The Social Implications of Evolutionary Psychology:

Linking Brain Biochemistry, Toxins, and Violent Crime[1]

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Introduction

Although recent neuroscientific research has revolutionized our understanding of brain function, studies in this field usually focus on the individual central nervous system (CNS). This emphasis has been necessary given the immense complexity of cytoarchitecture, neurochemistry, and function. Now, however, it is time to link our growing knowledge of brain function and evolutionary psychology to public policy. Such a linkage, with a particular focus on the links between neurotoxins and violent crime, shows the growing importance of evolutionary psychology, which—unlike earlier psychological theories—provides a solid framework for understanding new findings in neuroscience, toxicology, and behavior.

EVOLUTIONARY PSYCHOLOGY AND VIOLENCE

Evolutionary psychology teaches that human behavior needs to be understood in the perspective of hominid evolution and behavioral biology. In addition to describing the

repertoire of primate social behaviors as well as the likely developments associated with the appearance of hominids over the last 100,000 years, evolutionary psychology is open to insights from genetics, neuroscience, and ecology. As experience teaches us only too well, individuals differ in behavioral propensities for reasons that include genetic predispositions, personal experiences, and environmental contingencies.

Unlike classical behaviorism, for example, evolutionary psychologists recognize a species-typical repertoire of behavior that includes threat and aggression, as well as communication, bonding, sexuality, and other behaviors such as those linked with hunting and gathering. This approach, which integrates nature and nurture, facilitates analysis of the characteristic brain structures and neurotransmitter functions associated with distinct behavioral patterns in diverse situations. From this perspective, although it is important to understand the individual and environmental conditions that elicit particular behaviors, it is equally important to consider inhibitory processes. Even more important, by integrating ecological factors in behavioral analysis, evolutionary psychology makes it possible to reconsider how economic activities and public policies can modify the environment in ways that have unintended effects on individual behavior.

One promising area for such analyses concerns the harmful effects of toxins on brain chemistry and behavior (Gottschalk et al., 1991). Lead, for example, lowers intelligence and learning ability, as Ben Franklin learned from British printers.[2] More recently, neurotoxicologists have shown an association between lead uptake and poor impulse control, learning disabilities, and violence (Bellinger et al., 1994; Bryce-Smith, 1983; Cook et al., 1995; Cory-Slechta, 1995; Kahnet al., 1995; Minder et al., 1994; Needleman, 1989, 1999; Needleman & Gatsonis, 1991; Tuthill, 1996) In many instances,

exposure to lead and other toxins is due to human activities and can be exacerbated by governmental policies (Wollan, 1968). As a result, could differences in rates of violent behavior be traced to brain dysfunction that is made worse by ill-advised legal or bureaucratic decisions?[3]

From the perspective of evolutionary psychology, aggressive impulses and violent behaviors are part of the human behavioral repertoire. Among hominids, as in the social behavior of other primates, in addition to violent actions directed at potential predators, such behaviors sometimes occur between conspecifics. Although threat displays often occur within a band (especially in the context of behaviors that establish and maintain social dominance), within group bonding usually inhibits violent outcomes from aggressive interactions. In contrast, between group competition seems more likely to lead to a violent attack. On the one hand, aggressors may seek to deprive members of another band of access to crucial resources; on the other, individuals—and especially high-status males—sometimes respond to between-group threat with what has been classified as kin-based altruism. In short, from the perspective of evolutionary psychology, violent behavior is an element in the human repertoire that is normally inhibited within bonded groups but more likely to occur when directed to external threats to families or communities.

In a civilized society, the acts classified as "violent crime" represent a different form of aggressive behavior. Social norms and laws establish expectations that include those acts of within-group violence that are customarily inhibited by individuals experiencing aggressive impulses toward others. Consider two examples in terms of evolutionary psychology. First, I see a masked man approaching my house with a drawn

revolver at 8:00 P.M., take out my own gun, and shoot him as I open the door. This violent behavior could well be judged as an act of self-defense rather than a crime. Second, I see a salesman selling trinkets approaching my house at 2:00 P.M. on a sunny afternoon, take out my gun and shoot him as I open the door. In this case, I would probably be accused and convicted of murder. The first case is acceptable violent behavior if it can be judged under norms founded on the impulses of individual survival and defense of one's family. The second is violent crime if judged under norms that include civility to strangers and inhibition of aggressive impulses where no threat is involved.

From this perspective, when analyzing violent crime, evolutionary psychology can both clarify motives and—more important—explain the failure to inhibit aggressive impulses that contradict the law. And in addition to genetic predisposition and brain structure, recent research shows that the effects of toxins on neurotransmitter function are often a factor that can undermine normal inhibition of aggression (Masters, Hone, & Doshi, 1998). In the development of evolutionary psychology, this level of analysis may be especially important because it often reveals causal patterns that other psychological and sociological theories can neither predict nor explain.

BRAIN CHEMISTRY, ENVIRONMENTAL TOXINS, AND VIOLENT CRIME

Although the link between brain chemistry and violent crime may seem implausible, evidence that reduced exposure to toxins can lower the frequency of crime and other costly behaviors is provided by the Congressional ban on the sale of leaded

gasoline (Kitman, 2000).[4] In this case, the harmful effects of lead pollution from gasoline were apparently strongest during an infant's early neurological development. Although the correlation between each year's sales of leaded gasoline (as a measure of average exposure to fumes from tetraethyl lead) and that year's crime rate is virtually nil, the correlation rises sharply as the time lag between leaded gas sales and violent crime rates is extended; with a lag of 17 years, the correlation is over 0.90 (Table 2.1). Because children 17 years or younger rarely engage in violent crime, the very high correlation between lead gas sales and violent crime rates 18 to 26 years later points to fetal or neonatal exposure to lead as a significant but not generally noted factor in violent crime. As a result, these data suggest that the drop in U.S. homicide rates since 1991 was facilitated by the congressional ban on leaded gasoline (Masters, 2001).

Exploration of such questions is important because behavioral dysfunctions associated with neurotoxicity are often attributed to the individual's choice, education, or other personal defects. This tendency is noticeable even when the problem has been traced to a defect that is clearly beyond voluntary control. Several years ago, for instance, I presented a seminar on "Neuroscience and Learning" at the Harvard Graduate School of Education. At that time, three participants asserted that hyperactivity and other learning disabilities do not exist as CNS deficits but are merely "moral" failings of unruly children.

The consequence of the gap between neuroscientific findings and our educational system is often costly. In classes at Dartmouth College, it has not been unusual to discover about one student out of every ten with a previously undiagnosed learning disability. Indeed, when *Science* published an analysis of brain function among dyslexics

in three countries (Paulesu et al., 2001), the PET scans showing the brain loci not active among dyslexic children seem to have been—for some educators—the first concrete evidence that this condition has a basis in brain function.

Even where hyperactivity and learning disabilities are viewed as needing treatment, the neurological factors that might underlie each child's problem are often ignored. To be sure, a specific learning disability or behavioral problem may be traced to various factors. Among CNS characteristics that have been linked to hyperactivity (ADHD) are damage to a specific brain structure (the nucleus accumbens; Cardinal et al., 2001) as well as deficits in dopaminergic or serotonergic activity (Bellinger et al., 1994; Needleman & Gatsonis, 1991). Where neurotransmitter dysfunction is implicated, lead toxicity is often one of the factors involved (Brockel & Cory-Slechta, 1998).

Because hyperactivity due to a loss of impulse control can also be observed in violent behavior, the role of neurotoxins in ADHD deserves special attention. Although excessive cellular uptake of lead can be treated by chelation, teachers and physicians often give hyperactive children medications such as Ritalin without screening for known risk factors. In the United States alone, it has been estimated that as many as 11 million children are receiving Ritalin or other drugs that improve behavior by activating inhibitory circuits in the brain (such as dopaminergic pathways in the basal ganglia). For ADHD children, such medications provide a "quick fix" that masks underlying problems and creates a danger of long-term drug abuse from a "medication" that has effects parallel to those of cocaine (Walker, 1998). Indeed, journalistic reports that Ritalin has become a popular recreational drug underscore the need to adopt a more scientific approach to the analysis and treatment of learning disabilities or behavioral problems with an identified

neurological basis. Obviously, such uses of Ritalin can mask the problem and could actually increase the risks of violent behavior in later years.

Dealing with such issues is unlikely to be successful unless neuroscientific research is linked with the social dimensions of environment, individual behavior, and public policy. To illustrate the potential of such an approach, we here present evidence of the neurotoxic effects of two largely untested chemicals that are currently added to the drinking water consumed by 140 million Americans. These compounds—hydrofluosilicic acid (H_2SiF_6) and sodium silicofluoride (Na_2SiF_6)—are more generally called "silicofluorides" (SiFs).[5] Despite their widespread use, SiFs have never been properly tested for safety; as an EPA official put it, his agency has no evidence on "the health and behavioral effects" of silicofluorides.[6]

Because the public policy decisions responsible for this situation are not relevant for present purposes (Rymer, 2000), this chapter focuses on a series of questions that are essential in attempts to link neuroscience and evolutionary psychology to violent behavior. First, what characteristics of the suspected chemicals make the inquiry plausible and indeed necessary? (Part I: "Why Silicofluorides May be Harmful to Humans"). Second, based on known effects of these chemicals, what mechanism could trigger neurotoxic harm to humans? (Part II: "Biochemical Effects of Silicofluoride: Mechanisms of Neurotoxicity"). These two steps culminate in the description of biochemical mechanisms that are predicted to have specific biological and behavioral consequences, including increased risks of violence. Finally, given the research hypothesis developed to this point, is there empirical evidence consistent with the predicted effects? (Part III: "Testing the Hypothesis: Enhanced Lead Uptake and

Behavioral Dysfunctions Due to SiF"). As this outline suggests, in addition to building on research linking evolutionary psychology to neuroscience, analysis of this sort will also require knowledge of such disparate fields as chemistry, toxicology, and public policy.

WHY SILICOFLUORIDES MAY BE HARMFUL TO HUMANS

In the mid 1940s, the injection of sodium fluoride (NaF) in public water supplies was initiated in the United States as an experiment to ascertain whether rates of tooth decay would be reduced by fluoridated drinking water. In 1950, midway through a projected 10–12 year experiment, the U.S. Public Health Service -- allowed - the substitution of SiFs for NaF. Although tests had been conducted on NaF but not on SiFs, the implications of this shift have been generally ignored by both supporters and critics of public "fluoridation" of water supplies.[7]

Whereas NaF hydrolizes on injection into water, completely dissociating fluoride ion from sodium, no empirical evidence of dissociation rates of SiFs at 1 ppm was available when they were --judged acceptable -in 1950. At that time, the use of SiF was justified on the basis of a *theoretical* argument by P.J. McClure (of the Public Health Service) that the dissociation of SiFs would be "virtually complete."[8] Twenty-five years later, German laboratory studies by Westendorf revealed major differences between SiF and NaF. Under conditions comparable to those of a water treatment plant, SiFs are incompletely dissociated, and their residues have significant experimental effects on vital enzymes, including acetyl-cholinesterase (AChE) and serum cholinesterases (or

pseudocholinesterases), including butyryl-cholinesterase (BCh E) (Knappwost & Westendorf, 1974; Westendorf, 1975).[9]

Despite recent assertions of two EPA scientists (Urbansky & Schock, 2000),[10] this difference between NaF and SiF is consistent with other experimental findings. SiF anion $[SiF_6]^{2-}$ remains intact at pH 7 at room temperature. It must be exposed to boiling water at pH 9 in order to effect total fluoride release so that no residues of partially dissociated SiF remain in solution. Moreover, because the dissociation process is reversible, reassociation of SiF from its components is possible (e.g., when SiF treated water is used in cooking). Hence the assumed identity of NaF and SiF, which persists in many discussions of public health and dentistry (American Public Health Association, 2001; U.S. Department of Health and Human Services, 1000),[11] and was reinforced in the CDC's recent publication of a study group's "Recommendations" on Fluoridation,[12] can no longer be sustained without disconfirming existing research on these compounds.

When Westendorf set out to study SiF dissociation under more realistic conditions than had been tried previously, he used a refined technique. Measuring fluoride ion released from SiF at physiological conditions (pH 7.4, 37 ºC) in Ringer's solution at 1-5 ppm of total fluoride, Westendorf could only detect 67% of that fluoride with the fluoride ion specific electrode. He proposed that the remaining fluoride was still bound in a partially dissociated residue of SiF in the form of $[SiF_2(OH)_4]^{2-}$. Whether that particular species was the only SiF dissociation residue, Westendorf's finding was evidence for the survival of some partially undissociated SiF residue.

Translated into water plant parameters, Westendorf's findings would mean that dilution of SiFs to the 1 to 2 ppm level used in water fluoridation at the pH and

temperatures customarily obtaining in the water plant would induce each $[SiF_6]^{2-}$ ion to release only four fluorides to be replaced by hydroxyls. The concentration of resulting SiF dissociation residue $[SiF_2(OH)_2]^{2-}$ would be in the order of 1–5 ppm by weight. Incidentally, the same quantitative release of fluoride from SiF_4 would correspond with leaving behind the nonionic species $SiF_2(OH)_2$ at about the same concentration.

Thus, contrary to the total release of fluoride from SiF at water plant conditions (which has been assumed by supporters of fluoridation as a public policy),[13] Westendorf found only two-thirds fluoride release by actual experiment. Hence, at a pH close to common water plant practice, Westendorf's experiments show that SiFs are incompletely dissociated when injected in a public water supply and that the resulting residual complexes can have significant biochemical effects.

These characteristics of SiFs indicate that, in the absence of extensive testing of their safety, a harmful chemical may currently be distributed in the public water supplies of many communities. The scale of the potential problem is sufficient to justify concern, because over 90% of water fluoridation in the United States uses SiFs. With over 140 million Americans exposed to them (Centers for Disease Control, 1992), it is prudent to examine whether SiF residues or other harmful consequences of SiF injection in public water supplies (including the potential for reconstituting SiF in cooking or digestion) have neurotoxic effects that could modify behavior.

BIOCHEMICAL EFFECTS OF SILICOFLUORIDES AND MECHANISMS OF NEUROTOXICITY[14]

Enzymatic Inhibition

That SiF and NaF have different enzymatic effects was shown long before Westendorf completed his laboratory studies in 1975. In 1933, when reporting on his doctoral research, F.J. McClure (1933) reported that fluoride (in the form of NaF) can act as an enzyme inhibitor.

> Experimental evidence has established the fact that there is also a specific influence of fluorides on certain enzymatic changes associated particularly with carbohydrates and fats. Thus, the results of a systematic study conducted by Kastle and Loevenhart on the effect of antiseptics on the reactions of pancreatic and liver extracts revealed an effect of most substances and also a particularly remarkable destructive action of NaF on the reaction of lipase. . . . Dilutions of NaF as low as 1:15,000,000 [0.07 ppm] may inhibit the action of lipase on ethyl acetate as much as 50 per cent. . . . Leake et al have obtained evidence that NaF inhibits the action of this enzyme *in vivo*."[15]

Two years later (in 1935), Kick et al. found the excretion pathways of fluoride differ depending on whether test animals have ingested NaF or SiF (Kick et al., 1935).

Little additional work on the biological effects of these chemicals was conducted until Westendorf found that SiF inhibits AChE without a concentration threshold, whereas NaF inhibition of AChE starts at about 5 ppm of fluoride ion. Moreover, at equal fluoride levels beyond the NaF threshold level, SiF is about two to four times more powerful an inhibitor of AchE than NaF. The kinetics indicated that NaF inhibition was only competitive (i.e., worked by blocking the enzyme active site), whereas SiF inhibition was both competitive and noncompetitive. Competitive inhibition is explained

by the presence of hydrofluoric acid (HF), formed from free fluoride ion, which could find and occupy the active site in the enzyme molecule. That would occur whether inhibition was due to NaF or SiF, because both release free fluoride under physiological conditions at 1 ppm of fluoride. However, whereas NaF releases all of its fluoride ion by simple dilution/ionization, SiFs release fluoride ion in a complicated sequence of dissociation steps that depend on concentration and pH.

The chemical structures of likely SiF residues—$[SiF_2(OH)_4]^{2-}$ or $SiF_2(OH)_2$—would make each one a logical precursor for the creation of mono-silicic acid in the bloodstream. Mono-silicic acid is not a commonplace form of hydrated silica in blood and, according to the following hypothesis, has the potential for serious damage to health and behavior in a number of ways.

Residual Complexes Due to Incomplete Dissociation

A partially dissociated monomeric SiF species either survives into the stomach or is reformed there at gastric pH. It then passes into the bloodstream where it hydrolyzes to mono-silicic acid and/or forms low molecular weight silicic acid oligomers. These readily bind via their silanol hydroxyls to any polypeptide backbone with a reactable amine or hydroxyl. That alone would interfere with normal polypeptide structure and function. However, subsequent reaction of as-yet unreacted pendant silanols with one another would also create siloxane bonds or more linkages to the polypeptide backbone in such a way as to disrupt the natural chain folding of proteins.

A recent report (Coradin & Livage, 2001) amplifies this hypothesis and adds significantly to its credibility:

> The polymerization of silicic acid in aqueous solutions at different pH was followed by the colorimetric molybdosilicate method. The role of four amino acids (serine, lysine, proline and aspartic acid) and the corresponding homopeptides was studied. All four amino acids behave the same way and favor the condensation of silicic acid. Peptides exhibit a stronger catalytic effect than amino acids but they appear to behave in very different ways depending on the nature of side-groups and pH. Poly-lysine and poly-proline for instance lead to the precipitation of solid phases containing both silica and peptides. The role of these biomolecules on the polymerization of silicic acid is discussed in terms of electrostatic interactions, hydrogen bonds and solubility.

This report supports the proposition that silicic acid reaction with blood proteins could well be the root cause for SiF's powerful inhibition of AChE and "pseudo-cholinesterases" (PChEs), which are also known as "serum cholinesterases" and include butyryl-cholinesterase (BChE).

Effects of Cholinesterase Inhibition

The implications for human health of this SiF-induced biomechanism are numerous and in some instances can be extremely serious. One of the most important of these effects concerns the interference with cholinesterases. Although acetylcholinesterase (AChE) is known due to its regulatory role for acetylcholine, a neurotransmitter with multiple functions throughout the body, even today the role of

butyryl-cholinesterase (BChE) and its relationship to AChE is not entirely understood. According to Allderdice et al. (1991):

> Human tissues have two distinct cholinesterase activities: acetylcholinesterase and butyrylcholinesterase. Acetylcholinesterase functions in the transmission of nerve impulses, whereas the physiological function of butyrylcholinesterase remains unknown.

At least one function believed to be served by BChE is to protect AChE by scavenging toxins:

> Butyrylcholinesterase must be differentiated from acetylcholinesterase, which cannot hydrolyse succinylcholine. The physiological action of butyryl-cholinesterase remains unknown, although it can hydrolyse many drugs. (Lejus et al., 1998)

It is not inconceivable that the role of BChE as a protector of AChE goes beyond the capacity to hydrolyze drugs to a sacrificial role in absorbing heavy metals. In any case, powerful inhibition of BChE by SiF would indirectly modify an indirect impact on the proper function of AChE. Moreover, their interaction has been associated with brain dysfunction:

> Evidence about nonclassic functions of acetyl- (AChE) and butyryl-cholinesterase (BChE) during embryonic development of vertebrate brains is compared with evidence of their expression in Alzheimer disease (AD). Before axons extend in the early neural tube, BChE expression shortly precedes the expression of AChE. BChE is associated with neuronal and glial cell proliferation, and it may also regulate AChE. AChE is suggested to guide and stabilize growing axons.

> Pathologically, cholinesterase expression in AD shows some resemblance to that in the embryo. (Layer, 1995)

Regarding AChE inhibition, Westendorf found that fluoride released by NaF acted only in the competitive mode, but SiF had a much more powerful effect and acted in two modes. The first mode was competitive, as expected, due to the 67% of the SiF fluoride released as free fluoride. In addition, however, the nondissociated fluoride-bearing SiF residue enhanced net inhibition significantly in the noncompetitive mode. Westendorf suggested that the species $[SiF_2(OH)_4]^{2-}$ mentioned earlier somehow distorted the morphology of the AChE molecule, but he did not offer an explanation for how that occurred. Without referring to Westendorf's work at all, a hint of an explanation for this effect appeared in the English language literature a few years later (Margolis, 1976, as cited in Iler, 1979).

The "Margolis mechanism" discussed by Iler (1979) suggests how low molecular weight poly-silicic acid oligomers formed in the bloodstream could disrupt polypeptide chain morphology:

> The effect of silica was described by Margolis as due to the adsorption and denaturation of a globular protein, the Hageman factor. The proposed mechanism was that on sufficiently large particles or on flat surfaces of silica, the protein molecule was stretched out of shape by adsorption forces as it formed a monolayer on the surface. When the silica particles were very small, the molecular
>
> segments of the protein could become attached to different particles without segment stretching. . . . When protein is adsorbed on a larger silica particle or a

coherent aggregate of smaller particles, the chain stretched and certain internal hydrogen bonds which hold the protein molecule in a specific configuration are broken. On small single particles no such stretching occurs.

Any of the partially dissociated SiF species just described—for example, $[SiF_2(OH)_4]^{2-}$, SiF4, or $SiF_2(OH)_2$ derived from SiF_4—would be candidates for producing low molecular weight polysilicic acid oligomers in the bloodstream, after crossing over from the stomach at pH around 2. Most enzymes are globular proteins, so many enzymes besides AChE would be likely to experience at least noncompetitive inhibition by the "Margolis mechanism."

Ferry Molecules and Enhanced Heavy Metal Uptake

A wide array of nonenzyme polypeptides whose chain folding determines their function would also be subject to this morphological disruption. As a result, adverse effects of the partially dissociated SiF residue are not limited to adsorption by globular proteins or on flat surfaces. Given covalent bonding with any protein hydroxyl and amino sites by silicon-bound fluorine as described earlier, many other specific polypeptide morphology effects besides enzyme inhibition would also be susceptible to disruption.

Other mechanisms that enhance lead uptake or modify neurotransmitter function might also exist. For instance, if undissociated or reassociated SiF reaches the brain, its function as an AchE and BChE inhibitor could reinforce the effects of other cholinesterase inhibitors (such as organo-phosphate pesticide residues). Because Abou-Donia's experimental work shows that AChE inhibition has cumulative effects, this suggests that even relatively small residues might enhance the effect of other toxins in

this class (Abou-Donia, Goldstein, Dechovskaia, et al., 2001; Abou-Donia, Goldstein, Jones, et al., 2001).

It is especially noteworthy that Westendorf's SiF experimental data on incomplete dissociation are consistent with a biochemical mechanism that could enhance gut/blood lead transport and hence increase uptake of lead from environmental exposures. The compound Westendorf postulated as the partially hydrolyzed ionic species $[SiF_2(OH)_4]^{2-}$ closely resembles the $SiF_2(OH)_2$ molecule that we have proposed as a "ferry molecule" capable of chelating a heavy metal ion via the hydroxyls, with the enhanced ability to permeate lipophilic membranes due to the two residual fluorines. In addition, the two fluorines still bound to silicon at the 67% dissociation of SiF found by Westendorf could be due to survival of half hydrolyzed SiF_4 molecule, as well as to a two-thirds hydrolyzed $[SiF_6]^{2-}$.

If the strong noncompetitive enzyme inhibition by SiF found by Westendorf was the result of disruption of protein chain folding by low molecular weight polysilicic acid oligomers, a partly hydrolyzed SiF_4 molecule would be as likely to have that effect as the $[SiF_2(OH)_4]^{2-}$ anion. Defective protein morphology could result by the adsorption process suggested by Margolis or by covalent bonding between active silicon-fluorine bonds in partially dissociated SiFs with blood proteins.

The result could be the formation of molecules that can "ferry" a toxin such as lead to the brain or other organs, thus short-circuiting such natural detoxification enzymes as glutathione or metallothionines. Prior to Westendorf's research in Germany, although there was evidence that SiF had potentially harmful effects not found for NaF, there is little indication that American researchers were aware of this possibility.[16] The

shift from NaF to SiFs as fluoridation agents was endorsed in 1950, at which time no one could have known of Westendorf's findings (first partly revealed in 1974, when *Naturwissenschaft* carried a brief account of the findings more fully reported in Westendorf's thesis in 1975; Knappwost & Westendorf, 1974). The situation today differs due to the radical advances in neuroscience combined with the availability of extensive empirical evidence (including the English translation of Westendorf's thesis). Under these circumstances, it is now reasonable to test the hypothesis that children living in communities with SiF treated water are more likely to absorb lead from their environment and to exhibit behaviors that have been linked to lead neurotoxicity or cholinesterase inhibition. Because the Center for Disease Control monitors the chemicals used in water fluoridation, if geographic data are sufficiently precise these data can be used to test these hypotheses. Four types of data were available for statistical analysis: (1) the chemicals used for water fluoridation in each community; (2) children's blood lead levels from either state health surveys or the National Health and Nutrition Evaluation Survey (NHANES III), (3) socioeconomic and ecological data from the U.S. Census, and (4) rates of violent crime as reported by the Federal Bureau of Investigation (FBI). We began, therefore, by examining whether SiF usage is associated with an enhanced uptake of lead from such environmental sources as old housing with lead paint or high-lead levels in public water supplies (obviously, the absence of significant effects at this level would falsify the hypothesis). Then, having confirmed that blood lead uptake reflects something akin to the proposed "ferry molecules" or residual complexes due to SiF water treatment, we test whether the use of silicofluorides is associated with increased rates of

behavioral dysfunctions linked to blood lead, focusing on violent crime and substance abuse by criminals.

TESTING THE HYPOTHESIS: ENHANCED LEAD UPTAKE AND BEHAVIORAL DYSFUNCTIONS DUE TO SiF

To assess predictions of social phenomena based on neuroscientific and toxicological findings at the individual level, it is necessary to examine aggregate data with care. Geographically diverse samples of individuals need to be studied using multivariate statistical techniques to control for the effects of potentially confounding variables. More than one sample should be studied, and samples should be large enough to insure that tests of statistical significance are meaningful. For any one sample, moreover, it is useful to analyze the data in more than one way, using different statistical techniques (such as multiple regression, logistic regression, and analysis of variance) and examining subsamples to explore the incidence of observed effects among individuals of different race, age, or sex. Finally, but of particular importance, it is important to examine aggregate data *both* for a biological effect known to influence behavior (e.g., levels of blood lead as a test of uptake of a dangerous neurotoxin) *and* for behaviors that might have been made more likely by the toxin (e.g., substance abuse and violent crime).

Multiple analyses are therefore necessary to test the hypothesis that SiF-treated water exposes individuals to residues that enhance lead uptake (such as the “ferry molecules” described above) and thereby increase rates of behavioral dysfunction. As an

illustration of the methodological problems facing any such endeavor, at least four distinct empirical issues need to be addressed:

° Population samples should provide evidence of biological differences between those exposed and not exposed to the presumed source of neurotoxicity. In the present case, *do children living in communities with SiF-treated water have, controlling for other variables, higher blood lead levels?*

° These effects should include evidence consistent with the presumed mechanism. In the present case, *does exposure to SiF increase the risks of high blood lead from such known environmental sources of lead as old housing and lead levels over 15 ppb in public water supplies?*

° The effects should occur among different types of individuals—and, insofar as there is variation by population subgroups, the differences should correspond with previously known variations. In the present case, *how does SiF exposure affect blood lead levels among children of different races and ages—and, in particular, how do these effects relate to the generally higher blood lead levels usually found among blacks in the United States?*

° Behaviors previously linked to the toxins in question should be more frequent in times and places where the environmental problem of interest is present. In the present case, *are rates of crime and substance abuse higher in communities using SiF than in comparable localities whose water is not treated with these chemicals?*

The first three questions will be explored using several geographic samples for which we have data on children's blood lead levels (usually based on samples of venous blood

lead as well as capillary blood lead). First, for the state of Massachusetts, we have a data from capillary blood lead tests of children in 213 communities (constituting virtually all localities with a population over 3,000, including all but one of the communities using SiF-treated water).[17] This sample provided data for approximately 280,000 children, and was analyzed both for all 213 towns and for venous blood lead measurements in a subset of 76,566 children from 30 communities with and 30 communities without SiF treatment (Masters & Coplan, 1999a). Second, for the state of New York, we studied a sample of venous blood tests from 151,225 children in 103 communities with populations between 15,000 and 75,000 (Masters et al., 2000). Finally, we examined blood lead data for almost 4,000 children in the National Health and Nutrition Evaluation Survey III (NHANES III) who lived in 35 counties with populations of over 500,000 (Masters et al., 1999).

Whereas the first two of these samples had data by community, permitting unambiguous evidence of whether children were exposed to SiF, the NHANES III data (only available by county) were divided into counties with less than 10% of the population exposed to SiF, between 10% and 80% exposed to SiF, and more than 80% exposed to SiF. For most purposes, the best assessments here were a contrast between counties with less than 10% SiF exposure (on aggregate, about 6% of children in this category drank SiF-treated water) and counties with over 80% exposure (on aggregate, 92% of children in this group drank SiF-treated water).

For an epidemiological study of behavioral outcomes, we can then use national FBI county-level data for rates of violent crimes. This makes it possible to compare counties for the effects of industrial lead pollution and SiF-treated water while controlling

for socioeconomic and demographic factors using census data. For substance abuse, a sample of over 30,000 criminals in 24 cities studied by the National Institute of Justice (NIJ) was assessed for the association between cocaine use at time of arrest and age of first substance abuse. Although further studies are desirable, it should be evident that these datasets are sufficiently diverse to provide a reasonable test of the twin hypotheses that SiF-treated water contains residues (such as the postulated "ferry molecules") which enhance lead uptake, and that the resulting neurotoxicity is associated with costly behavioral dysfunctions.

Higher Blood Lead Levels Where Silicofluorides Are in Use

In Massachusetts communities using SiF, children's average blood lead levels were higher and the probabilities of a level over 10μg/dL were greater:

> 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 = .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 = .0006; DF 3, 209, F 6.073). The prevalence rate of individuals whose capillary blood lead was above the maximum permissible level of 10μg/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 < .0001; DF 3,212, F 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.[18]

Overall, roughly four times as many SiF-treated communities as nonfluoridated or NaF treated communities have over 3% children with blood lead over 5μg/dL. Moreover, these effects are evident where environmental lead sources are below average, but they are exacerbated when lead levels in water or the percent of old houses are above average. For instance, in communities using sodium fluoride where first draw lead in pubic water exceeded 15 ppb, average blood lead levels were actually lower (1.9μg/dL) than in communities using this chemical with less lead in their water (2.11μg/dL). In contrast, in 25 communities using fluosilicic acid with over 15 ppb lead in water, children's blood lead averaged 3.27μg/dL compared with only 2.31μg/dL in 26 communities using fluosilicic acid where lead in 90th percent first draw water was under 15 ppb. Effects in a smaller number of communities using sodium silicofluoride were comparable, with blood lead averaging 4.38μg/dL where first draw lead was above 15ppb (n = 1) compared with 2.37 where lead in water was under 15 ppb (n = 6).[19] (For further analysis of the hypothesis that SiF residues enhance uptake of lead from environmental sources such as old housing or lead in public water supplies, see next section.)

The association between SiFs and higher blood lead was confirmed by comparing a subsample of 30 nonfluoridated Massachusetts communities with 30 matched communities using SiF (Table 2.2). Here, although the SiF-treated towns had 50% more lead in public water supplies, more poor, and more minorities, they also had slightly

higher per capita income, higher elementary school budgets, and a larger percentage of college graduates. None of these differences fully explain why 1.94% of screened children had blood lead levels in excess of 10μg/dL where SiF was in use, whereas only 0.76% had such high blood lead in the comparable nontreated towns.

New York data are consistent with an association between the use of SiF and higher venous blood lead levels among children. Overall, the percentage of children with venous blood lead over 10μg/dL was significantly higher (DF 3, 104, $F = 9.13$, $p = .0001$) if water was treated with fluosilicic acid (4.52%) or sodium silicofluoride (4.20%) than if water was untreated (3.78%) or treated with sodium fluoride (3.05%). Among blacks tested, 20.6% of the 8,685 exposed to SiF had venous blood lead over 10μg/dL, whereas only 7% of the 9,556 blacks in non-SiF communities had blood lead at this level (with similar effects at different blood lead level cutting points) (Masters et al., 2000, p. 1093). Although communities using SiF had somewhat higher levels of seven risk factors associated with higher blood lead (Table 3.3), these sources of lead uptake do not fully explain the results; on the contrary, as hypothesized, SiF enhances lead uptake from environmental sources and hence increases the odds of high blood lead even more where these factors are present (see next section).

Data from the Third National Health and Nutrition Evaluation Survey (NHANES III) were only available for the subset of about 4,000 children living in 35 counties having populations of over 500,000. Using the CDC's 1992 Fluoridation Census, the percent of each county's population receiving silicofluoride-treated water was calculated, and each county was assigned to one of three groups. As noted, 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, the association between more SiF usage and elevated blood lead was statistically significant ($p < 0.001$), with high/low risk ratios in the range of 1.5 to 2.0, depending on age and race.

Enhanced Uptake of Lead from Environmental Sources

We have predicted that the lead uptake from environmental sources of lead is significantly higher where SiF-treated water exposes children to residues, including compounds like the suggested "ferry molecules." As a result, mere association between SiF usage and higher blood lead levels is insufficient to test the research hypothesis. Two-way or three-way analysis of variance (ANOVA), which simultaneously considers the relative association between several predictive variables, can also indicate whether the combination of two or three of these predictors (as measured by the "interaction term" of the ANOVA) has significantly stronger effects than the sum of their independent effects. Our hypothesis predicts significant interaction terms between SiF usage and such environmental risk factors as lead in public water supplies or paint in old housing. Conventionally, when a two- or three-way ANOVA has a significant interaction term, statisticians often give weight to the results because such effects are rarely due to measurement error in one of the variables.

The data from Massachusetts (Masters & Coplan, 1999a) are clearly consistent with the research hypothesis that SiF-treated water carries residual complexes, including "ferry molecules" that enhance lead uptake from the environment:

> When both fluoridating agents and 90th percentile first draw lead levels are used as predictors of lead uptake, the silicofluoride agents are only associated with substantially above average infant blood lead where lead levels in water are higher than 15ppm. This interaction between the use of silicofluorides and above average lead in water as predictors of children's lead uptake is statistically significant (p = .05; DF 3,204, F 2.62). To confirm this effect, we assessed the extent to which silicofluoride usage 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 □g/dL) in the 26 towns not using these agents.[20]

These effects show a tendency for SiF to increase the harmful effects of known risk factors of blood lead uptake that were consistently found in analyses of other samples.

To assess the overall vulnerability of those in high-risk environments in the New York sample, we assigned to each individual a value indicating whether his/her community was above or below the median for each of the seven covariate risk factors in Table 3.3. We then used these as covariates in our analysis, dividing the sample of

individuals into those who live in communities with four or fewer risk factors and those who live in communities with five or more risk factors. Although exposure to five or more risk factors increases the risk of blood lead above 10μg/dL, exposure to this number of risks where SiF is used more than doubles a child's chance of having elevated blood lead. As is shown below, these effects were confirmed by computing age-adjusted logistic regressions of odds ratios for venous blood lead over 10μg/dL for children living in communities using SiF compared with those not using these chemicals (Figures 1 and 2 in Masters et al., 2000, p. 1095).

The NHANES III data are less useful for such statistical analyses due to smaller sample size and organization of data by county (which makes it difficult to assume that a high level of an environmental variable applies to each child in a given county). Such limitations reinforce the importance of assessing interaction effects in different racial and age groups of children.

SiF Exposure and Blood Lead Levels among Children of Different Races and Ages

Prior studies have generally shown that minorities—and especially blacks—are particularly at risk for high levels of blood lead. NHANES III data, showing average blood lead levels for black, Hispanic, or white children aged 3–5 (Figure 2.1) and 5-17 (Figure 2.2) provide a useful urban sample. For each race and each age, lead levels are significantly higher for children exposed to SiF-treated water ($p < .0001$), with effects of

exposure to SiF that are significantly worse for minorities than for whites, and worse for blacks than for Hispanics.

Because a similar effect had already been noted for children in our New York State sample, we sought a more precise measure of the impact of SiF-treated water on environmental factors associated with higher blood uptake for blacks as compared with whites. For white and black children living in towns above and below the median for each risk factor, we computed the odds ratio for higher blood lead among those exposed versus not exposed to SiF-treated water (1.0 equals chances that are 50–50 whether water does or does not have these chemicals). Logistic regression was used to assess these odds ratios. The results show that SiF-treated water consistently increases the odds of high blood lead, but that this effect is exacerbated where risk factors for high blood lead are above average. Moreover, as seen in other statistical tests, this enhancement of environmental risks by SiF is much greater for black children than for whites.

In the Massachusetts sample, the vulnerability of black children is also evident. When data are analyzed by community, although average blood lead levels are significantly higher where silicofluorides are in use, average blood lead was substantially higher where blacks comprise a larger proportion of the population (Figure 2.3). Consistent with established findings, higher blood lead levels are also found in communities with an above average proportion of pre-1940 housing (where lead paint is often found) and in communities with more blacks in the population. When silicofluoride use is added to the analysis, however, the higher levels of children's blood lead usually associated with communities with larger black populations is *only* found where there are *both* more older housing *and* silicofluorides in water treatment (Figure 4 in Masters et al.,

2000). From this perspective, the enhanced lead uptake due to exposure to silicofluoride-treated water seems to be a critical factor explaining high blood lead among American blacks.

In the New York sample, the vulnerability of blacks is also evident from the effect of exposure to SiF on the proportion of children with various blood lead levels (Figure 2.4). Virtually all black children in the New York sample with blood lead levels of 10–15μg/dL or 15–20μg/dL lived in SiF communities. In contrast, blacks with less than 5μg/dL of blood lead were less likely to live in SiF communities. Although it has long been noted that blacks tend to be more vulnerable to lead uptake (due to characteristics such as low calcium in diet, which is perhaps associated with lactose intolerance), SiF water treatment increases this risk substantially.

Data from the NHANES III sample also are consistent with this effect. In the counties with fewer percent living in poverty and where silicofluorides are NOT in use, there is virtually no difference between the average blood lead levels of whites (3.62μg/dL) and blacks (3.90μg/dL). For similar counties with silicofluoride use, blood lead in white children averages 4.62μg/dL, whereas it is 5.95μg/dL among blacks. Similar increases occur in the counties with above average poverty: in both environments, blacks are affected more strongly than whites by SiF-treated water. Hence a two-way ANOVA for the sample as a whole shows that SiF treatment is a significant predictor of higher blood lead ($F = 6.63$, $p = .0042$), whereas community poverty is not significant ($F < 1$).

Similar results for the increased lead from environmental risk factors in Massachusetts indicate that the harmful effects of SiF-treated water are not primarily due

to toxins in the SiF delivered to water treatment plants (Masters et al., 2000). Rather, mechanisms like that of the postulated ferry molecule or other residual complexes from SiF apparently increase the uptake of lead from old housing and from lead in public water supplies. Because the policy of water fluoridation has been justified by the poor dental health of minorities, it is ironic that the principal chemicals used for this purpose seem to have especially deleterious effects on blacks and other minorities.[21]

[@B]Increased Violent Crime and Other Behavioral Dysfunctions

Because lead is a neurotoxin that lowers dopaminergic function in the inhibitory circuits of the basal ganglia, it is not surprising that researchers have repeatedly found that higher bodily burdens of lead are linked to increased rates of violent crime (Stretesky & Lynch, 2001). Individual data to this effect imply that ecological data ought to show that communities with industrial lead pollution are associated with higher rates of violent crime. Such research reveals effects at the *social* level and illustrates how governmental decisions could improve human health and welfare by reducing the impact of environmental poisons.

Geographic variations in violent behavior had been analyzed before our research turned to SiF. Because data on individual offenders had indicated that violent behavior could be linked to the toxic effects of lead or manganese, crime rates in 1991 were compared for all U.S. counties with or without EPA-reported toxic releases of either of these heavy metals. Using aggregate data for all U.S. counties, both heavy metals significantly contribute to higher rates of violent crime, with a significant “interaction” effect showing that the combination of lead and manganese has a stronger effect than the sum of each toxin separately. With counties as the unit of analysis, multiple regression

equations, including other factors associated with crime, such as poverty, unemployment, and race, indicate that lead pollution was probably an *additional* contributory factor in 1991 crime rates (Table 2.4).

It is logical to predict that if lead pollution is a factor in violent crime, and SiF increases the uptake of environmental lead, then using SiF in water treatment should be associated with higher rates of violent crime. Using a multiple regression model including both lead and manganese pollution (as measured in the EPA's Toxic Release Inventory) and percent of county receiving SiF-treated water, as well as socioeconomic and demographic factors linked to violent behavior, this prediction was tested for 1985 rates of violent crime in all U.S. counties (Table 2.5). The results show not only that SiF usage is a significant additional factor for higher crime rates, but that once SiF is included in the analysis, toxic releases of lead and manganese are no longer significant predictors of county-level violent crime rates. Consistent with this analysis, although crime rates are always increased by industrial releases of manganese, the national data show that this effect is aggravated where silicofluorides are used (Figure 2.5).

Because the choice of variables in a multiple regression model can sometimes influence the outcome, a slightly different set of variables was used in regression equations to predict county level rates of violent crime in both 1985 (Table 2.6) and 1991 (Table 2.7). In both cases, SiF is a significant predictor of violence. Moreover, the contrast between Tables 2.4 and 2.5 indicates that, where SiF is not used in public water supplies, industrial pollution with either lead or manganese has a much weaker impact on violent crime rates. This finding is consistent with the evidence that SiF enhances heavy metal uptake by biochemical mechanisms like those outlined earlier.

Other population-level tests of behavioral harm due to silicofluoride usage are limited by the lack of reliable measures of conditions such as hyperactivity (ADHD) which have been linked to lead toxicity. An exception, however, is an NIJ study of substance abuse by violent offenders. This study recorded the age of first use of alcohol and drugs as well as drug use at the time of arrest for a sample of over 30,000 criminals from 24 cities. Such data are especially relevant because BChE has recently been found to "accelerate cocaine metabolism in such a way as to potentially lessen the behavioral and toxic effects of cocaine" (Carmona et al., 2000).[22] As a result, BChE inhibition by SiF residues would increase the effect of cocaine, leading to the prediction that drug use would be more pronounced among violent offenders in cities that inject SiF in public water supplies.

Once again, the data are consistent with the hypothesis. In the NIJ sample, controlling for the percent of blacks in the population (which by itself is never significant), use of SiFs was significantly associated with the average age of the first use of alcohol ($p = .06$), of PCP ($p = .0155$), and of crack ($p = .027$) (Masters & Coplan, 1999b). Moreover, the age of first use of alcohol, crack, or cocaine is significantly associated with rates of violent crime (in each case, $p < .0001$), and crimes rates are significantly higher in the 13 sampled cities using fluosilicic acid (2,123 per 100,000) or the 6 cities using sodium silicofluoride (1,704 per 100,000) than in the 5 cities not using SiF (1,289 per 100,000) (Masters & Coplan, 1999b).

As a check, rates of drunken behavior per capita were analyzed in our county dataset. To illustrate yet another statistical technique, step-wise regression was used: The best of a set of predictor variables was identified and the variance it accounted for was

removed, then the next best predictor of the remaining variance was identified, and so on until no additional significant variables remained. In this analysis, SiF was one of seven variables that significantly predicted rates of drunken behavior whereas five variables (including the EPA's Toxic Release Inventory for lead and manganese) had no significant effect on county level rates (Table 2.8).[23]

In all samples studied, therefore, we found evidence that the behavioral effects of SiF residues increase rates of costly behaviors that have previously been linked to lead. As a result, the evidence suggests that a moratorium on the use of SiF in public water supplies would be a relatively low-cost policy capable of lowering rates of substance abuse and violent crime. Indeed, given indications that hyperactivity is often linked to lead toxicity, such an initiative might also reduce learning disabilities and improve educational outcomes.

CONCLUSION

The foregoing analysis, like the controversy over lowering the permissible levels of arsenic in American public water supplies, suggests that conflicts between science and public policy may be of increasing importance in coming years. In such issues, the central concern has hitherto been cancer and other mortal diseases. As our analysis shows, it is now time to link neuroscience, evolutionary psychology, and toxicology to such social behavior as violence. Just as the ban on leaded gasoline seems to have lowered rates of violent crime since 1991, other initiatives may have substantial benefits by reducing the risks of dysfunctional behavior caused by toxins.

To illustrate a policy derived from this approach, I have proposed a moratorium on injecting fluosilicic acid or sodium silicofluoride in a public water supply until extensive testing proves their safety. Such testing is especially necessary for chemicals that are distributed to the general public in a manner not subject to individual choice. Moreover, because prudent policy initiatives need to consider costs as well as benefits, the use of untested chemicals cannot be justified merely on the presumed benefit to a single medical condition. It must be stressed that this proposal only concerns the use of fluosilicic acid or sodium silicofluoride in water treatment. Although there is much controversy over the costs and benefits of water fluoridation using sodium fluoride as well, our data do not indicate that NaF is a major factor in enhancing children's blood lead levels.

In comprehensive cost-benefit analyses of chemicals in our environment, behavioral harm may often be more costly or more widespread than cancer and other mortal diseases. As neuroscientists and evolutionary psychologists unravel biological factors in human social behavior, scientists and policymakers in other fields can no longer ignore the costs of learning disabilities, substance abuse, or criminal behaviors that have often proven resistant to traditional treatments or governmental policies based on sociological and economic theories of behavior. In the era of Prozac, Ritalin, and brain imaging with PET and other technologies, ignoring the revolutionary advances of neuroscientific research is neither prudent nor reasonable.

NOTES

1. Research on silicofluoride toxicity described in this paper has been conducted in collaboration with Myron J. Coplan (Intellequity Consulting, Natick, MA), whose expertise in chemical engineering and the history of fluoridation has been essential. Former vice president of a multinational firm, Coplan's experience includes direct professional work with silicofluorides, as well as numerous areas of public policy. Our collaboration reflects the extent to which research on many issues linking environmental toxicity, brain chemistry, and public policy can no longer be conducted by a solitary researcher.

2. See the letter from Benjamin Franklin to Benjamin Vaughan "on the bad Effects of Lead taken inwardly" (31 July 1786), in Lemay (1987).

3. For the example to be discussed below, see the Web site http://www.dartmouth.edu/~rmasters/ahabs.htm.

4. This article includes especially revealing on the origins of adding tetrethyl lead to gasoline with knowledge of the toxicity of these additives—and on the continued sales of these products in the Third World.

5. See Myron J. Coplan and Roger Masters, "Should Silicofluoride Be Used to Fluoridate Municipal Water?" Submitted to Congressman Kenneth Calvert, Chair of Subcommittee on Energy and the Environment, Committee on Science, U.S. House of

Representatives. April, 2000; idem, Response to EPA Staff Unsupportable Dismissal of Evidence of Adverse Silicofluoride Health Effects. Report to EPA, June 12, 2000; idem, Scientific Misconduct at EPA. Report to Hon Kenneth Calvert, Chair of Subcommittee on Energy and the Environment, Committee on Science, U.S. House of Representatives, September 25, 2000.

6. "To answer your first question on whether we have in our possession empirical scientific data on the effects of fluosilicic acid or sodium silicofluoride on health and behavior, our answer is no. . . . We have contacted our colleagues at NHEERL and they report that with the exception of some acute toxicity data, they were unable to find any information on the effects of silicofluorides on health and behavior." Robert C. Thurnau (Chief, Treatment Technology Evaluation Branch, Water Supply and Water Resources Division, U.S. EPA National Risk Management Research Laboratory, Cincinatti, OH) to Roger Masters, November 16, 2000.

7. For example, in 1951, a principal proponent of extending water fluoridation—Francis Bull—explicitly told a dental convention never to mention the chemicals to be used and admitted he had no evidence on toxicity. This practice has persisted. For instance, in the recent report on Oral Health in the United States—2000, Surgeon General Satcher speaks of "fluoridation" without mentioning the chemicals used. With few exceptions, critics of water fluoridation have long addressed the issue in similar terms.

8. Years later, several experiments were published that purported to confirm this prediction, but the studies used an ion specific electrode method that required conditions unlike those of a water treatment facility and the reported results rounded figures to the nearest whole number (thereby hiding evidence of incomplete dissociation).

9 An English translation of Westendorf's doctoral dissertation is available at http://www.dartmouth.edu/~rmasters/slub.htm. To access, follow instructions at the end of the "forward" to the translation by Jakob von Moltke. Although this research seems to have escaped the attention of U.S. health authorities, it provides important evidence that SiF-treated water is not "just like" NaF treated water that has not been taken into account by either critics or supporters of water fluoridation.

10. Available on the Web at http://fluoride.oralhealth.org/papers/urbansky.pdf.

11. National Institutes of Health, Transcript of Proceedings, Surgeon General's (Koop) Ad Hoc Committee on "Non-Dental Health Effects of Fluoride," Day I (April 18, 1983), Bethesda, MD: Stenotech, Inc., 1983), I, esp. 132–139 (Dr. Frank Smith's description of the experimental studies of "fluoride absorption" and "fluoride in blood" without reference to specific chemicals to which research animals were exposed).

12. Fluoride Recommendations Work Group, "Recommendations for Using Fluoride to Prevent and Control Dental Caries in the United States" (2001), available at http:www.cdc.gov/mmwr/. See also CDC Press Release "CDC releases new guidelines of

fluoride use to prevent tooth decay" (2001), available at http://www.cdc.gov/od/oc/media/pressrel/4r010817.htm.

13. For a detailed critique of Crosby's methodology, including his selective use of rounding to whole numbers to hide incomplete dissociation, see Myron J. Coplan's correspondence with the APHA, available at http://www.dartmouth.edu/~rmasters/ahabs.htm.

14. For a fuller analysis of this topic, from which the following section is adapted, see Myron J. Coplan, Reply to APHA Oral Health Section Objections to Proposed APHA Resolution (July 2000).

15. It should be noted that the fluoride level in this experiment was far lower than 1 ppm.

16. By the same token, though McClure was interested in amyulase inhibition by fluorides, there is no indication that he was aware of fluoride inhibition of AchE (McClure, 1939).

17. We thank Adrian Bailey and James Sargent for making available to us these data, for which they previously showed the role of lead residues from industrial activities (Bailey et al., 1994).

18. Regarding Masters and Coplan (1999), pp. 440–441: A footnote added to this passage indicates, “Towns using sodium fluorosilicate reported lower first draw water lead values (11.7 ppb) than unfluoridated towns (21.2 ppb) or towns using sodium fluoride (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 ($p < .0001$, 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).”

19. Regarding Masters and Coplan (1999), it should be noted that the increment in average children’s blood lead from use of sodium fluoride (compared with unfluoridated water) is relatively small (0.14μg/dL) if lead levels in 90th percentile first draw water are under 15 ppb; if lead levels in water are above 15 ppb, children’s blood lead levels are actually 0.28μg/dL lower where sodium fluoride is used than where water is not fluoridated. In contrast, where lead in the water is above 15 ppb, the increment compared with nonfluoridated communities is 1.09μg/dL for the 25 communities using fluosilicic acid and 2.2μg/dL in the one community using sodium silicofluoride. Put another way, comparing the effect of fluoridating water with silicofluorides on uptake of lead above 15 ppb in the public water supply rather than below that level is roughly three or four times worse if the chemical agent is a silicofluoride rather than sodium fluoride (Table II, p.

443). Analysis of variance shows that this effect, measured as the interaction between silicofluorides and lead levels in water over 15 ppb, is statistically significant: $p = .042$; $F = 4.18$ (Figure 1, p. 444).

20. In Masters and Coplan (1999), after controlling for other sources of lead, silicofluoride usage remained significant, which (a footnote adds) "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)."

21. The January 2002 issue of *American Journal of Public Health* had two articles germane to this issue. On prejudices and discrimination in the delivery of dental health care to poor minority children, see Mofidi et al. (2002, pp. 53–58). For the serious epidemic of dental health among blacks in Harlem, where water is treated with silicofluorides, see Zabos et al. (2002, pp. 49–52).

22. The entire abstract of this study is worth citing:

> Butyrylcholinesterase (BChE) is known to metabolize cocaine in humans. In the present study, three different experiments were performed to determine whether the addition of horse serum-derived BChE would accelerate the metabolism of cocaine. In the first experiment, the addition of BChE to squirrel monkey plasma in vitro reduced the half-life of cocaine by over 80%, decreased the production of the

metabolic product benzoylecgonine, and increased ecgonine methyl ester formation. The effect of BChE on cocaine metabolism was reversed by a specific BChE inhibitor. In the second, in vivo, experiment, exogenously administered BChE reduced peak cocaine concentrations when given to anesthetized squirrel monkeys. Finally, incubation of cocaine with added BChE in human plasma in vitro resulted in a decrease in cocaine half-life similar to that observed with squirrel monkey plasma. The magnitude of the decrease in cocaine half-life was proportional to the amount of added BChE. Together, these results indicate that exogenously administered BChE can accelerate cocaine metabolism in such a way as to potentially lessen the behavioral and toxic effects of cocaine. Therefore, BChE may be useful as a treatment for cocaine addiction and toxicity.

23. In a stepwise regression for violent crime for 1991, percent SiF was fifth of seven variables removed (with a stronger standardized coefficient than either –pe capita income or population density); percent SiF also was a significant predictor in 1990, 1992, 1994, and 1995.

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Table 1

Correlations Between Gasoline Sales and U.S. Violent Crime Rates Lagged by Increasing Time Intervals (1976- 1997) (Source: FBI, Supplementary Homicide Reports, 1976-97)

Year lag	Correlation	n	Year Lag	Correlation	n
0	-0.906	26	25	0.910	24
1	-0.897	27	26	0.900	23
2	-0.88	28	27	0.885	22
3	-0.85	29	28	0.882	21

4	-0.79	30		29	0.878	20	
5	-0.74	30		30	0.874	19	
6	-0.675	30		31	0.859	18	
7	-0.610	30		32	0.856	17	
8	-0.542	30		33	0.868	16	
9	-0.465	30		34	0.878	15	
10	-0.369	30			*Average 25-34:*		0.879
11	-0.247	30		35	0.891	14	
12	-0.111	30		36	0.880	13	
13	0.050	30		37	0.819	12	
	Average 0-13:		-0.57	38	0.728	11	
14	0.236	30		39	0.642	10	
15	0.431	30		40	0.439	9	
16	0.618	30			*Average 37-40:*		0.702
17	0.778	30					

Table 1 (cont'd)

	Average 14-17:		0.516
18	0.902	30	
19	0.961	30	
20	0.979	29	
21	0.964	28	
22	0.956	27	

23	0.939	26
24	0.919	25

Average 18-24: 0.95

Table 2

Percent Screened with Blood Lead Above 10µg/dL and Other Characteristics, Matched Sample of 30 Nonfluoridate and 30 Silicofluoride Communities – Massachusetts

	30 Non-fluoridated Communities	30 Fluoridated Communities
Population(1,000s)	837.3	845.1
Children 0-5 years	57,031	56,446
% children screened with >10µmg/dL	0.76	1.94
Lead in water (ppb)	21	30
4th grade MEAP	5440	5455
% Poor	4.6%	5.1%
% Nonwhite	6.6%	11.5%
% AB.	23.6%	30.5%
Income per capita	$116,600	$19,600

Table 3

Community Demographics and Risk Factors, New York Sample: Distribution of 1990 U.S. Census Variables in 105 NY State Communities of Population 15,000-75,000 by SiF Status

	SiF	No SiF
Demographics		
Number of Communities	28	77
Mean Community Size	34,778	25,627
Children 0-5 as % of Pop.	8.50%	8.00%
No. Children 0-5 years per Community	2,960	2,046
No. children tested, 1994-1998		
Total Number of VBL Tests	56,934	94,291
Total Number Capillary Tests	36,791	68,357
Total of all Blood Lead Tests	93,725	162,648
Percent of Tests for VBL	61%	58%
Risk factors associated with high blood lead		
Housing pre 1939	49.4%	23.3%
% Age 0-5 in Poverty	22.3%	8.5%
% Unemployed	3.5%	2.5%
% B.A.	7.4%	9.3%
Pop density (per Sq. Km)	155	143
Total Population	973,785	1,973,336

Per Capita Income	$14,698	$19,415

Table 4

Multiple Regression Analysis of Violent Crime Rates in US – 1991

Variable	Unstandardized Coefficient	t-value
p-value		
Population Density	82.42	20.24
<.0001		
Per capita income	-.0007	-2.74
<.0001		
Unemployment		NOT SIGNIFICANT
% Black Poverty	40.06	2.33
<.05		
% Hispanic Poverty	62.11	2.79
<.005		
Police per Capita	153423	l6.56
<.0001		
Infant Death Rate	1.813	2.78
<.005		
% housing pre-1950	526.75	-13.43
<.0001		
Public water per capita	225.34	4.07
<.0001		

Median Grade Complete	24.68	3.50
<.005		
Lead TRI present	40.80	4.67
<.0001		
Manganese TRI	58.71	6.68
<.0001		
Alcohol Death Rate	101.62	11.55
<.0001		
#Alcohol x Lead	21.48	2.54
<.05		
#Alcohol x Manganese	55.40	6.54
<0001		
#Lead x Manganese	34.89	4.11
<0001		
#Alcohol x Lead x Manganese	19.21	2.27
<.05		

DF 17, 2783; adjusted r-square = 0.369; F = 97.45; p < .0001

-- interaction terms.

Table 5

Factors Influencing U.S. Violent Crime Rate, 1985. Results of multiple regression on data from 2880 US Counties. (Variables Listed in Order of Strength of Standardized Coefficient)†

Variable	Standardized Coefficient	t-value	p-value
% Black	2798	15.90	.0001
Poverty/Wealth Ratio	.2262	6.56	.0001
Population Density	.1956	9.38	.0001
% SiF	.1150	6.19	.0001
% HS Graduate	.0795	3.46	.0005
Per Capita Income	.0457	1.85	.0642
% Houses pre 1939	-.1071	5.09	.0001
Population	-.02587	0.82	n.s.
Lead Toxic Releases	.0042	0.262	n.s.
Manganese Toxic Releases	.0196	1.246	n.s.

DF 10, 2869; R squared = .3238; F = 137.401; p = .0001

† Note: When both % of population on silicofluorides and toxic release inventory (TRI) of lead and manganese are included in the analysis, silicofluoride usage is a significant predictor of violent crime whereas heavy metal pollution ceases to have a significant additional effect. This probably explains the significance of the variable "public water supply per capita" in the 1991 multiple regression in Table 4, which was calculated before RDM knew of the issue of silicofluoride toxicity.

Table 6

Factors Associated with Rates of Violent Crime: Results of Multiple Regression on Data from All U.S. Counties, 1985

Variable	Coefficient	Std. Error	Std. Coefficient	t-value	p-value
Intercept	-0.005056				
**** %SiF**	**0.000368**	**0.000133**	**0.045**	**2.78**	**0.0055**
Unemployment	0.000076	0.000013	0.106	5.99	0.0001
PC Income Blacks…	-9.92E-09	5.69E-09	-0.029	1.74	0.0816
PC Income	9.53E-08	1.91E-08	0.115	4.99	0.0001
Median Grade…	0.000205	0.000069	0.082	2.97	0.003
Median year…	0.000003	0.000004	0.0123	0.72	0.4722
% Black	0.00005	0.000003	0.313	17.56	0.0001
% Graduate…	-0.000022	0.000007	-0.096	2.96	0.0031
% Rural	-0.000027	0.000001	-0.350	18.73	0.0001

Confidence intervals

Variable	95% Lower	95% Upper	90% Lower	90% Upper	Partial F
**** % SiF**	**0.000108**	**0.000628**	**0.00015**	**0.000587**	**7.72**
Unemployment…	0.000051	0.000101	0.000055	0.000097	35.86
PC Income Blacks	-2.11E-08	1.25E-09	-1.93E-08	-5.50E-10	3.04
PC Income	5.78E-08	1.33E-07	6.39E-08	1.27E-07	24.89
Median grade…	0.00007	0.00034	0.000091	0.000318	8.83
Median year…	-0.000005	0.000011	-0.000004	0.00001	0.52

Table 6 (cont'd)

% Black	0.000044	0.000056	0.000045	0.000055	308.54
% Graduate …	-0.000036	-0.000007	-0.000034	-0.00001	8.79
% Rural	-0.00003	-0.000024	-0.000029	-0.000024	350.75

Table 7

Factors Associated with Rates of Violent Crime: Results of Multiple Regression on Data from All U.S. Counties, 1991

Variable	Coefficient	Std. Error	Std. Coefficient	t-value	p-value
Intercept	-0.026874				
**** %SiF**	**0.000922**	**0.00019**	**0.076136**	**4.84725**	**0.0001**
Unemployment	0.000064	0.000017	0.062928	3.692	0.0002
PC Income Blacks…	-3.96E-09	8.09E-09	-0.007926	0.489639	0.6244
PC Income	1.28E-07	2.63E-08	0.108872	4.869223	0.0001
Median Grade…	0.000504	0.000095	0.140963	5.304905	0.0001
Median Year…	0.000014	0.000006	0.039495	2.411564	0.0159
% Black	0.00008	0.000004	0.351002	20.358866	0.0001
% Graduate…	-0.000058	0.00001	-0.178521	5.719072	0.0001
% Rural	-0.000041	0.000002	-0.376415	20.749842	0.0001

Confidence intervals

Variable	95% Lower	95% Upper	90% Lower	90% Upper	Partial
**** %SiF**	**0.000549**	**0.001295**	**0.000609**	**0.001235**	**23.50**
Unemployment	0.00003	0.000098	0.000035	-0.000038	13.64
PC Income Blacks…	-1.98E-08	1.19E-08	-1.73E-08	9.36E-09	0.24
PC Income	7.65E-08	1.80E-07	8.48E-08	1.71E-07	23.71
Median Grade…	0.000317	0.00069	0.000347	0.00066	28.14
Median year…	0.000003	0.000026	0.000004	0.000024	5.82

Table 7 (cont'd)

% Black	0.000072	0.000088	0.000074	0.000087	414.48
% Graduate…	-0.000078	-0.000038	-0.000075	-0.000041	32.71
% Rural	-0.000045	-0.000037	-0.000044	-0.000038	430.56

Table 8

Factors Associated With Rates of Drunkenness Per Capita, 1991: Stepwise Regression on Data from 3139 U.S. Counties (Variables Listed in Order of Entry) †

Variable	Standard Coefficient	F to Remove	Total Adj R-square after variable entry
% HS Graduate	-0.316	171.334	.089
% Hispanic	0.208	150.238	.143
Median Year Housing Built	0.147	68.230	.158
% Black	-0.123	48.825	.168
% SiF	0.054	10.832	.171
Per Capita Income	0.051	4.896	.173
Pop Density	0.037	4.355	.174

† Notes: F to remove criterion was 4. Variables not entered: population size, unemployment, social inequality [poverty/per capita income], Lead TRI, Manganese TRI.

To confirm that the year chosen did not influence the result, the same stepwise regression was run for rates of drunkenness in all U.S. counties for the years from 1990 through 1995. The % of county population receiving silicofluoride treated water was a significant predictor for 1990, 1992, 1994, and 1995 (in each case, one of six variables) as well as in 1991 (above, one of seven variables). The strength of the standardized coefficient was similar in all these cases. For 1993, however, % SiF was not significant and was replaced by unemployment as one of six significant predictors. In contrast,

when the same stepwise regression model was used to predict property crime rates in these years, %SiF was not significant for 1990, 1991, 1992, and 1995, and had a negative coefficient for 1993 and 1994 (for each year, six or seven variables were significant). These results are consistent with the hypothesis that the behavioral effects of silicofluoride treated water were associated with enhanced lead uptake or other neurotoxic effects that weaken impulse control.

Figure Captions

Figure 1. Average blood lead for NHANES III - Children 3-5 (Counties over 500,000). Mean blood lead is significantly associated with fluoridation status (DF 3, $F = 17.14$, $p < .0001$) and race (DF 2, $F = 19.35$, $p < .0001$), as well as for poverty income ratio (DF 1, $F = 66.55$, $p < .0001$). Interaction between race and fluoridation status: DF 6, $F = 3.33$, $p = .0029$.

Figure 2. Average blood lead NHANES III - Children 5-17 (Counties over 500,000). Significance: fluoridation status (DF 3, $F = 57.67$, $p < .0001$), race (DF 2, $F = 28.68$, $p < .0001$), Poverty-Income Ratio (DF 1, $F = 252.88$, $p < .0001$). Interaction between race and fluoridation status: DF 6, $F = 11.17$, $p < .0001$.

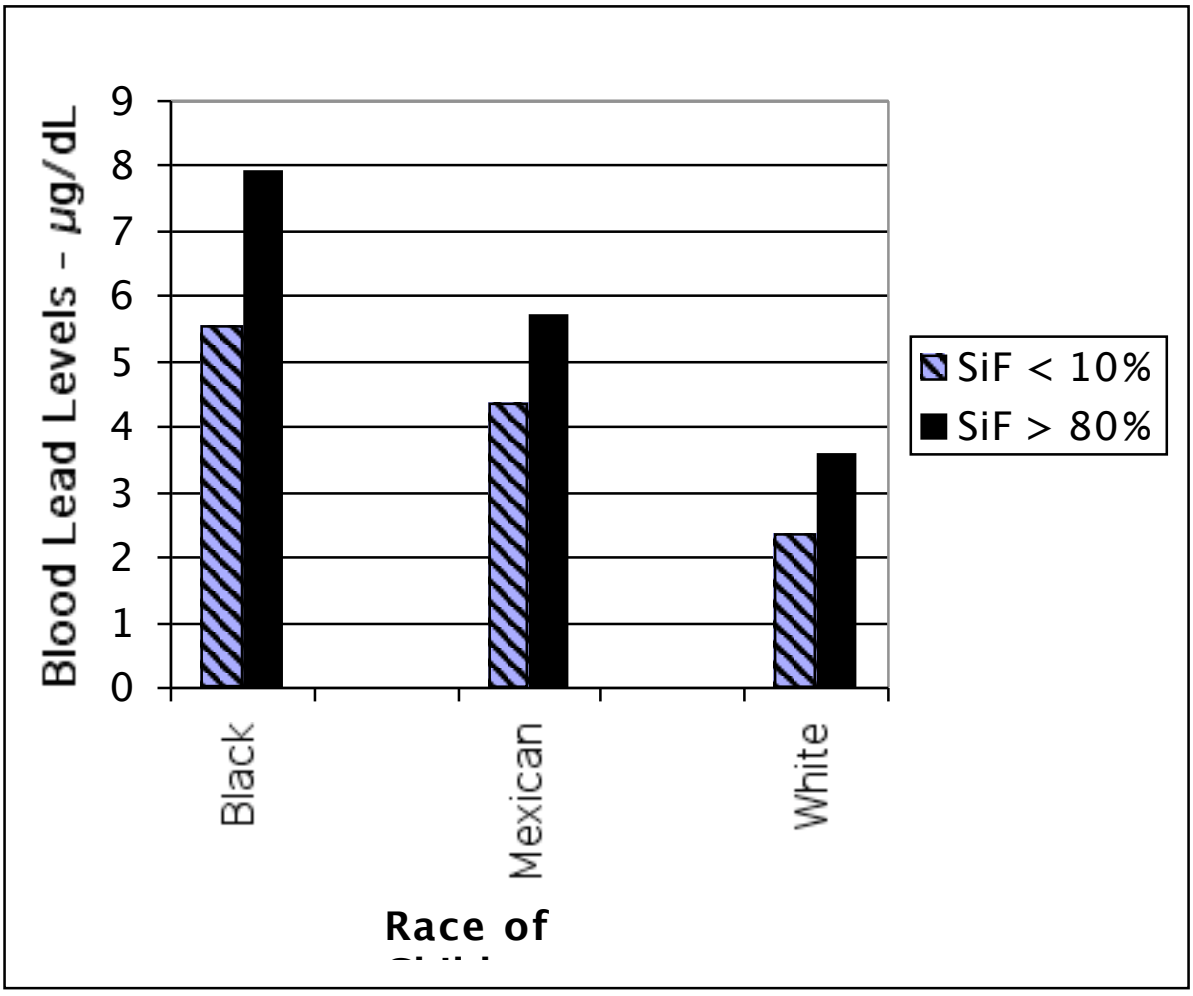
Figure 3. Average capillary blood lead in Massachusetts by community, effects of silicofluoride use and percentage of the population that is Black. Significance: silicofluoride use, $p = .0001$; % Black, $p = .0001$; Interaction between SiF use and % Black, $p = .0001$.

Figure 4. Venous blood lead levels in Black children (Communities of 15,000-75,000), with and without SiF treatment.

Figure 5. Violent crime rates in counties with and without toxic releases of lead and manganese (EPA toxic release inventory, TRI). Recalculated from Masters et al. (1998).

Figure 6. Manganese TRI & silicofluorides as factors in violent crime (1991). Significance: SiF usage, $F = 27.60$, $p = .0001$; manganese pollution, $F = 79.00$, $p = .0001$; interaction between SiF and Mn: $F = 3.74$, $p = .0239$. For the 369 US counties where over 60% received water treated with SiFs and there is no Toxic Release Inventory record

for manganese, the violent crime rate in 1991 (3.53 per 1000) was intermediate between rates in the 109 counties with manganese TRI and no SiFs (4.40) or the 217 counties with between 0.1 and 60% receiving SiFs (3.49). Where both SiFs are delivered to over 60% of the population and manganese TRI is present, the crime rate was 5.34. In 1991, the national county average was 3.12 violent crimes per 1000.


Blood Lead Levels - µg/dL
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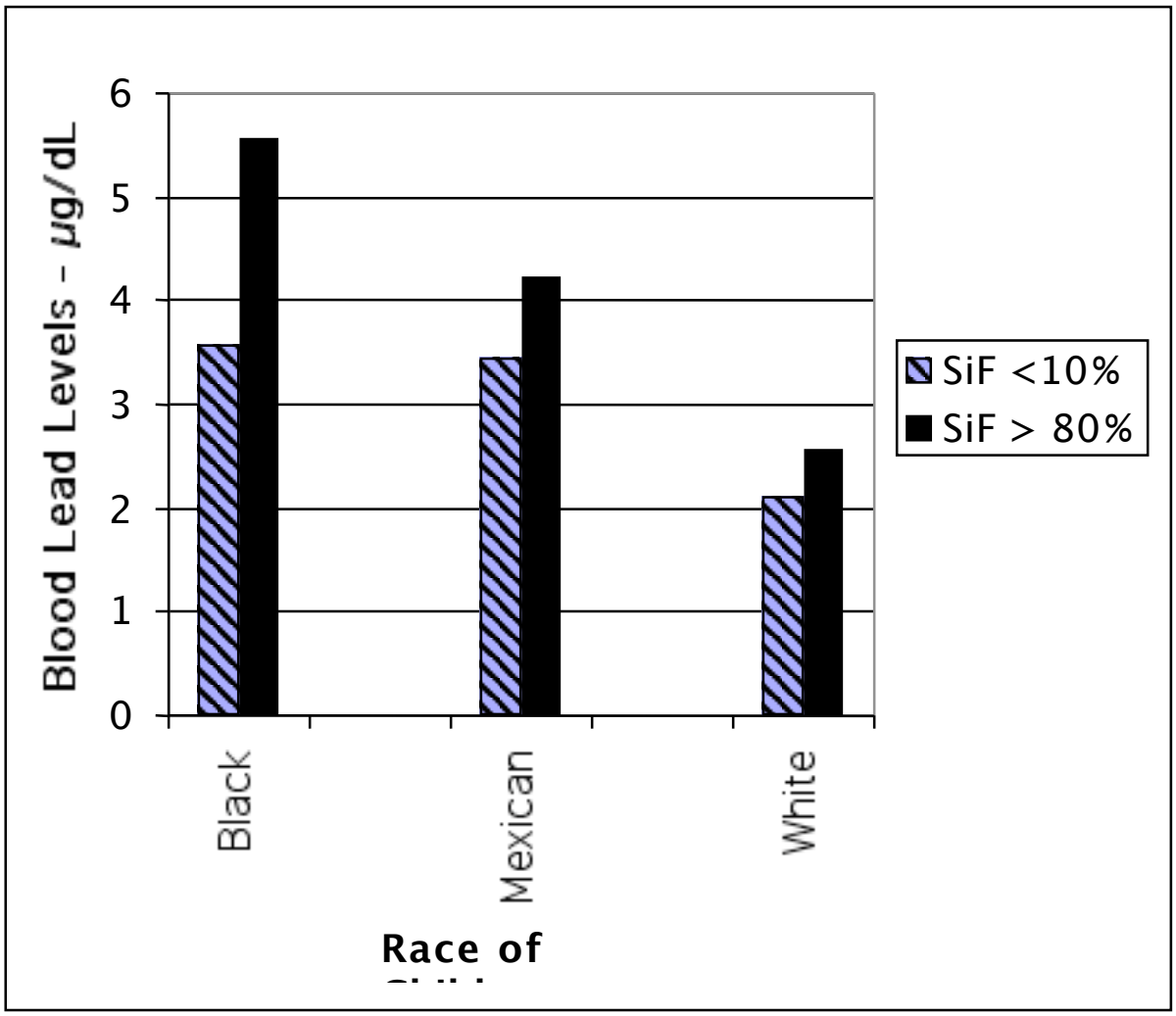
8
7
6
5
4
3
2
1
0
Black
Mexican
White
Race of
SiF < 10%
SiF > 80%


Blood Lead Levels - µg/dL
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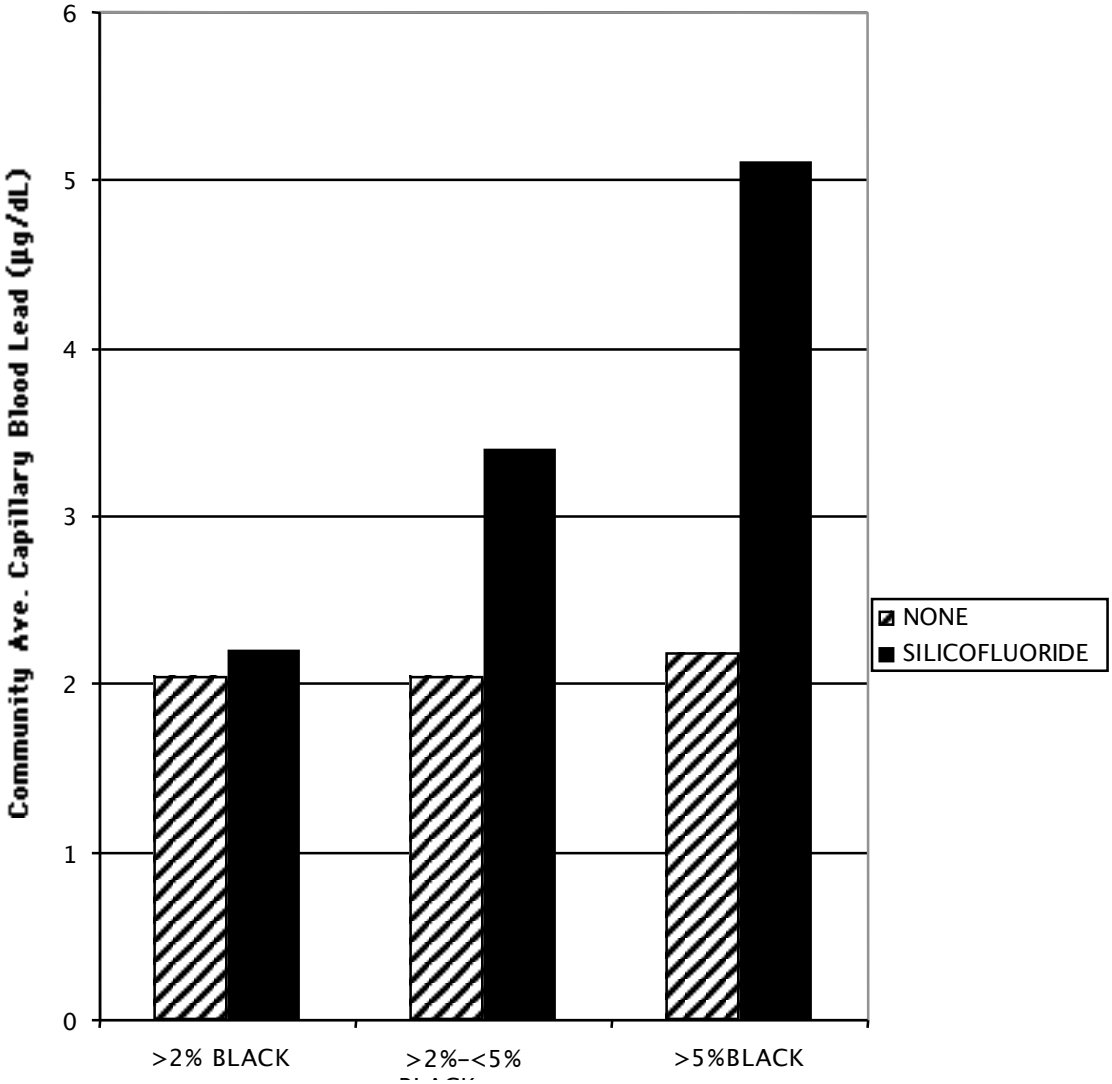
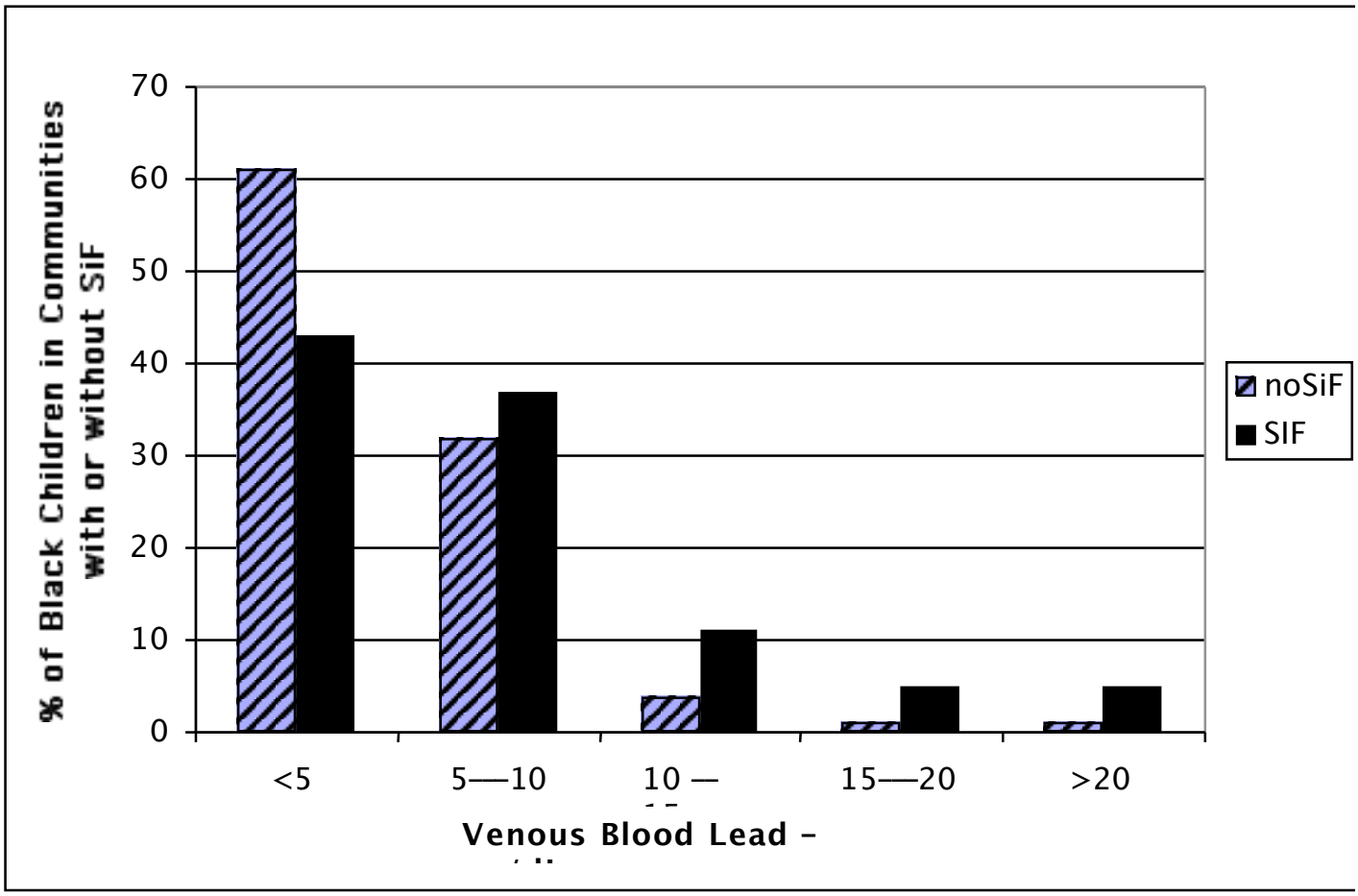
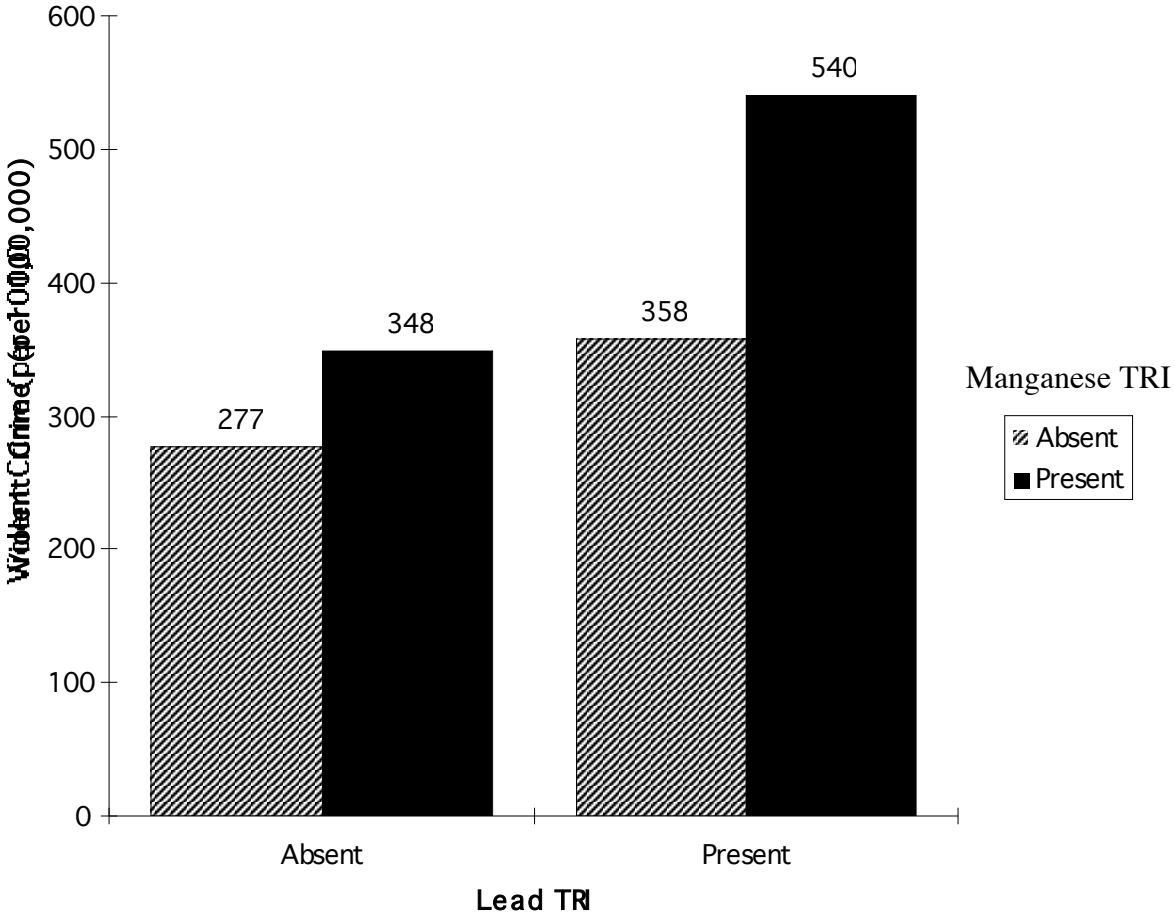
5
4
3
2
1
0
Black
Mexican
White
Race of
SiF <10%
SiF > 80%


Community Ave. Capillary Blood Lead (µg/dL)
6
5
4
3
2
1
0
>2% BLACK
>2%-<5%
>5%BLACK
NONE
SILICOFLUORIDE


% of Black Children in Communities with or without SiF
70
60
50
40
30
20
10
0
<5
5—10
10 —
15—20
>20
Venous Blood Lead –
noSiF
SIF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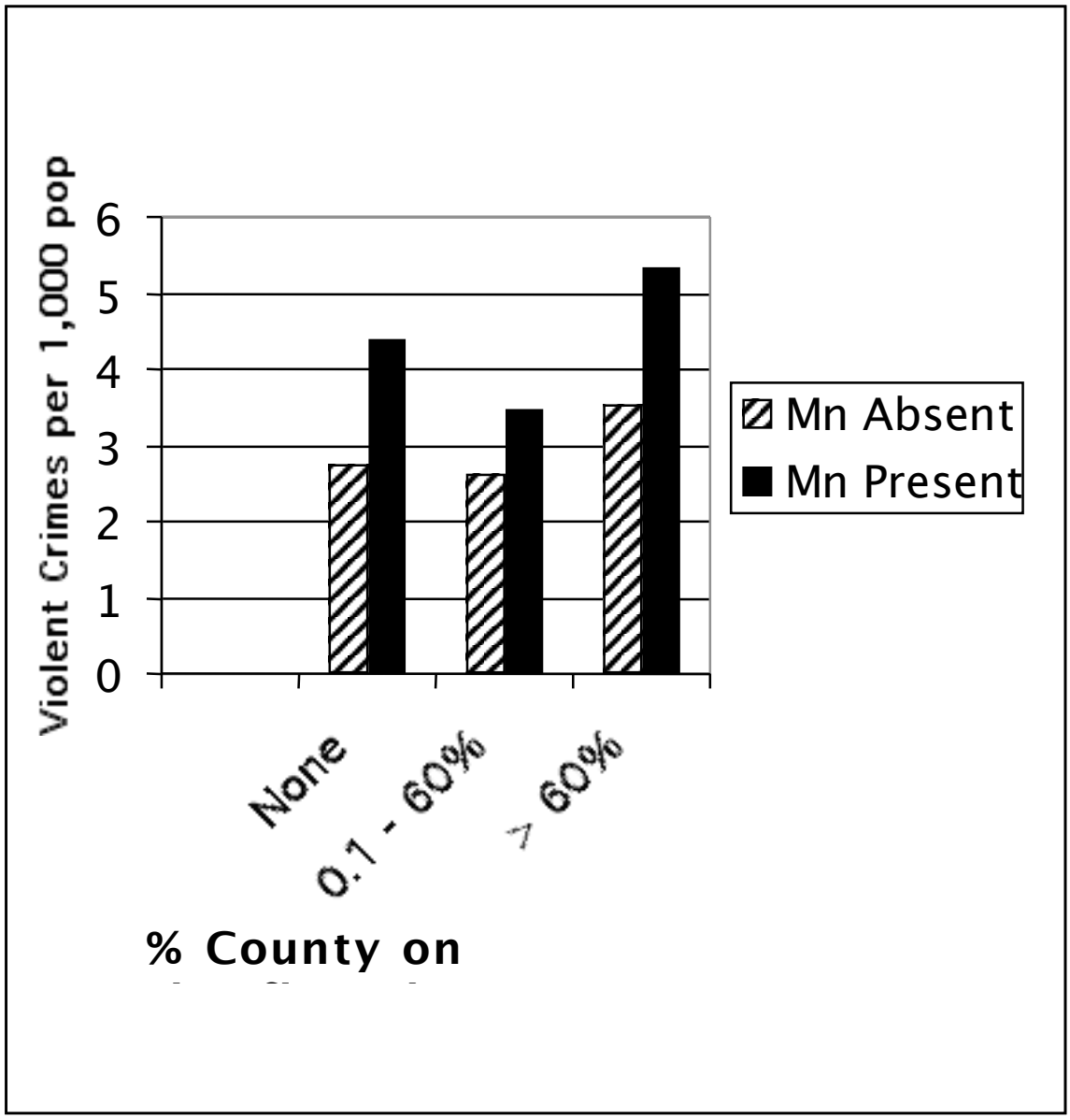
400
300
200
100
0
277
348
358
540
Absent
Present
Lead TRI
Manganese TRI
Absent
Present


Violent Crimes per 1,000 pop
6
5
4
3
2
1
0
None
0.1 - 60%
> 60%
% County on
Mn Absent
Mn Present