberies, and aggravated assaults, covering all of these crimes and attempted crimes regardless of whether arrests or convictions were ever made. These crime data were not reported in a uniform format across police departments. Each department provided a list of every reported violent crime by some geographic identification over the longest period available from 2000 to 2011.

Philadelphia, Milwaukee, and Jacksonville all provided the point location of each reported crime. We also engaged Spatial Insights, Inc., a geographic information services company, to "reverse geocode" these point locations to zip codes. Houston and Chicago provided street addresses, which we mapped onto zip codes using the "Geocode+Maps," software from GeoLytics, Inc., with a success rate of 99 percent.

Continued on next page

The data from different cities covered varying periods: 11 years (Jacksonville, 2001–2011), 10 years (Chicago, 2001–2010), nine years (Houston, 2000–2008), and six years (Milwaukee, 2005–2010, and Philadelphia, 2004–2009). As expected, the relative prevalence of these crimes is fairly uniform across the cities, with homicides being least common, followed by rapes, and aggravated assaults and robberies occurring much more frequently.

Our empirical method applied the standard "Granger Causality" statistical regression test to identify any causal relationship between changes in these violent crimes and changes in residential property prices.

In addition to the proposition that lower crime rates lead to higher property values, we also consider the possibility that higher residential property prices lead to reductions in violent crimes, and that violent crime rates and property prices are correlated with some other, third factor. We also recognize that there may be significant "multicollinearity" between the different types of violent crimes, so we group the crimes into the two variables of homicides and nonhomicides.

A more detailed description of our methodology and the summary statistics from the regression analysis is provided in Appendix A, Tables A-1 and A-2, on pages 52 and 54.

Results

Our main specification focused on the relationship between changes in violent-crime rates and changes in housing prices by zip code across five cities—Chicago, Houston, Jacksonville, Milwaukee, and Philadelphia. The Granger causality analysis found, first, the wholly unremarkable finding that, by zip code, housing prices in any one year affect housing prices in the following year to a statistically significant degree. (see Table A2 in Appendix A on page 54) In technical terms, changes in median residential property prices in an area in one year have a positive coefficient and are statistically significant in explaining the change in median residential property prices in that area in the following year.

More important, the causality test found that changes in the incidence of murders in a zip code in one year affect or explain, to a statistically significant degree, changes in residential property prices in the following year. Fewer murders in one year, therefore, "Granger cause" higher residential property prices the next year, and higher numbers of murders "Granger cause" lower residential property prices the following year.

More specifically, a reduction in homicides of one in a zip code Granger causes a 1.52 percent increase in home prices in that same zip code the following year. This relationship is symmetrical: Each additional homicide in a zip code, compared to the year before, is associated with a 1.52 percent reduction in home property values in the following year. This large of an effect from changes in homicide levels is not unexpected, given that the average number of homicides per zip code, per year across the cities sampled is only 5.51 per year.

These results are not affected by the underlying facts that through most of this period, murders were generally declining and housing prices were generally rising. The regression analysis takes account of these underlying facts and isolates and analyzes rates of changes in each of the variables across hundreds of observations by zip code and year.

The impact of falling rates of violent crimes other than homicides, however, is much more difficult to determine. Indeed, the regression analysis initially found the *opposite* effect from homicides. Falling rates of nonhomicide were associated with small declines in housing prices, or, since the relationship is symmetrical, rising nonhomicide crime rates were accompanied by slight increases in housing prices. These results, however, were *not* statistically significant.

In fact, these results likely reflect a recurring, underlying problem with nonhomicide crime data. The murder rate is widely considered the most reliable measure of violent crime. This is because virtually all murders are reported to the police. By contrast, a significant share of all rapes, robberies, and assault are not reported. Comparing "deaths from assault" (homicides) as reported by the Center for Disease Control with FBI reports of murder and non-negligent manslaughter, we find that more than 92 percent of all murders are reported to police.[18] But National Crime Victimization Surveys report that, on average, 45 percent of rapes, 59 percent of assaults, and 62 percent of robberies are reported to police. In addition, the rates at which these other violent crimes are reported vary from year to year, so that changes in their reported rates may not reflect changes in their actual incidence.

Therefore, an observed increase in these crimes may reflect shifts in reporting patterns rather than actual changes in crime rates. This effect may be very large in a small geographic area such as a zip code. When the police increase their presence in an area (such as a zip code), reported crimes may rise even when the actual incidence of those crimes is unchanged or even falls. Changes in the numbers of reported robberies, rapes, and aggravated assaults in a zip code may also be associated with increases in urban density when, for example, new businesses arrive and new residences are built in an area. Such a development could result, at once, in higher reported crime rates and higher property values, both reflecting the third variable of accelerated development. Along with many other researchers on the impact of crime, we therefore focus this analysis on homicides.

The technical specifications and results of the Granger causality analysis are provided in Appendix A at page 51.

In addition, Appendix B beginning on page 55 provides the results of six tests of the robustness of our main results. The tests demonstrate that the basic findings on the impact of changes in homicide rates are not affected by alternate specifications. In particular, we tested the sensitivity of our results to an increased number of lags, the inclusion of squared terms, the use of average rather than median property-price data, one-step estimation rather than two-step estimation, the inclusion of differing depths of lags, and an uncollapsed instrument matrix. These results confirm the impact of changes in homicide rates on changes in housing prices in zip code-size areas.

Impact of homicides on the value of the housing stock across a city or metropolitan area

Although our analysis was conducted at the zip-code level, we can use the results to estimate the impact of changes in homicide rates on the value of the overall housing stock of a city or metropolitan area. These estimates assume that the effect of reductions in homicide rates does not vary based on the absolute number of homicides in an area, and that people are as likely to move between cities or metropolitan areas in response to changes in homicide rates in their neighborhoods as they are to move within the same city or metropolitan area.

Using these assumptions we can estimate how much the value of the housing stock in the five cities examined here would be expected to rise in response to specified reductions in the homicide rates in those cities. These estimates should be accurate for the cities examined in this study, since they are all cities with accessible suburbs or nearby metropolitan areas that can provide potential new residents, and therefore increased demand for housing in areas with falling homicide rates.

As noted earlier, by combining the average number of homicides in those cities with our regression results, we find that a 10 percent reduction in homicides corresponds to a 0.83 percent increase in residential property values and prices the following year. A 25 percent reduction in homicides in these cities could push housing prices up by nearly 2.1 percent. This calculation allows us to estimate the overall gain in residential property values that could accompany a 10 percent reduction in homicides at the citywide or metropolitan areawide level. Moreover, we can extend this analysis to cover other cities considered here, so long as the relevant data on housing stock is available.

This analysis draws on total market value estimates for metropolitan areas from the Zillow Real Estate Market Reports of December 2010.[17] Since market value estimates for 2011 are not available, we estimate the effect on total residential property values in 2011 if the cities or their metropolitan areas had reduced homicides by 10 percent in 2010. These are high-end estimates that provide a rough measure of the wealth gains that could accompany a 10 percent reduction in homicide rates. Houston is omitted from this analysis because Zillow does not provide an estimate of total housing-market value in that city's metropolitan area. Therefore, these estimates cover the other seven cities.

This analysis shows that a 10 percent reduction in homicides could increase the value of the residential housing stock by $4.4 billion in the Boston metropolitan area, by $2.4 billion in the Dallas metropolitan area, by $2.2 billion in the Chicago metropolitan area, and by $600 million in the Jacksonville metropolitan area. Similarly, a 10 percent reduction in homicides would boost the total value of all residential housing by $3.2 billion across the Philadelphia metropolitan area, by $2.9 billion in the Seattle metropolitan area, and by $800 million in the Milwaukee metropolitan area. (see Table 5)

TABLE 5

Housing values rise as homicides decline

Estimated impact of a 10 percent reduction in homicides in 2010 on total residential housing values in 2011, by metropolitan area ($ billions)

Metropolitan area	Value of all housing, 2010	Increase in value of all housing, 2011
Boston	$532,000,000,000	$4,400,000,000
Chicago	$266,000,000,000	$2,200,000,000
Dallas	$234,000,000,000	$2,400,000,000
Jacksonville	$75,000,000,000	$600,000,000
Milwaukee	$99,000,000,000	$800,000,000
Philadelphia	$391,000,000,000	$3,200,000,000
Seattle	$349,000,000,000	$2,900,000,000

Source: Zillow Real Estate Market Reports of December 2010 and author's calculations

The large gains in the value of a city's housing stock associated with a 10 percent decline in homicides suggest that a successful effort to reduce violent crime could generate large revenue gains from the property taxes applied to those higher home values. Unfortunately, data constraints preclude our estimating those revenue gains with confidence because housing stock data cover metropolitan areas, and in each case, these metropolitan areas encompass city and suburban jurisdictions with varying property tax rates.

Estimating other direct savings and intangible benefits of reducing violent crime

Economists, political scientists, and sociologists have examined the various costs that violent criminals impose on their victims and communities. We have reviewed this research to establish the best-available methodologies for estimating those various costs and consequently calculated the savings and benefits that should follow from reductions in the incidence of those crimes.

We estimate that a 10 percent reduction in violent crimes nationwide would generate direct and indirect saving of $20 billion per year. Similarly, a 25 percent reduction in those crime rates would generate benefits estimated at $50 billion per year.

The various costs associated with violent crimes

Most analysts distinguish between the direct or tangible costs of crimes and their indirect or intangible costs. The direct costs include, first, the value of property destroyed or damaged in the course of violent crimes, surviving victims' medical expenses and lost earnings from crime-related injuries, the productivity losses for those victims associated with the aftermath of these crimes over both the short and long term, and the productivity losses for murder victims based on their expected earnings for the remainder of their working lives.

The direct costs also include the expenditures by cities, counties, and states to apprehend, prosecute, and incarcerate the perpetrators of these crimes. Other direct costs include various types of private spending undertaken to avoid crime, including expenditures for home security systems, car alarms, security guards, and other security services. Finally, there are the economic losses entailed in moving accused or convicted people from a city's labor force to its jails and prisons and sacrificing the productivity and other benefits associated with their working, paying taxes, and buying goods and services. These last costs may seem problematic to some readers, because criminals are not commonly thought of as potentially productive members of a community. Yet most criminals do work when they are not incarcerated, and we use the minimum wage to estimate these earnings.

To calculate these direct costs of crime, we use what researchers commonly call the "cost-of-illness" approach, which disaggregates these direct costs into the separate elements listed above.

While the direct costs of crime are significant, as are the associated direct savings from reducing such crime, the estimated value of the indirect and more intangible costs of violent crimes is much larger. These indirect costs focus on phenomena that have no universally accepted measure of their values, especially the pain, suffering, and reduced quality of life that result from being a surviving victim or potential victim of violent crime. Researchers have developed various ways of estimating these intangible costs using a "hedonic model," a "contingent valuation" approach, a jury-award method, or some combination of these approaches. We examine each of these approaches in detail in this section of the report.

The "cost-of-illness" approach to calculating the direct costs of violent crime

The "cost-of-illness" approach was first developed by public health experts to measure both the direct and intangible economic costs of illnesses and diseases. Its application to crime begins by identifying all of the distinct losses or costs associated with a crime and then estimating the value of each element. Most of such analyses of crime rely on FBI Uniform Crime Reports and the Department of Justice National Crime Victimization Surveys. The FBI Uniform Crime Report provides estimates of the incidences of a wide range of criminal activities based on reports of known offenses and arrests from various American law enforcement agencies. These crime incidence statistics cover eight serious or "Part I" offenses, including four offenses classified as violent crimes (murder, rape, robbery, and aggravated assault) and four others classified as property crimes (burglary, larceny-theft, motor vehicle theft, and arson).[20]

The Department of Justice National Crime Victimization Surveys collect information on the victims of those crimes, including their out-of-pocket costs for medical treatment, property losses, and lost earnings. These data are drawn from a national sample of 42,000 U.S. households covering 76,000 individuals. The Bureau of Justice Statistics uses these data for an annual publication presenting a variety of

statistics on rapes and sexual assaults, robberies, aggravated assaults, burglaries, larceny-thefts, and motor vehicle thefts.

Using these data the Bureau of Justice Statistics published one of the first comprehensive studies of the costs of crime in 1984.[21] That study calculated that the direct victim-related costs of crime in 1981 nationwide totaled $22.9 billion (2010 dollars). The bureau's follow-up study 10 years later found that direct victim-related costs grew to $27.4 billion (2010 dollars) from 1981 to 1991. These reports were important early contributions to the research on the costs of crime.

To estimate the direct costs of violent crimes in the United States today using this approach, we begin by updating the calculation of the direct economic costs borne by the victims of violent crime. Based on National Crime Victimization Surveys data for 2006–2010, we estimate the average out-of-pocket medical costs, property losses, and lost earnings of victims of rape, robbery, and assault over that five-year period.[22] This analysis shows, unsurprisingly, that victims of aggravated assault incur the largest average medical costs ($1,969) and highest total average costs ($2,133), and victims of completed robberies incur the largest average property losses ($1,263). (see Table 6)

TABLE 6

The direct costs of violent crime excluding murder

Average out-of-pocket costs for victims of rape, robbery and assault based on estimates from the national crime victimization survey, 2006–2010 ($2010)

Crime	Medical	Property	Lost earnings	Total
Rape/Sexual Assault	**$201**	**$28**	**$17**	**$246**
Rape	$314	$41	$24	$379
Completed Rape	$510	$41	$45	$596
Attempted Rape	$151	$40	$3	$195
Sexual Assault	$50	$8	$5	$63
Robbery	**$244**	**$927**	**$67**	**$1,238**
Completed	$122	$1,263	$59	$1,444
Attempted	$535	$107	$87	$729
Assault	**$128**	**$10**	**$51**	**$188**
Aggravated Assault	$526	$8	$61	$596
Completed	$1,969	$14	$150	$2,133
Attempted	$0	$6	$18	$24
Simple Assault	$27	$10	$48	$82

Source: U.S. Department of Justice, National Crime Victimization Survey, 2006-2010.

Using a similar approach we also estimate the direct victim-related costs of the fourth violent crime: murder. This calculation assumes no out-of-pocket medical costs or property losses so the victim's lost earnings over a lifetime constitute the only victim-related costs of the crime. To estimate the value of those earnings, we use data on the average age of murder victims from the National Vital Statistics System and data on average income by age reported by the U.S. Census Bureau's Current Population Survey. We apply a discount value of 3 percent to derive the net present value of the lifetime earnings of murder victims, which we estimate to average $925,000. Using this figure, the results in Table 3, and the incidence of violent crimes, we estimate that violent crimes nationwide in 2010 imposed direct costs on victims totaling $14.6 billion. (see Table 7)

TABLE 7

The total direct cost of violent crimes

Victim-related costs from violent crimes including murder, nationwide, 2010

Crime	Direct costs per offense	Offenses in 2010	Total direct costs
Murder	$924,562	14,748	$13,635 million
Rape	$379	84,767	$32 million
Robbery	$1,238	367,832	$455 million
Aggravated Assault	$596	778,901	$464 million
Total	--	**1,246,248**	**$14,587 million**

Source: FBI Uniform Crime Reports (2010); Department of Justice National Crime Victimization Survey, and author's calculations.

Calculating the costs of violent crime for the criminal justice system is more complex because the data on these costs are not usually disaggregated by types of crime. To estimate the law enforcement, judicial, and correctional costs of violent crimes, we start with total U.S. expenditures for police protection, judicial and legal services, and corrections in 2007, reported by the Bureau of Justice Statistics.[23] We then adjust the 2007 data to 2010 dollars.[24] Next, we use FBI Uniform Crime Report arrest data to calculate arrests for violent crimes as a share of all arrests.

In 2010 murders accounted for 0.1 percent of all arrests, rapes accounted for 0.15 percent, robberies accounted for 0.9 percent, and aggravated assaults accounted for 3.1 percent. We apply these shares to the data on the aggregate costs of police and the judicial system. By this approach, we can estimate that the policing of

violent crimes cost $4.6 billion in 2010, and the adjudication of those accused of violent crimes cost $2.2 billion.

These estimates, however, are very conservative. Police departments and courts give much higher priority to violent crimes. Therefore it is very likely that the share of police and judicial resources devoted to these crimes substantially exceeds their share of all crimes.

The corrections costs for violent crimes are also difficult to measure. Some studies estimate correctional costs per offense by multiplying the average cost per inmate for all U.S. jails and prisons by the number of inmates incarcerated for each offense, and dividing that result by the number of total offenses committed each year. But this approach assumes that the number and distribution of violent criminal offenses committed each year remains constant, which is not the case.

For a more accurate estimate, then, we start with data from the Bureau of Justice Statistics on the number of inmates incarcerated in jails and prisons in 2010, and total correctional costs at federal, state, and local levels. On this basis we calculate that the cost of incarceration per inmate in 2010 was $33,400.[25] Next we multiply the number of 2010 convictions for each of the four violent crimes by the average sentence for each crime, the percentage of each sentence actually served, and the estimated annual cost per inmate.[26] Using this approach we estimate that the correctional costs for the four violent crimes nationwide totaled $15.4 billion in 2010.

The costs of the criminal justice system for violent crimes in 2010, therefore, totaled $22.2 billion: $4.6 billion (policing) + $2.2 billion (courts) + $15.4 billion (corrections).

Other direct costs of these crimes involve the economic losses from the foregone productivity or economic output of those convicted of violent crimes. To estimate these costs we start with data on the pre-arrest personal incomes of convicted felons based on a 2002 national survey of inmates.[27] These data suggest that convicted criminals earn about 40 percent of the U.S. average personal income. Using the approach adopted above to calculate correctional costs, we can estimate the lost income attributable to criminals for each type of violent crime based on the average income of convicted felons, the average age at sentencing for each type of violent crime, and the average sentence served for each violent crime. We estimate that in 2010 violent crimes nationwide cost the U.S. economy some $5.4 billion in income, which those convicted would otherwise have produced.

Based on these calculations, the direct costs of violent crimes in 2010 totaled $42.2 billion nationwide: $14.6 billion (victim-related costs) + $22.2 billion (police, courts, and correctional costs) + $5.4 billion (lost work product of criminals). This is equivalent to a cost of $137 for every person in the United States.

Therefore, a 10 percent reduction in violent crimes nationwide would save Americans nearly $1.5 billion in victim-related costs and $2.2 billion in law enforcement and judicial costs while increasing economic output by $540 million. Similarly, a 25 percent reduction in these crimes would save Americans $3.6 billion in victim-related losses and nearly $5.6 billion in law enforcement and judicial spending while increasing the economy's output by nearly $1.4 billion annually.

Estimating the intangible costs of violent crime

The hedonic model approach for estimating these intangible costs

The academic literature on the costs of crimes includes extensive analysis of indirect or intangible costs, notably the pain, suffering, and diminished quality of life experienced by victims of violent crimes. This literature includes numerous studies that apply so-called hedonic models drawn from housing markets. Hedonic models are designed to reveal people's underlying preferences about the characteristics or attributes of a good, and then use those findings to estimate its value. In a housing market the value of a property can be estimated based on the number of bedrooms and bathrooms, the size of the lot, the location and characteristics of the neighborhood, and so on.

Cost-of-crime studies that use hedonic pricing assume that people reveal their preferences about crime levels when they purchase their homes, based on crime levels in that area. These studies apply econometric models that control for other variables that influence housing prices in order to isolate the monetary value that homebuyers place on reduced risk of crime.

The first study to adopt a hedonic model approach to estimate the intangible costs of crime used a sample of single-family home sales in Rochester, New York, in 1971.[28] After controlling for the characteristics of the properties and neighborhoods as well as other variables, the author found that an increase in per capita

property crime of 4.5 percent was associated with 3 percent lower home values. Using these findings the author estimated that the social and personal costs of a property crime in Rochester in 1971 averaged about $2,880 (in 2010 dollars).

A broader study from 1999 analyzed the relationship between crime rates and urban flight for 127 American cities from 1970 to 1993.[29] Applying a hedonic model to FBI data on rapes, robberies, aggravated assaults, burglaries, larcenies, and auto thefts, the authors concluded that a 10 percent increase in those crimes led to a 1 percent decline in a city's population, and that those population losses were associated with identifiable reductions in housing prices. Finally, a 2010 study focused on housing prices and crime rates in Miami-Dade County, Florida, from 1999 to 2007 and found that a 1 percent increase in violent crime per acre reduced housing prices by about 0.25 percent.[30]

A key strength of this approach is that it relies on market data, which generally provide the most reliable information on the true value of goods. The challenge lies in identifying all the factors other than crime that influence housing prices so the relationship between crime and housing prices can be isolated. This is particularly difficult for factors such as poverty, which may be correlated with both crime and low real estate values.

Critics also note that an observed correlation between changes in crime rates and changes in housing prices may represent a causal relationship that runs from crime to housing prices and not in the other direction. Our own analysis of this relationship avoids the pitfalls of hedonic modeling by using the Granger Causality regression, which explicitly tests for and establishes the direction of the effect. In addition, this approach is often unable to distinguish the costs of different types of crimes, because rates for the various kinds of crimes tend to rise and fall together.

The contingent-valuation approach to estimating intangible costs

In the absence of market data on a public good such as reduced crime or clean air, some economists rely instead on surveys that measure how much people say they would be willing to pay for those public goods. This approach is called "contingent valuation," because the willingness-to-pay values reported in those surveys are contingent on the conditions presented in the survey. Contingent valuation analysis was first used in environmental economics, but it is now commonly applied to crime.

A 2001 analysis, for example, used a 1998 survey of more than 1,200 people to estimate how much people would be willing to pay to reduce gun violence by 30 percent.[31] The study estimated a willingness-to-pay value of $24.5 billion, or about $1.2 million per gunshot injury. A 2004 analysis similarly surveyed 1,300 adults to estimate how much Americans would be willing to pay for a 10 percent reduction in murders, rapes, robberies, assaults, and burglaries.[32] Based on the survey the authors reported that the total personal and social costs of these crimes are between 1.5 times and 10 times greater than had been found previously—a result that they attributed to their capturing a range of intangible as well as direct costs.

Here, too, many economists question the reliability of this approach. The responses from the surveys sometimes contradict basic economic axioms, especially in the environmental area, when respondents say that they would be willing to pay the same or similar amounts to reduce water pollution by widely different amounts.[33] Another criticism is that contingent valuation suffers from "hypothetical bias" because survey respondents have no actual stake in the result.

Finally, some analysts argue that the surveys do not measure people's actual economic preferences but rather their general approval or disapproval about a public good such as reduced gun violence or cleaner air. So respondents may derive a sense of satisfaction from expressing their (theoretical) willingness to pay a high price for less crime or cleaner air, but in practice they might strongly oppose a new tax for the same purpose.

Despite these criticisms the application of contingent valuation analysis to crime has certain advantages. In contrast to its use in the environmental area, respondents generally express a willingness to pay more for greater reductions in crime. On balance, a panel of experts recently concluded that a contingent valuation approach can produce estimates that provide a credible starting point for assessing the value of greater public safety, even though it may overstate people's actual willingness to pay for it.[34]

The jury-award approach to estimating intangible costs

This approach also applies people's stated views on the intangibles costs of crime but in a less hypothetical way. This approach relies on data from jury awards in civil suits that compensate victims of violent crimes for pain and suffering. A 1988

study used jury awards to estimate these costs for victims of 10 types of crimes, including rape, robbery, and assault.[35] This study also introduced other intangible costs unexamined by previous researchers, including the costs to victims' mental health and anxieties related to perceived "risks of death."

A subsequent study published by the U.S. Department of Justice's National Institute of Justice also used jury awards to estimate the costs arising from the pain and suffering of rape, robbery, and assault victims.[36] This study also estimated a variety of costs associated with murders, which other researchers usually had left unexamined, including the pain and suffering of third parties, medical costs, and lost productivity.[37]

While the jury award approach provided a new way of capturing the intangible costs of crimes, it also raised certain concerns. For instance, since litigation is often costly the cases that are pursued civilly may involve unusually violent and injurious acts, creating an upward bias in the cost estimates.[38] In addition, some analysts argue that jury awards are inappropriate because they represent *ex-post* or "willingness-to-accept" estimates of the cost of crime rather than *ex-ante* or "willingness-to-pay" estimates derived, in principle, from contingent valuation surveys. Researchers have found that willingness-to-accept values are typically two to three times larger than comparable willingness-to-pay values,[39] which critics point to as support for the view that jury awards incorporate an upward bias.

In the end we find that the most credible estimates of the intangible costs were derived from a very large and recent study that used a modified jury compensation approach to calculate per-offense pain and suffering estimates for violent crime, entitled, "The Cost of Crime to Society: New Crime-Specific Estimates for Policy and Program Evaluation."[40] The authors based their pain and suffering estimates on Jury Verdict Research (2004), which provides data on jury verdicts for individual injuries such as gunshot wounds, knife wounds, and rape-related injuries, based on their level of severity.[41] The authors then used 2007 data gathered by the Bureau of Justice Statistics on the probability that each offense leads to various injuries, to estimate pain and suffering costs for individual offenses. Moreover, as shown in Table 9 on page 32, the aggregate estimates of direct and intangible costs found by the "Cost of Crime to Society," a study sponsored by the National Institute on Drug Abuse and the National Institutes of Health,[42] are very close to those derived in a leading contingent valuation analysis that is also included in Table 9.[43]

The limitations of all crime-related cost estimates

The data used in all of these analyses of crime and their costs have certain limitations. Estimates of the income losses suffered by victims of violent crimes, for example, generally assume that those victims are broadly representative of the whole population. In fact, data from the National Crime Victim Survey suggest that victims of rape, robbery, and assault have lower-than-average annual incomes: 35 percent of the victims of violent crimes between 2006 and 2010 lived in households with incomes of less than $20,000, compared to 20 percent of all American households.[44] Similarly, data from municipal police departments suggest that 70 percent to 80 percent of homicide victims in large cities have criminal records.[45] This suggests that the lifetime earnings losses attributed to murder victims may be overstated.

At the same time other factors suggest that most estimates of the costs of crime are understated. Most notably, crime is notoriously underreported. Data from the National Crime Victim Survey indicate that less than 60 percent of all violent crimes are reported to the police: these respondents report that only 45 percent of rapes, 59 percent of assaults, and 62 percent of robberies are reported to the police. If those data are correct, then the estimates of the costs to victims of violent crime in most of the current research including this study are substantially understated.

Between the various data issues and the approaches for estimating costs, researchers have produced a wide range of estimates. The estimates of the direct or tangible costs of one murder, for example, range from $1.3 million to $1.5 million, while the estimates for the indirect or intangible costs of one murder range from $2.9 million to more than $8.5 million. Similarly, the estimates of the direct costs of a rape or sexual assault vary from $7,642 to $41,774, and the estimated indirect or intangible costs range from $94,115 to $200,746. As noted above, we find that the per-offense estimates derived by the "Cost of Crime to Society" study, on balance, are the most reliable available. (See Table 8 for a breakdown of these intangible cost estimates by four key researchers.)

TABLE 8

Estimates of Costs of Violent Crime

Survey of recent research on the direct and intangible costs of violent crimes, per offense (in 2010 dollars)

Crime	Cohen (1988)	Miller et al (1996)	Cohen et al (2004)	McCollister et al (2010)
Murder	**N/A**	**$4,425,284**	**$9,098,564**	**$9,844,715**
Direct Costs	N/A	$1,543,022	N/A	$1,294,771
Intangible Costs	N/A	$2,882,263	N/A	$8,549,945
Rape & Sexual Assault	**$103,471**	**$130,477**	**$222,305**	**$242,521**
Direct Costs	$9,357	$7,642	N/A	$41,774
Intangible Costs	$94,115	$122,836	N/A	$200,746
Robbery	**$25,522**	**$12,059**	**$217,615**	**$26,711**
Direct Costs	$2,258	$3,457	N/A	$21,672
Intangible Costs	$23,265	$8,602	N/A	$5,040
Assault	**$24,375**	**$14,114**	**$65,660**	**$33,394**
Direct Costs	$855	$2,344	N/A	$19,737
Intangible Costs	$23,520	$11,770	N/A	$13,607

Sources: Mark A. Cohen, "Pain, Suffering, and Jury Awards: A Study on the Cost of Crime to Victims," Law & Society Review 22 (3) (1988): 537–555; Ted R. Miller, Mark A. Cohen, and Brian Wiersema, Victim Costs and Consequences: A New Look (Washington: National Institute of Justice Research Report, U.S. Department of Justice, 1996); Mark A. Cohen and others, "Willingness-To-Pay for Crime Control Programs," Criminology 42 (1) (2004): 89–109; Kathryn E. McCollister, Michael T. French, and Hai Fang, "The Cost of Crime to Society: New Crime-Specific Estimates for Policy and Program Evaluation," Drug and Alcohol Dependence 108 (1–2) (2010): 98–109. The estimates from Cohen and others' 2004 study assume a discount rate of 3.0 for hypothetical bias. The McCollister and others' 2010 estimates do not include intangible costs associated with a victim's embarrassment or humiliation.

The costs of violent crimes for eight U.S. cities and the benefits and savings from reducing those crimes

Earlier in this report we provided the details behind our estimate that violent crimes across the United States cost Americans nearly $200 billion per year, including $46 billion in direct costs and nearly $150 billion in indirect, intangible costs. (see Table 4 on page 12) To estimate those intangible or indirect costs, we rely, as noted, on the recent "Cost of Crime to Society" study conducted by Kathleen McCollister, Michael French, and Hai Fang, which draws on jury award data to estimate the value of the pain and suffering arising from injuries from gunshot wounds, knife wounds, and physical assaults.[16]

Based on these data the authors calculated the average pain and suffering costs for each type of violent crime. We adjusted their figures to 2010 dollars. While jury awards may introduce an upward bias, we suspect that the substantial underreporting of most types of violent crimes introduces a larger downward bias. While we consider these estimates to be the most reliable available, we are confident that the actual, total costs of all violent crimes, direct and intangible, are probably even higher than those reported here.

The direct economic costs of those violent crimes cover the medical, property, and work- or productivity-related costs borne by surviving victims and by victims of homicide; the costs of policing, courts, and correctional facilities for those who commit violent crimes; and the value of the work or productivity-related losses of those arrested for violent crimes. For the eight cities examined in this report, those direct costs come to nearly $3.7 billion per year, ranging from an estimated $89 million per year in Seattle to more than $1.1 billion per year in Chicago. On a per-resident basis, these direct annual costs range from $144 per resident of Seattle to $472 per resident of Philadelphia.[17] (see Table 9 on next page)

TABLE 9

The multibillion dollar cost of violent crime

Estimated direct costs of violent crime, by city, 2010 ($ millions)*

City	Victim costs	Law enforcement and justice system costs				Criminal productivity costs	Total costs	Cost per resident
		Federal	State	Local	Total			
Boston	$72	$12	$48	$42	$102	$24	$198	$308
Chicago	$426	$64	$265	$218	$547	$132	$1,104	$396
Dallas	$145	$20	$86	$69	$175	$43	$363	$278
Houston	$268	$47	$187	$159	$393	$91	$752	$330
Jacksonville	$78	$12	$47	$40	$100	$24	$202	$246
Milwaukee	$92	$14	$55	$46	$115	$27	$235	$388
Philadelphia	$299	$41	$171	$139	$351	$86	$736	$472
Seattle	$21	$7	$26	$23	$56	$12	$89	$144
Total	$1,401	$217	$885	$786	$1,839	$439	$3,679	$320 (ave)

*The criminal justice system cost estimates presented here are distributed among federal, state and local governments based on national data on the distribution of those costs, as reported by the Bureau of Justice Statistics, and the incidence of violent crime in each jurisdiction. The actual costs borne by each of our eight cities may differ from those presented here based on how those costs are distributed between state and local governments.

Source: Authors calculations. The estimates of justice system costs by level of government are based on data from the Bureau of Justice Statistics.

While Table 9 includes the total law enforcement and justice system costs for violent crimes at each level of government, we also disaggregate those costs into three components: police protection, the judicial system, and corrections. These data show that correctional-system operations account for nearly 72 percent of the total costs, ranging from $38 million per year for Seattle to $397 million per year for Chicago. Of the remaining justice-system expenditures, police operations for the eight cities account for an average of about 19 percent, and the judicial system accounts for the remaining 9 percent. (see Table 10)

The annual intangible or indirect costs per resident for the pain and suffering of the victims of violent crimes, on average, are nearly four times greater than the annual direct costs of those crimes per resident. These intangible costs for the eight cities total nearly $14 billion per year, ranging from $216 million per year in Seattle to $4.2 billion per year in Chicago. On a per-resident basis, these annual intangible costs average $1,202 per crime and range from an estimated $350 for Seattle to $1,905 for Philadelphia.

The large differences among the eight cities in both intangible and direct costs per resident reflect differences in both total violent crime rates and the rates of different types of violent crime. In particular, there are significant differences in murder

TABLE 10

The costs of protecting against violent crime

Law enforcement and justice system expenditures on violent crimes, by city, 2010 ($ millions)

City	Police protection	Judicial system	Corrections	Total
Boston	$21	$10	$71	$102
Chicago	$101	$49	$397	$547
Dallas	$31	$15	$129	$175
Houston	$80	$38	$275	$393
Jacksonville	$20	$10	$76	$106
Milwaukee	$22	$11	$83	$115
Philadelphia	$64	$31	$256	$351
Seattle	$13	$6	$38	$56
Total	$352	$170	$1,319	$1,839

Source: Thad W. Hall & Kyle Kalhoefer, Justice Expenditure and Employment: 1982-2007 (Washington: Bureau of Justice Statistics, 2011)

TABLE 11

The cost of violent crime per resident in eight U.S. cities

Annual intangible costs of violent crimes, by city, total and per resident, direct costs per resident, and total costs per resident, 2010

City	Intangible costs ($ millions)	Intangible costs per resident	Direct costs, per resident	Total costs per resident
Boston	$734	$1,142	$308	$1,447
Chicago	$4,206	$1,486	$388	$1,874
Dallas	$1,444	$1,106	$278	$1,383
Houston	$2,655	$1,165	$330	$1,494
Jacksonville	$802	$977	$246	$1,221
Milwaukee	$900	$1,486	$388	$1,873
Philadelphia	$2,970	$1,905	$472	$2,378
Seattle	$216	$350	$144	$492
Total/Average	$13,920	$1,202	$320	$1,520

Source: Author's calculations

rates across the eight cities, and the direct and indirect costs per resident are much higher for murders than for the other violent crimes. The total annual costs of violent crimes per resident in the eight cities average $1,520, ranging from $492 per resident per year in Seattle to $2,378 per resident per year in Philadelphia. (see Table 11 on previous page)

Based on these cost calculations, a 10 percent reduction in violent crimes would generate estimated direct savings or benefits totaling $368 million per year for all eight cities, ranging from $9 million per year for Seattle to $110 million for Chicago. The total annual savings and benefits, direct and indirect, from a 10 percent reduction in violent crime for all eight cities come to nearly $1.8 billion, ranging from $30 million in Seattle to $531 million in Chicago.

A 25 percent reduction in violent crime in these eight cities would generate direct savings or benefits totaling $921 million per year, ranging from an estimated $22 million in annual benefits in Seattle to $276 million in annual benefits for Chicago. A 25 percent reduction in violent crime would generate total benefits and savings, direct and indirect, of nearly $4.5 billion per year for all eight cities, ranging from $76 million in Seattle to more than $1.3 billion in Chicago. (see Table 12)

TABLE 12

Total costs of violent crime and total savings and benefits

Annual direct and indirect costs of violent crimes and annual direct savings and total benefits from reducing those crimes by 10 percent and 25 percent, by city, 2010 ($ millions)

City	Costs			10% Crime Reduction		25% Crime Reduction	
	Direct	Intangible	Total	Direct Savings	All Benefits	Direct Savings	All Benefits
Boston	$198	$734	$932	$20	$93	$50	$233
Chicago	$1,104	$4,206	$5,310	$110	$531	$276	$1,327
Dallas	$363	$1,444	$1,807	$36	$181	$91	$452
Houston	$752	$2,655	$3,407	$75	$341	$188	$852
Jacksonville	$202	$802	$1,004	$20	$100	$51	$251
Milwaukee	$235	$900	$1,135	$24	$114	$59	$284
Philadelphia	$736	$2,970	$3,705	$74	$371	$184	$926
Seattle	$89	$216	$305	$9	$30	$22	$76
Total	$3,679	$13,927	$17,605	$368	$1,761	$921	$4,481

Source: FBI Uniform Crime Reports (2010), McCollister et al. (2010), author's calculations

These estimates of the benefits of lower violent-crime rates are conservative because they do not include a range of secondary benefits associated with reduced crime. Cities known to be safer places often experience increased tourism, which can generate substantial gains for businesses located there and for city governments that collect revenues on those gains. As we have established, falling crime rates also are associated with higher residential and commercial property values, which enrich households and businesses and generate higher property tax revenues for city governments.

In addition, falling crime rates generally result in lower insurance rates and payments, further supporting businesses and households.[46] Finally, falling violent crime rates can improve a city's general economic and business environment, leading to higher overall levels of investment and population gains.

Some of the direct savings from reducing violent crime will accrue directly to municipal budgets. To calculate the benefits for the budgets in the eight cities, we focus first on costs that come directly from those budgets. These estimates are provided in Table 9 on page 32, using local law enforcement and justice-system costs. These local budgetary costs for violent crimes come to $786 million per year for all eight cities, ranging from $23 million in Seattle to $218 million in Chicago. Once again, these estimates are conservative. They are based on local law enforcement and justice system costs divided by violent crimes' share of all crimes, when violent crimes usually claim a disproportionate share of such spending.

The direct budgetary costs of violent crimes and corresponding budgetary benefits from reducing those crimes also include tax revenues on the income of victims who would have been able to work in the days following a crime and on foregone income by those convicted of violent crimes. To estimate the magnitude of these additional revenues from reducing violent crime, we analyzed U.S. Census Bureau data on the share of total household income that each city collects in local taxes. These implicit tax rates average 4.3 percent and range from 3 percent in Dallas and Houston to 8.6 percent in Philadelphia.

Next, we sum the foregone income of the victims of crimes and the criminals, using the methodology described earlier. This foregone income totals $1.84 billion per year for the eight cities, ranging from $33 million for Seattle to $557 million for Chicago. We multiply this foregone income by the implicit tax rate for each city to estimate the revenues foregone as a result of violent crime.[47] Our estimates of these foregone local revenues total $83 million per year for the eight cities, ranging from $1 million for Seattle to $33 million for Philadelphia. (see Table 13)

TABLE 13

The municipal costs of violent crime

Annual municipal revenue effects of violent crime from foregone income, by city, 2010 ($ millions)

City	Victim-Related Costs	Criminal Productivity Costs	Total Foregone Income	Local Taxes as % of Household Income	Foregone Tax Revenues
Boston	$72	$24	$96	7.0%	$7
Chicago	$426	$132	$557	3.3%	$18
Dallas	$145	$43	$188	3.0%	$6
Houston	$268	$91	$359	3.0%	$11
Jacksonville	$78	$24	$103	4.0%	$4
Milwaukee	$92	$27	$120	2.5%	$3
Philadelphia	$299	$86	$385	8.6%	$33
Seattle	$21	$12	$33	3.3%	$1
Total	**$1,401**	**$439**	**$1,841**	**4.3% (ave)**	**$83**

Source: U.S. Census Bureau, Federal, State and Local Governments, Government Finances; U.S. Census Bureau, 2010 American Community Survey; and authors' calculations

The total municipal budgetary effects from reducing violent crime in the eight cities by 10 percent and 25 percent are calculated by summing those foregone revenues and the budgetary savings from lower local police, judicial, and correctional system spending. The municipal budget savings from a 10 percent reduction in violent crimes total $82 million per year for the eight cities, ranging from $2 million per year in Seattle to $24 million per year in Chicago. The local budget savings from a 25 percent reduction in violent crime total $205 million per year for all eight cities, ranging from $6 million per year in Seattle to $59 million per year in Chicago. (see Table 14)

TABLE 14

Annual municipal cost and savings from reducing violent crime

Local budgetary costs of violent crime and local budgetary savings from 10 percent and 25 percent reductions in those crimes, by city, 2010 ($ millions)

City	Foregone Tax Revenues	Police & Justice System Spending	Total Revenue & Spending Costs	Saving: 10% Reduction in Violent Crime	Saving: 25% Reduction in Violent Crime
Boston	$7	$42	$49	$5	$12
Chicago	$18	$218	$236	$24	$59
Dallas	$6	$69	$75	$8	$19
Houston	$11	$159	$170	$17	$43
Jacksonville	$4	$40	$44	$4	$11
Milwaukee	$2	$46	$49	$5	$12
Philadelphia	$33	$139	$172	$17	$43
Seattle	$1	$23	$24	$2	$6
Total	$83	$736	$819	$82	$205

Source: Author's calculations

Alternative uses of the municipal savings from reducing violent crime

Finally, we present in graphical form alternative uses of the direct annual budget savings from reducing violent crime in each of the eight cities by 25 percent, as well as our earlier findings on the annual direct costs of those crimes to local budgets and examples of what the annual savings from the reductions in those crimes could be used for at the city level. (see next page)

As noted earlier, our criminal justice system cost estimates are distributed among federal, state, and local governments based on national data on the distribution of those costs as reported by the Bureau of Justice Statistics. The actual costs borne by each of our eight cities may differ from those presented here based on how costs are distributed between state and local governments. Some of the projected savings presented here, therefore, would accrue to state governments as well as city governments. Here we attribute those estimated savings to the local level to illustrate the budgetary impact of achieving reductions in violent crime of 10 percent or 25 percent.

Boston

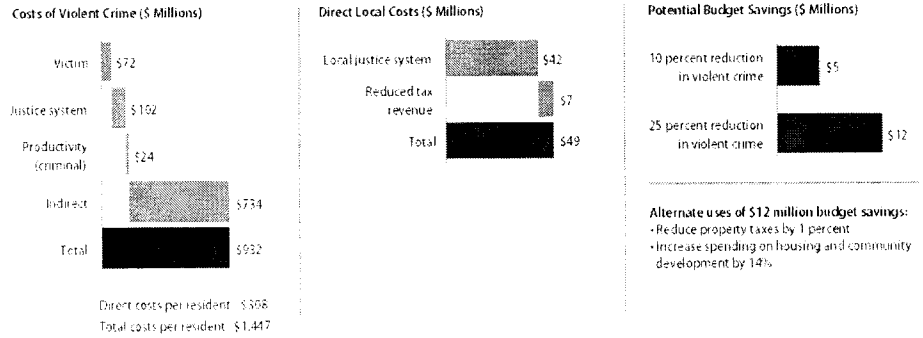
In 2010 the Boston Police Department received reports of 73 murders, 256 rapes, 1,926 robberies, and 3,564 aggravated assaults. Boston experienced twice as many violent crimes per capita as the nation as a whole and the second-highest rate of aggravated assaults of the eight cities. These violent crimes cost the city $198 million in 2010 or $308 per resident.

A 25 percent reduction in these violent crimes would generate an estimated $12.1 million in annual savings for the Boston city budget, including $10 million in local justice-system savings and $2 million in additional tax revenue. These extra savings would be enough for the city to consider a mix of boosting city spending on housing and community development by up to 14.4 percent or reducing property taxes by up to nearly 1 percent. (see Figure 2)

FIGURE 2

How Boston benefits from reducing violent crime

The costs and potential budget savings ($ millions)



Source: Author's calculations

Chicago

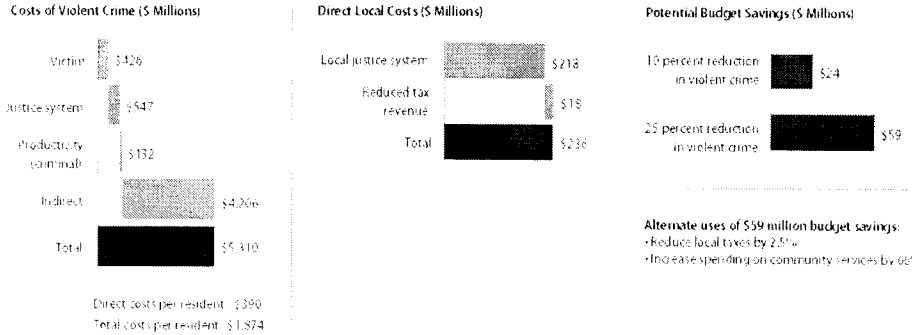
In 2010 Chicago had a reported 432 murders, 1,262 rapes, 14,213 robberies, and 13,757 aggravated assaults for a total of 29,664 violent crimes. The city's violent-crime rate was more than 2.5 times the national average and these crimes cost the city more than $1.1 billion in 2010, or nearly $400 per resident.

A 25 percent reduction in violent crime in Chicago would generate an estimated $59 million in direct cost savings to the Chicago city budget, including $54 million in law enforcement and justice-system savings and $5 million in increased revenues. These savings would enable Chicago to consider a mix of reducing all local taxes by up to 2.5 percent or increasing city spending on community services by up to 66 percent. (see Figure 3)

FIGURE 3

How Chicago benefits from reducing violent crime

The costs and potential budget savings ($ millions)



Source: Author's calculations

Dallas

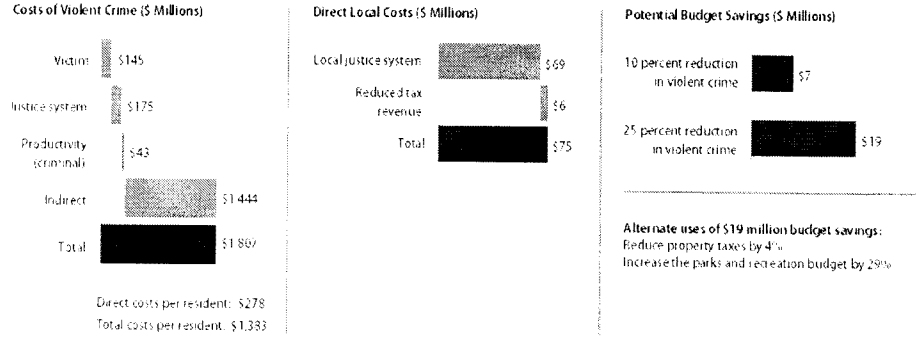
Dallas law enforcement received reports of 148 murders, 505 rapes, 4,487 robberies, and 4,021 aggravated assaults in 2010 for a total of 9,161 violent crimes. While Dallas was one of the safer cities in our sample of cities, it nevertheless sustained an estimated $363 million in direct costs from violent crimes, equivalent to $278 per resident.

Reducing violent crime in Dallas by 25 percent would generate $18.7 million for the Dallas city budget, including $17.2 million in law enforcement and justice-system savings and $1.4 million in increased revenues. These savings would enable Dallas to consider a mix of reducing property taxes by up to 4.3 percent or increasing the parks and recreation budget by up to 29 percent. (see Figure 4)

FIGURE 4

How Dallas benefits from reducing violent crime

The costs and potential budget savings ($ millions)



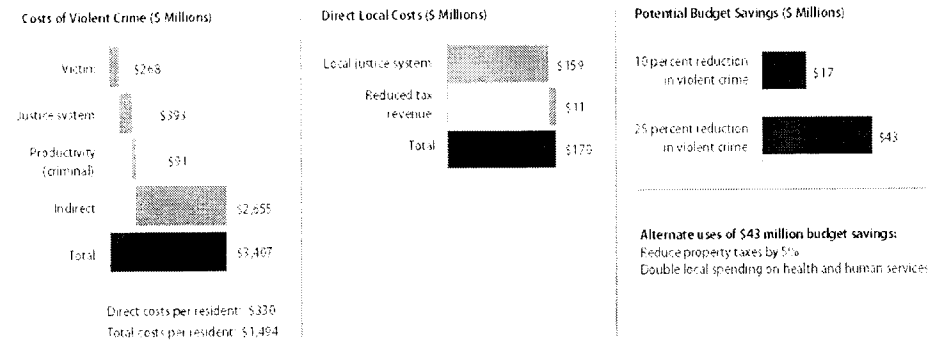
Source: Author's calculations

Houston

Houston law enforcement in 2010 received reports of 269 murders, 712 rapes, 9,449 robberies, and 12,061 aggravated assaults, a total of 22,491 violent crimes. While these crimes represented less than 3 percent of all arrests in Houston that year, they cost the city $752 million or $330 per resident.

A 25 percent reduction in violent crime would generate $43 million in savings for the Houston city budget, including $40 million in law enforcement and justice-system savings and $3 million in tax revenues. These savings could enable the city to consider a mix of doubling city spending on health and human services or cutting property taxes by 5 percent. (see Figure 5)

FIGURE 5
How Houston benefits from reducing violent crime
The costs and potential budget savings ($ millions)



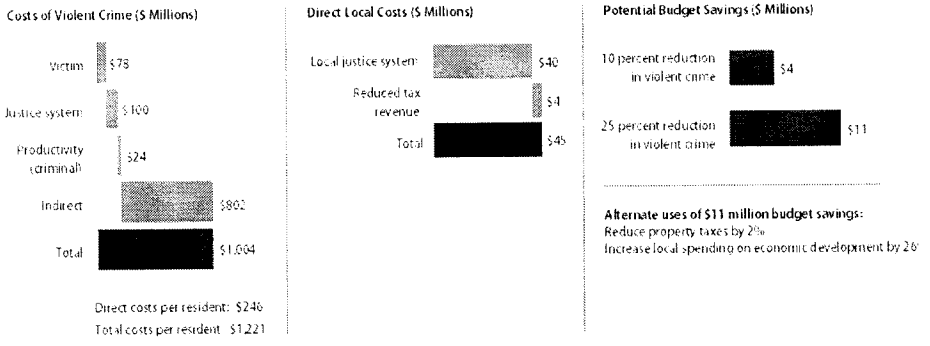
Source: Author's calculations

Jacksonville

The Jacksonville Police Department received reports of 80 murders, 316 rapes, 1,693 robberies, and 3,380 aggravated assaults in 2010, a total of 5,469 violent crimes. Jacksonville was the second safest of the eight cities based on violent crimes per capita. Nevertheless, these crimes cost Jacksonville and its citizens more than $200 million in 2010 or $246 per resident.

A 25 percent reduction in these violent crimes would generate an estimated $11.1 million in city budget savings, which would enable Jacksonville to consider a mix of cutting its property taxes by up to 2 percent or increasing local spending on economic development by up to 26 percent. (see Figure 6)

FIGURE 6
How Jacksonville benefits from reducing violent crime
The costs and potential budget savings ($ millions)



Source: Author's calculations

Milwaukee

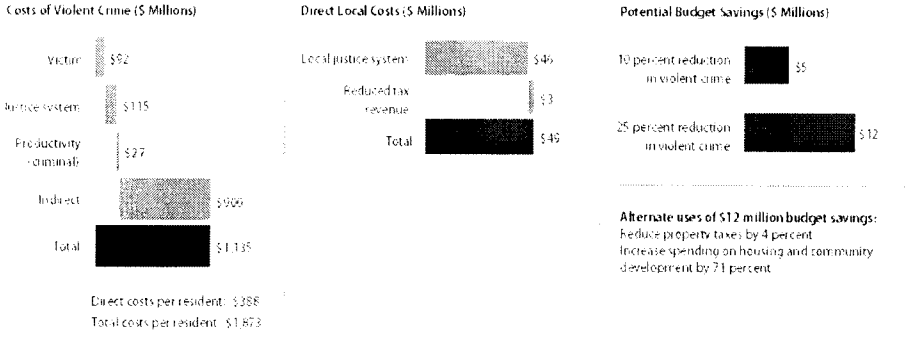
In 2010 Milwaukee law enforcement received reports of 94 murders, 197 rapes, 2,932 robberies, and 3,106 aggravated assaults, a total of 6,329 violent crimes. Milwaukee has one of the highest rates of violent crime of the eight cities, with more than one reported violent crime for every 100 residents. Even though violent crimes accounted for less than 4 percent of all arrests in Milwaukee, they cost the city $235 million or $388 per resident.

If Milwaukee reduced violent crime by 25 percent, the city government would save $12.3 million, which could enable the city to consider a mix of cutting property taxes by up to 4 percent or increasing spending on housing and community development by up to 71 percent. (see Figure 7)

FIGURE 7

How Milwaukee benefits from reducing violent crime

The costs and potential budget savings ($ millions)



Source: Author's calculations

Philadelphia

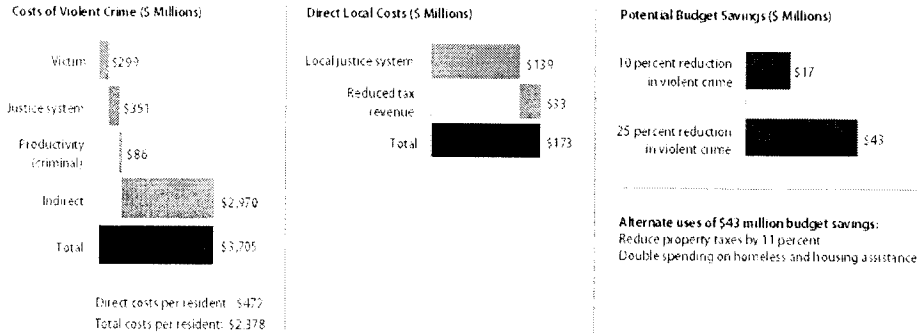
Philadelphia law enforcement received reports in 2010 of 306 murders, 945 rapes, 8,363 robberies, and 8,921 aggravated assaults, a total of 18,535 violent crimes. The violent-crime rate in Philadelphia is three times the national average. The direct costs of these violent crimes in Philadelphia totaled $736 million in 2010 or $472 per resident.

Reducing these crimes by 25 percent would generate $43 million for the Philadelphia city budget, including $35 million in law enforcement and justice system savings and $8 million in new revenues. This revenue would enable the city to consider a mix of a cut in local property taxes of up to 11 percent or up to a doubling of spending on homeless and housing assistance. (see Figure 8)

FIGURE 8

How Philadelphia benefits from reducing violent crime

The costs and potential budget savings ($ millions)



Source: Author's calculations

Seattle

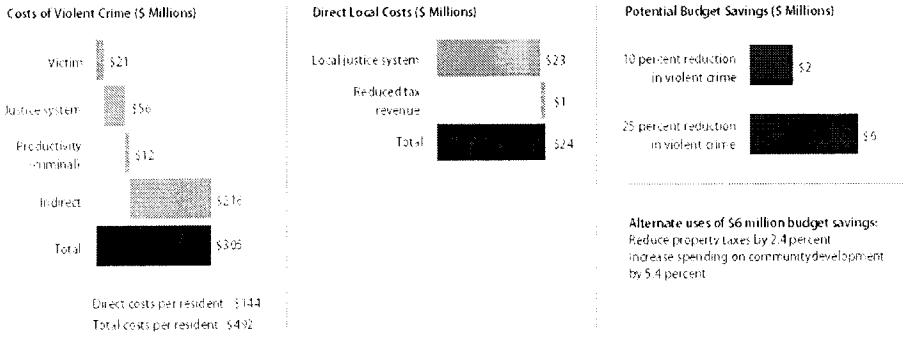
Seattle police in 2010 recorded 19 murders, 96 rapes, 1,429 robberies, and 1,971 aggravated assaults. Seattle had the lowest crime rates of the eight cities. Nevertheless, violent crime cost Seattle $89 million in 2010 or $144 per resident.

Reducing violent crime by 25 percent would produce savings for the Seattle city budget of $14.4 million, which could enable the city to consider a mix of up to a 2.4 percent cut in property taxes or an increase in city spending on neighborhoods and development of up to 5.4 percent. (see Figure 9)

FIGURE 9

How Seattle benefits from reducing violent crime

The costs and potential budget savings ($ millions)



Source: Author's calculations

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Appendix A

Methodology to test the impact of changes in violent crime rates on housing values

To isolate any causal relationship between violent crimes and property prices, we use the Granger causality test, a widely employed statistical regression method developed by Nobel Laureate Clive Granger. Granger's key insight in developing this method is that time series data on two related variables allow one to test for a statistical causal relationship between those data. Stated in technical terms, recursive substitution of a dynamic system can reduce it to a bivariate system, so long as the data include many lags of the dependent variable in the regression. Using this method, a time series X can be said to Granger-cause a time series Y if the lagged values of X provide statistically significant information about future values of Y in a regression that also includes lagged values of Y as independent variables.[50]

The main regression equation for this analysis is:

$$\Delta(\log(\text{Median Residential Property Prices}_{it})) = \beta_1 \cdot \Delta(\log(\text{Median Residential Property Prices}_{it-1})) + \beta_2 \cdot \Delta(\text{Homicides}_{it-1}) + \beta_3 \cdot \Delta(\text{NonHomicides}_{it-1}) + \gamma_t + \Delta\varepsilon_{it}$$

The dependent variable is the log differences of residential property price for geographic area i at time t: $\Delta(\log(\text{Median Residential Property Prices}_{it})$. The independent variables are the lagged value of the dependent variable and the lagged values of $\Delta(\log(\text{Homicides}_{it}))$ and $\Delta(\log(\text{NonHomicides}_{it}))$, the differenced number of homicides and nonhomicide violent crimes. Time-specific fixed effects are captured by γ_t, although they are dropped in the table.[51] (See Table A-1 on next page)

TABLE A-1
Summary Statistics at Zip Code or Census Tract Level

BOSTON, 2006-2007 (40 Observations)				
Variable	Mean	Std. dev.	Min	Max
Homicide	2.68	4.54	0	19
Nonhomicide	269.80	249.34	11	916
Rapes	9.75	9.15	0	33
Aggravated assault	165.85	158.04	11	583
Robbery	94.20	76.60	0	300
Median property value	$499,611	$387,261	$235,000	$2,600,000
Mean property value	$601,214	$428,991	$246,935	$2,600,000
CHICAGO, 2001-2010 (560 Observations)				
Variable	Mean	Std. dev.	Min	Max
Homicide	9.07	10.61	0	44
Nonhomicide	646.92	602.39	12	2322
Rapes	31.23	28.47	0	124
Aggravated assault	326.32	331.73	4	1438
Robbery	289.38	251.11	5	1010
Median property value	$220,117	$92,541	$57,500	$647,500
Mean property value	$250,919	$110,998	$71,676	$796,811
HOUSTON, 2000-2008 (868 Observations)				
Variable	Mean	Std. dev.	Min	Max
Homicide	3.06	3.46	0	26
Nonhomicide	242.98	222.98	1	1365
Rapes	8.47	7.94	0	50
Aggravated assault	125.38	115.58	0	567
Robbery	109.13	110.42	0	823
Median property value	$148,797	$86,220	$43,146	$745,520
Mean property value	$171,216	$111,823	$48,838	$788,328

JACKSONVILLE, 2001-2011 (308 Observations)				
Variable	Mean	Std. dev.	Min	Max
Homicide	3.23	3.92	0	25
Nonhomicide	193.35	179.27	9	930
Rapes	7.39	6.13	0	36
Aggravated assault	108.83	111.32	4	607
Robbery	77.16	67.76	0	322
Median property value	$133,501	$52,386	$40,000	$334,900
Mean property value	$153,938	$59,816	$50,752	$316,542

MILWAUKEE, 2005-2010 (166 Observations)				
Variable	Mean	Std. dev.	Min	Max
Homicide	3.31	4.79	0	31
Nonhomicide	233.11	245.09	1	981
Rapes	6.80	7.29	0	25
Aggravated assault	108.39	121.87	0	569
Robbery	117.93	119.90	0	425
Median property value	$135,271	$51,099	$38,000	$291,750
Mean property value	$153,579	$84,798	$57,368	$864,979

PHILADELPHIA, 2004-2009 (276 Observations)				
Variable	Mean	Std. dev.	Min	Max
Homicide	10.74	9.93	0	54
Nonhomicide	494.01	434.01	12	2207
Rapes	22.92	20.69	0	110
Aggravated assault	233.83	206.70	6	1046
Robbery	237.26	208.10	5	1052
Median property value	$154,846	$97,884	$35,900	$450,000
Mean property value	$179,738	$128,137	$41,855	$740,840

SEATTLE, 2000-2007 (976 Observations)				
Variable	Mean	Std. dev.	Min	Max
Homicide	0.23	0.60	0	6
Nonhomicide	32.32	40.02	0	399
Rapes	1.09	1.56	0	13
Aggravated assault	18.32	21.96	0	196
Robbery	12.90	17.99	0	181
Median property value	$339,846	$126,941	$129,840	$1,100,000
Mean property value	$375,827	$157,568	$131,110	$1,271,009

Source: Author's calculations

TABLE A.2
Results of Granger causality regression analysis

	Specification 1[49]	Specification 2[50]
Variables	$\Delta\{\log(MedProp_{it})\}$	$\Delta\{\log(MedProp_{it})\}$
$\Delta\{\log(MedProp_{it-1})\}$	0.737*** (0.0947)	0.649*** (0.113)
$\Delta Homicides_{it-1}$	-0.0160** (0.00650)	-0.0168** (0.00673)
$\Delta NonHomicidesi_{it-1}$	0.000446*** (0.000167)	0.000766*** (0.000179)
Observations	2,739	2,740
Number of geographic areas	396	396
Number of instruments	35	31
AR(1)	0	0
AR(2)	0.222	0.253
Hansen	0.214	0

** p < 0.05, *** p < 0.01; Standard errors in parentheses

Source: Author's calculations

The results for Specification 1 are derived from data for Chicago, Jacksonville, Houston, Milwaukee and Philadelphia. We excluded the data from Boston from our main regression specification because Boston could only provide two years of crime data by zip code. We also excluded data from Seattle from the initial regression because Seattle could only provide crime data by census tract. Census tracts cover smaller areas than zip codes, increasing the impact of changes in crime rates relative to the zip code-based analysis for the other cities. Specification 2, however, includes the crime and property value data from Boston and Seattle as well as the five original cities. These results produce a small, statistically significant positive effect from reductions in nonhomicide crime rates. We do not consider those results to be as reliable, however, given the data constraints for Boston and Seattle.

Appendix B

Testing the robustness of the impact of violent crime on housing prices

Robustness test—one lag and two lags of independent variables

As the first check for the robustness of our results, we compare the results with one lag of the independent variables included to the results with two lags of the independent variables. Our main regression is listed again in column (1) of Table B-1. Column (2) includes a second lag of changes of median residential property prices, homicides, and nonhomicides. The magnitude of all coefficients increase under this specification, however their significance declines.

Still, the Wald test shows joint statistical significance at the 10 percent level, and just above the 5 percent level, for homicides. Nonhomicides are less significant, but still so at the 10 percent level. We chose to only include one lag in our main specification since we have a short unbalanced panel and the overall sample size is degraded sharply with the inclusion of additional lags.

Additionally, since we expect the second lag of differences is correlated with the third lag of levels, we can only include lags four deep and greater in our instrument matrix. Finally, with the second lags included, the AR(2) test for autocorrelation barely rejects the null hypothesis of no autocorrelation in levels under the two-lag specification. (see Table B-1)

TABLE B-1

Test of robustness—one lag and two lags of independent variables

	Specification 1	Specification 2[10]
Variables	$\Delta(\log(MedProp_{it}))$	$\Delta(\log(MedProp_{it}))$
$\Delta(\log(MedProp_{it-1}))$	0.737*** (0.0947)	1.0004*** (0.218)
$\Delta(\log(MedProp_{it-2}))$		-0.0632** (0.210)
$\Delta(Homicides_{it-1})$	-0.0152** (0.00684)	-0.0151* (0.0868)
$\Delta(Homicides_{it-2})$		-0.0247** (0.0114)
$\Delta(NonHomicides_{it-1})$	0.000260 (0.000192)	0.000235 (0.000425)
$\Delta(NonHomicides_{it-2})$		0.000820** (0.000379)
Observations	1,911	1,657
Number of geographic areas	254	253
Number of instruments	35	31
AR(1)	0	0.004
AR(2)	0.214	0.067
Hansen	0.256	0.759
Wald Test (homicides)	N/A	0.0541
Wald Test (non-homicides)		0.0878

* p < 0.1, ** p < 0.05, *** p < 0.01; Robust standard errors in parentheses

Source: Author's calculations

For our second test of robustness, we test for quadratic effects (Table B-2, below). Again, our main regression specification is in column (1). In column (2) we include a regression where differences of the squares of the crime variables are added as explanatory variables. The results show that the coefficients for these terms are small and the coefficients are insignificant. We conclude that quadratic effects have only little influence on our results. (see Table B-2)

TABLE B-2
Test of robustness–quadratic effects

	Specification 1	Specification 2[53]
Variables	$\Delta(\log(MedProp_{it}))$	$\Delta(\log(MedProp_{it}))$
$\Delta(\log(MedProp_{i,t-1}))$	0.737***	0.894***
	(0.0947)	(0.761)
$\Delta(Homicides_{i,t})$	-0.0152**	-0.0298**
	(0.00684)	(0.0223)
$\Delta(Homicides_{i,t-1})^2$		0.000695
		(0.000465)
$\Delta(NonHomicides_{i,t})$	0.000260	0.000445
	(0.000192)	(0.0003918)
$\Delta(NonHomicides_{i,t-1})^2$		-1.79e-07
		(1.35e-07)
Number of geographic areas	1,911	1,911
Number of instruments	254	254
AR(1)	35	51
AR(2)	0	0
Hansen	0.214	0.163

* p < 0.1, ** p < 0.05, *** p < 0.01; Standard errors in parentheses

Source: Author's calculations

The third robustness test we conduct compares the use of median residential property price data and average residential property price data, which were also available from DataQuick. Because DataQuick does not take steps to compare repeat transactions of similar homes, there is a chance that outliers exist in the dataset that they used to calculate average prices by geographic area. Since we have no means to measure the existence of outliers, we chose to use the median prices that they report rather than the mean prices since medians are relatively invariant to outliers. In Table B-3, below, we include in column (2), a regression with mean residential property prices. (see Table B-3)

TABLE B-3
Test of robustness–median and average property price data

	Specification 1	Specification 2[54]
Variables	$\Delta(\log(MedProp_{it}))$	$\Delta(\log(MedProp_{it}))$
$\Delta(\log(MedProp_{i,t-1}))$	0.737***	0.591***
	(0.0947)	(0.112)
$\Delta(Homicides_{i,t})$	-0.0152**	-0.0122**
	(0.00684)	(0.00566)
$\Delta(NonHomicides_{i,t})$	0.000260	0.000236
	(0.000192)	(0.000170)
Number of geographic areas	1,911	1,909
Number of instruments	254	396
AR(1)	35	35
AR(2)	0	0.001
Hansen	0.214	0.253

* p < 0.1, ** p < 0.05, *** p < 0.01; Standard errors in parentheses

Source: Author's calculations

Robustness test 4: One-step and two-step estimators

The two-step estimator is preferable to the one-step estimator in that it is robust to panel autocorrelation and heteroskedasticity, but the standard errors are biased downwards and so researchers have historically provided both one-step and two-step results. In our main specification, however, we resolve the downward bias by employing the Windmeijer (2005) finite-sample correction. We only include one-step results as a robustness check for the sake of convention, and they can be seen in column (2) of Table B-4, below.

TABLE B-4

One-step and two-step estimators

	Specification 1	Specification 2[1,2]
Variables	$\Delta(\log(MedProp_{it}))$	$\Delta(\log(MedProp_{it}))$
$\Delta(\log(MedProp_{it-1}))$	0.737***	0.740***
	(0.0947)	(0.0860)
$\Delta(Homicides_{it-1})$	-0.0152**	-0.0212**
	(0.00684)	(0.00614)
$\Delta(NonHomicides_{it-1})$	0.000260	0.000459***
	(0.000192)	(0.000196)
Number of geographic areas	1,911	1,911
Number of instruments	254	254
AR(1)	35	35
AR(2)	0	0.001
Hansen	0.214	0.189

* p < 0.1; ** p < 0.05; *** p < 0.01; Standard errors in parentheses

Source: Author's calculations

Robustness test 5: Depth of lags in the instrument matrix

Our fifth robustness check tests the sensitivity of our results to different depths of lags in the instrument matrix. Our main specification includes all lags greater than three deep, and is reported in column (1) of Table B-5 below. Column (2) reports a specification with all lags greater than four deep and the results are roughly similar. Column (3), however, includes all lags greater than two deep and shows quite a large difference. We attribute this to overidentification that occurs under this specification, which can be seen by examining the p-value of the Hansen test, which only barely rejects the null hypothesis of no overidentification. (see Table B-5)

TABLE B-5

One-step and two-step estimators

	Specification 1	Specification 2[2,3]	Specification 3[2,4]
Variables	$\Delta(\log(MedProp_{it}))$	$\Delta(\log(MedProp_{it}))$	$\Delta(\log(MedProp_{it}))$
$\Delta(\log(MedProp_{it-1}))$	0.737***	0.791***	0.718***
	(0.0947)	(0.114)	(0.0769)
$\Delta(Homicides_{it-1})$	-0.0152**	-0.0127**	-0.00346**
	(0.00684)	(0.00734)	(0.00166)
$\Delta(NonHomicides_{it-1})$	0.000260	0.000223	0.000057
	(0.000192)	(0.000202)	(0.0000683)
Number of zip	1,911	1,911	1,911
Number of instruments	254	254	254
AR(1)	35	32	38
AR(2)	0	0	0
Hansen	0.214	0.210	0.286

* p < 0.1; ** p < 0.05; *** p < 0.01; Standard errors in parentheses

Source: Author's calculations

Robustness test 3 – collapsed and uncollapsed instrument matrix

Our final robustness check compares a collapsed instrument matrix to one that is not collapsed. Our main specification, collapsed, is presented in column (1) of Table B-6 below. Column (2) displays the results when the instrument matrix is left uncollapsed. When uncollapsed the number of instruments explodes, going from 35 to 141. Correspondingly, the Hansen test indicates that the model is strongly overidentified. The coefficients and significance levels remain roughly similar, except on the lagged dependent variable for which the magnitude of the coefficient drops considerably. (see Table B-6)

TABLE B-6

Collapsed and uncollapsed instrument matrix

	Specification 1	Specification 2[ss]
Variables	$\Delta(\log(MedProp_{it}))$	$\Delta(\log(MedProp_{it}))$
$\Delta(\log(MedProp_{it-1}))$	0.737***	0.485***
	(0.0947)	(0.129)
$\Delta(Homicides_{it-1})$	-0.0152**	-0.0138**
	(0.00684)	(0.00430)
$\Delta(NonHomicides_{it-1})$	0.000260	0.000362***
	(0.000192)	(0.000135)
Number of geographic areas	1,911	1,911
Number of instruments	254	254
AR(1)	35	141
AR(2)	0	0.001
Hansen	0.214	0.224

* p< 0.1, ** p< 0.05, *** p< 0.01; Standard errors in parentheses

[ss] Arellano-Bond estimator

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47 These estimates of tangible victim costs, justice system costs, and criminal productivity costs for violent crimes are based on the per-offense cost and the incidence of offenses reported in each city in 2010 by the FBI's Uniform Crime Reports. Per-offense victim costs are based on direct out-of-pocket costs reported by victims of violent crimes in the NCVS surveys for 2006–2010. Per-offense police and judicial system costs are based on BJS data on U.S. justice system expenditures and arrest data from the FBI. Per-offense corrections costs are based on BJS data on the average cost per inmate in all U.S. jails and prisons, the number of convicted violent criminals in 2010, and the average incarceration time for criminals convicted of each type of violent crime. Estimates of criminal productivity costs are based on 2002 BJS survey data on the pre-arrest incomes of jail inmates, their average age at sentencing, and their average sentence served. Finally, justice system costs borne by federal, state, and local governments are derived from BJS data, which in turn come from the Census Bureau Annual Government Finance Survey and Annual Survey of Public Employment.

48 Jean Lemaire, "The Cost of Firearm Deaths in the United States: Reduced Life Expectancies and Increased Insurance Costs," The Journal of Risk and Insurance 72 (3) (2005): 359–374.

49 We assume here a one-to-one relationship between growth in local economic activity and growth in local tax revenues, based on extensive evidence on the sensitivity of local sales, business, and income taxes to local economic growth. See: Jesse Edgerton, Andrew F. Haughwout, and Rae Rosen, "Revenue Implications of New York City's Tax System," Current Issues in Economics and Finance Second District Highlights 10 (4) (2004): 1–7.

50 In this case, Granger causality testing is being performed with panel data, which can lead to panel bias if conducted using a standard OLS model. This occurs because the lagged dependent variable is endogenous to the error term. In other words, if in period one a geographic area experiences a negative shock to the dependent variable that is not explicitly modeled, then the error term will soak up the entire shock and the fixed effect for that geographic area will appear lower in every period. In period two, the lagged dependent variable and the fixed effect will both be lower, and so there is a positive correlation between a regressor and the error, and the model is inconsistent. To account for this issue, we explore the Granger causality between violent crime and property prices using the Arellano-Bond (1991) two-step linear generalized method of moments estimator, which was designed for dynamic panel analysis where there are few time periods but many individuals, or in our case, geographic areas. We implement the Arellano-Bond estimator using the STATA program xtabond2, developed by David Roodman and described in Roodman (2006). See, Manuel Arellano and Stephen Bond, "Some Tests of Specification for Panel Data: Monte Carlo Evidence and an Application to Employment Equations," Review of Economic Studies 58 (2) (1991): 277–297; David Roodman, "How to Do xtabond2: An Introduction to 'Difference' and 'System' GMM in Stata," Working Paper 103, Center for Global Development, 2006, available at http://repec.org/nasug2006/howtodoxtabond2.cgdev.pdf.

51 The difference transformation eliminates fixed effects, but the independent variables are still potentially endogenous, and so we draw instruments from within our dataset. Specifically, we assume all independent variables are potentially endogenous and so we include third and deeper lags of each independent variable in levels as GMM-style instruments. The year dummy variables are assumed to be strictly exogenous and included as IV-style instruments, or in other words we use the year dummy variables as their own instruments. We also employ small-sample adjustments, corrected standard errors, and we collapse the instrument matrix to avoid overidentification.

52 xtabond2 logMedPriceR L(1/1).logMedPriceR L(1/1).homicides L(1/1).nonHomicides yr*, gmmstyle(homicides nonHomicides logMedPriceR, lag(3 .)) ivstyle(yr*) twostep noleveleq nocons robust small

53 xtabond2 logMedPriceR L(1/1).logMedPriceR L(1/1).homicides L(1/1).nonHomicides yr*, gmmstyle(homicides nonHomicides logMedPriceR, lag(3 .)) ivstyle(yr*) twostep orthog noleveleq nocons robust small

54 xtabond2 logMedPriceR L(1/2).logMedPriceR L(1/2).homicides L(1/2).nonHomicides yr*, gmmstyle(homicides nonHomicides logMedPriceR, lag(4 .)) ivstyle(yr*) twostep noleveleq nocons robust small

55 xtabond2 logMedPriceR L(1/1).logMedPriceR L(1/1).homicides L(1/1).homicidesSq L(1/1).nonHomicides L(1/1).nonHomicidesSq yr*, gmmstyle(homicides nonHomicides logMedPriceR homicidesSq nonHomicidesSq, lag(3 .)) ivstyle(yr*) twostep noleveleq nocons robust small

56 xtabond2 logAvgPriceR L(1/1).logAvgPriceR L(1/1).homicides L(1/1).nonHomicides yr*, gmmstyle(homicides nonHomicides logAvgPriceR, lag(3 .)) ivstyle(yr*) twostep noleveleq nocons robust small

57 xtabond2 logMedPriceR L(1/1).logMedPriceR L(1/1).homicides L(1/1).nonHomicides yr*, gmmstyle(homicides nonHomicides logMedPriceR, lag(4 .)) ivstyle(yr*) twostep noleveleq nocons robust small

58 xtabond2 logMedPriceR L(1/1).logMedPriceR L(1/1).homicides L(1/1).nonHomicides yr*, gmmstyle(homicides nonHomicides logMedPriceR, lag(2 .)) ivstyle(yr*) twostep noleveleq nocons robust small

59 xtabond2 logMedPriceR L(1/1).logMedPriceR L(1/1).homicides L(1/1).nonHomicides yr*, gmmstyle(homicides nonHomicides logMedPriceR, lag(3 5)) ivstyle(yr*) twostep noleveleq nocons robust small

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Received from J. William Hirzy a package containing the following:

1. A cover letter from him to Acting Administrator Perciasepe

2. A TSCA section 21 petition and accompanying reference documents

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0.1% of the total city population and only 1.5% of the population under age 6 years.

Any attempt to correlate WLLs with incidence of EBL is also confounded by the fact that incidence of EBL in the US for children aged <6 years declined from 3.96% in 2000 to 2.0% in 2003 (*11*). This 1.96% decline is of the same order, or even higher, than the predicted 1.5% increase in Washington, DC due to the high WLLs. If the impacts of the high WLLs are to be quantified, methods that can account for the reduction in the national incidence of EBL must be considered.

For this work, additional modeling was conducted using the International Commission for Radiation Protection (ICRP) biokinetic model, to more precisely identify the population(s) most sensitive to lead in water. The ICRP biokinetic model has been successfully used to predict seasonal or weekly trends in BLLs (*12, 13*). We confirmed that the population most sensitive to EBL from high WLL is children aged ≤1 year consuming reconstituted infant formula. Moreover, the modeling indicates that some evidence of EBL due to consumption of formula in the first year of life should persist until age ≤30 months (Supporting Information 1). This result is consistent with expectations based on other research (*5*−*7*). Thus, children aged ≤1 year and children aged ≤30 months were selected as target populations for this research.

Blood Lead Data. *CDC Database.* A blood lead database from the 2004 CDC study (*3*) was obtained through the Freedom of Information Act.

Children's National Medical Center (CNMC) Blood Lead Database. A study of blood lead was reviewed and approved by the Institutional Review Board at Children's National Medical Center. The CNMC data containing >28,000 records from 1999−2007 were sorted and data for children aged ≤30 months were extracted. If there were multiple measurements of BLL for the same individual, a convention was followed in which the highest recorded blood lead for each child was retained and all other measurements were deleted (*14, 15*). This approach ensures that calculations of EBL incidence in the population are not skewed by multiple measurements from the same individual.

The 1999 CNMC data are treated differently in this work because no 1998 data are available. The convention of removing multiple blood lead measurements per child makes the 1998 data influential on the 1999 data set (children often have blood lead measurements at 1 and 2 years). Thus, with one exception, only CNMC data from 2000−2007 are used in this work.

Results

After discussing temporal trends in WLLs throughout the city from 2000 to 2007, the effects of WLLs on EBL for children aged ≤1.3 years are examined. Thereafter, a neighborhood analysis is presented for children aged ≤30 months.

Temporal Trends in WLLs in Washington, DC. The 90th percentile WLLs (Figure 1) increased after the switch to chloramine disinfectant in November 2000 (*1*). The exact point at which the WLLs began to rise after the switch in disinfectant cannot be precisely determined. Therefore, 2001 is considered a transition year and data are divided into halves (data from January to June 2001 are termed 2001a and data from July to December are termed 2001b). Other support for dividing 2001 in half is presented in Supporting Information 2. The 90th percentile WLLs remained higher than the EPA regulatory "action level" of 15 ppb from 2001 to 2004 (Figure 1) before dropping back below the action level in 2005. The drop in WLLs in 2005 is temporally linked to dosing of an orthophosphate corrosion inhibitor (from August 2004 onward) to mitigate high WLLs.

Following a January 31, 2004 front page *Washington Post* article that revealed the widespread problem with elevated

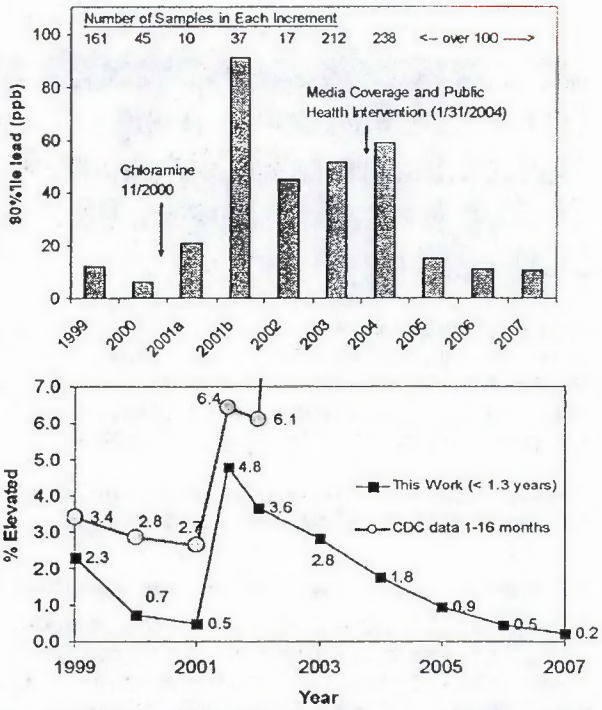

FIGURE 1. Temporal variation of lead in water (90th percentile water lead) and key events related to lead exposure in Washington, DC (top). Trends in EBL incidence for children aged ≤1.3 years (bottom).

WLLs, the public was eventually instructed to flush their water lines >10 min before collecting water for cooking and drinking. More than 20,000 lead filters were also mailed to homes with high risk of elevated WLLs in early 2004. Assuming these strategies were effective in largely abating human exposure to elevated WLLs, mid-2001 to early 2004 is the time period of greatest unprotected exposure to high WLLs.

Correlation Between EBL and WLLs for Children ≤1.3 Years of Age. Although the most highly impacted population is children aged ≤1 year there are insufficient data for this population group to support a statistically valid analysis. Only 0.62% of the overall CNMC data are for children aged ≤9 months and only 6.6% of the data are for children ≤1 year of age. The age group closest to the target population with adequate data (27% of the overall data) is children aged ≤1.3 years.

The incidence of EBL for children aged ≤1.3 years continued its decades long decline from 1999 through the first half of 2001 (Figure 1). But in the second half of 2001 the incidence of EBL abruptly increased by 9.6 times versus the first half of 2001. This 4.3% increase (from 0.5% to 4.8%) is not inconsistent with expectations presented in Supporting Information 1, especially considering that 90th percentile WLLs were higher in late 2001 than in 2003 (rough predictions in Table S1 are based on 2003 data). In 2002 and 2003, the incidence of EBL was ≥4 times higher than in 2000. In fact, EBL incidence did not return to levels observed in 2000 until about 2005, when lead in water once again met EPA standards. A proportions test in *R* (*16*) determined that the EBL incidence in the years 2001, 2002 and 2003 is greater than in 2000 with >95% confidence. A linear correlation between the incidence of EBL and the 90th percentile lead from 2000 to 2007 (see Supporting Information 5, Figure S7) is very strong (R^2 = 0.81).

The CDC database (*3*) was analyzed for the same trends. The incidence of EBL for children aged 1−16 months showed trends similar to the CNMC data (Figure 1, bottom). The absolute values of the CNMC data and the CDC data

Toxic Substances Control Act
Section 21 Citizens Petition
in RE: Hydrofluorosilicic Acid

Attn: Robert Perciasepe
Administrator (Acting)