Can Fluoridation Affect Water Lead(II) Levels and Lead(II) Neurotoxicity?[a,b]

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Abstract

Recent reports have attempted to show that certain approaches to fluoridating potable water is linked to increased levels of lead(II) in the blood. We examine these claims in light of the established science and critically evaluate their significance. The completeness of hexafluorosilicate hydrolysis is of paramount importance in ensuring that total water quality is maintained. The possible impacts of such complexes as Pb^{II}–F–SiF_5 or $PbF_x^{(2-x)}$ are discussed as are the contributions of fluoridation byproducts to total acid content. We calculate the fractional distribution of aqueous species based on known chemical equilibria and show the species concentrations for a selected model tap water with a composition that would favor lead fluoride and silicofluoride complexation. We discuss and quantitatively show the effects of other complexing anions, such as carbonate or hydroxide. Overall, we conclude that no credible evidence exists to show that water fluoridation has any quantifiable effects on the solubility, bioavailability, bioaccumulation, or reactivity of lead(0) or lead(II) compounds. The governing factors are the concentrations of a number of other species, such as (bi)carbonate, hydroxide, or chloride, whose effects far exceed those of fluoride or fluorosilicates under drinking water conditions. Lastly, we consider some previous epidemiological studies of lead(II) exposure and how recent papers fare methodologically.

Background

Controversy over water fluoridation varies in nature and intensity. Recent papers have implications for water fluoridation since they suggest that certain adverse health or social conditions may stem from interactions between lead(II) and inorganic fluoro-compounds, specifically, fluorosilicates and fluoride.[1-3] In order to assess the validity of these assertions, it is necessary to have a firm foundation of the aqueous chemistry of H_2SiF_6 and HF, as well as of Pb. There is a considerable body of fundamental chemical literature on these species. Nonetheless, some gaps do remain,[4] and little

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[b]Several minor modifications and corrections from the actual AWWA Annual Conference Paper have been made to this version.

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effort has been expended in combining the known chemistry into one comprehensive and authoritative volume. Accordingly, we believe it to be worthwhile to revisit the chemical concepts and relationships involved in water fluoridation at a fundamental level in light of well-established science, and thus to examine the potential validity of some of the hypotheses of adverse chemical interactions suggested by the recent papers.[1-3] We have previously reviewed the state of knowledge in considerable detail,[4] but here emphasis is placed on the chemistry and conditions of most relevance to the public drinking water treatment and health communities.

The sheer number of people consuming fluoridated potable water makes fluoridation issues relevant. In 1992, the Centers for Disease Control Fluoridation Census found that 62.1% of the U.S. population served by public suppliers drank fluoridated water.[5] The CDC also surveyed utilities regarding fluoridating agents (see Table 1). Most commonly used are hexafluorosilicic acid (H_2SiF_6) or its sodium salt (Na_2SiF_6), which hydrolyze to produce fluoride ion upon dilution (eqs 1-2). However, sodium fluoride (NaF) is sometimes used as a direct fluoride source (eq 3).

$$H_2SiF_6(aq) + 4\ H_2O \rightarrow 6\ HF(aq) + Si(OH)_4(aq) \quad (1)$$

$$Na_2SiF_6(aq) + 4\ H_2O \rightarrow 4\ HF(aq) + 2\ NaF(aq) + Si(OH)_4(aq) \quad (2)$$

$$NaF \rightarrow Na^+(aq) + F^-(aq) \quad (3)$$

Table 1. Water fluoridation chemicals used by U.S. public water suppliers in 1992

	hexafluorosilicic acid	sodium hexafluorosilicate	sodium fluoride
formula	H_2SiF_6	Na_2SiF_6	NaF
common synonyms	fluosilicic acid fluorosilicic acid hydrofluosilicic acid	sodium silicofluoride sodium fluorosilicate	—
population served	80,019,175 62.6%†	36,084,896 28.2%†	11,701,979 9.2%†
utilities using	5876	1635	2491

*Total US population: 258,544,000. Population and utility data were taken from reference 4.
†Percentages are based on total population of 127.8 million persons drinking fluoride-fortified public water and does not include those drinking water naturally high in fluoride.

Hexafluorosilicic acid is a cheap and readily available source of fluoride. However, it is difficult to handle and the handling costs can only be offset by the volume discount in large water treatment plants. Although more systems rely on sodium fluoride than sodium hexafluorosilicate, these serve only 9.2% of the U.S. population. Because sodium fluoride is the easiest of the three to handle and dispense, small systems are the primary users of NaF. Although the EPA regulates drinking water, the US Public Health Service has been involved in water fluoridation for historical reasons (primarily because the practice of fluoridation pre-dates EPA). The purpose of fluoridating water is the prevention of dental caries; therefore, the publication of water fluoridation how-to manuals falls

under the purview of the CDC. These manuals discuss dosing and other practical matters of concern to the treatment plant operator.[6]

Drinking water contains a large number of chemical species, including disinfection byproducts, residual oxidants, dissolved organic matter, trace metals, minerals, and additives (such as fluoride). As a result, drinking water science is a complicated interplay among the chemical constituents as well as the physical conditions. We think it is useful to frame the issues recently raised about adverse interactions between aqueous lead and fluoridation species,[1-3] in a question-answer format, to help water managers, scientists, and engineers understand and respond to them.

1. What is the residual concentration of hexafluorosilicate ion (SiF_6^{2-}) after hydrolysis and how fast does hydrolysis take place?

Hexafluorosilicate ion reacts with water to produce fluoride ion and an assortment of silicon oxyanions,[7-8] *e.g.*, SiO_3^{2-}, SiO_4^{4-}, $Si(OH)O_3^{3-}$. We represent the oxyanions as $Si^{IV}(aq)$ without further speciation at this time.

$$SiF_6^{2-} + n\ H_2O \rightleftharpoons Si^{IV}(aq) + 6\ HF(aq) \qquad (4)$$

The actual speciation of silicon oxyanions is a function of acidity, *i.e.*, $[H^+]$. Busey *et al.*[9] showed that virtually 100% of the hexafluorosilicate is hydrolyzed to silicon oxyanions at pH 6, even when there is a free fluoride concentration of 0.01 M. Meanwhile, fluoridated drinking water contains only ~1 mg/L of fluoride, which equates to 5×10^{-5} M. Previous investigations[10-11] found a non-negligible concentration of residual SiF_4 when this gas was passed through water. Ciavatta[10] *et al.* investigated fluorosilicate equilibria with $0.3 \# [H^+] \# 3$ *molal* [*m*, mol F^- (kg water)$^{-1}$] and ionic strength fixed at 3 M, adjusted with $LiClO_4$. They concluded that the mixed ligand species $SiF(OH)_3$ and $SiF(OH)_2(H_2O)^+$ are significant contributors to total silicon(IV) in addition to SiF_4, SiF_6^{2-}, and $HSiF_6^-$ under these conditions. Nonetheless, their results showed that fluoro-complexes comprised less than 5 mol% of the total silicon(IV) in 0.01 *m* H^+ and 10^{-4} *m* F^-. Korobitsyn *et al.*[11] examined the hydrolysis of sodium hexafluorosilicate in sodium carbonate solution. Their work was geared towards an industrial process for producing sodium fluoride and is not directly applicable here.

The use of chemical shift information derived from ^{19}F NMR spectrometry in understanding the formation of fluoro-ligated species is well-established.[12-17] Fluoride ligand exchange occurs rapidly between HF and SiF_6^{2-} at temperatures above –10 °C,[14] and the identification of aqueous fluorosilicate species and the measurement of the concomitant equilibrium constants has been done almost entirely by ^{19}F NMR spectroscopy and spectrophotometry.[15-17] The *Gmelin Handbook of Inorganic Chemistry* tabulates values for the equilibrium constants expression (6) of the hydrolysis reaction (5) at temperatures from 0 to 60 °C.[18]

$$SiF_6^{2-} + 4\ H_2O \rightleftharpoons Si(OH)_4 + 4\ H^+ + 6\ F^- \qquad (5)$$

$$K = \frac{[Si(OH)_4][H^+]^4[F^-]^6}{[SiF_6^{2-}]} \quad (6)$$

The *smallest* value at ambient temperature reported for K is $10^{-31.6}$. Using this value at $[H^+] = 10^{-6}$ M and $[F^-] = 5 \times 10^{-5}$ M, the ratio $[Si(OH)_4]/[SiF_6^{2-}] = 1.6 \times 10^{18}$. Note that less than 1% of fluoride exists as HF at drinking water acid levels (*i.e.*, pH > 5.2) since $pK_a^{HF} = 3.17$.[19] Even if the hydrolysis constant were off by a factor of 1000, it would not matter. There would still be essentially no hexafluorosilicate ion. A fractional distribution plot in *Gmelin*[18] shows that other fluorosilicates (*i.e.*, SiF_4 and SiF_5^-) also drop off dramatically as free fluoride concentration, and not $[F^-]_T$, decreases towards 10^{-4} M, even in silica-saturated 4 M perchloric acid. For this solution, total fluoride concentration is expressible as (7), neglecting any mixed fluorohydroxo-ligated species:

$$[F^-]_T = [HF] + [F^-] + 4\,[SiF_4] + 5\,[SiF_5^-] + 6\,[SiF_6^{2-}] \quad (7)$$

Crosby studied the dissociation of sodium hexafluorosilicate and hexafluorosilicic acid in deionized water.[20] He found that about 99 mol% of the hexafluorosilicate had hydrolyzed when added to water to produce a 1 mg/L fluoride solution; however, the pH of this solution was 4.20, considerably below a potable water pH. An important factor must be considered in potable water fluoridation as Crosby explained:

> It should be remembered that the actual ionic population of most public drinking-water supplies is somewhat different from the experimental conditions used in the present and previous studies. Thus, the pH is normally adjusted to about 7 to 8, and the presence of additional salts may further influence the equilibrium owing to the formation of complexes with calcium and other metals.

If the pH of a treated drinking water is too low, it is adjusted to comply with regulations (or consumer complaints) and minimize corrosion. Crosby's results were obtained in a water that was demineralized and completely devoid of buffering agents. Consequently, the dissociation of hexafluorosilicate was hindered by the drop in pH. Thus, Crosby's fractional dissociation data cannot be applied directly to a potable water supply without correcting them for pH. Of course, that correction is the effect we have computed above, namely, the complete hydrolysis of fluorosilicates. This is precisely what Crosby was emphasizing. This observation hints at the effect on pH, which we shall come back to shortly.

Interestingly enough, a number of species actually promote the dissociation of hexafluorosilicate, including ferric ion.[21] While the compound $PbSiF_6 \cdot 2H_2O$ can be synthesized, it decomposes quickly in moist air and slowly when dry.[22] Perhaps then lead(II) itself promotes hexafluorosilicate decomposition, such as through the formation of plumbous fluoride. Because moist air promotes this compound's destruction, we can infer that it would not be stable in aqueous solution at all. There is essentially no hexafluorosilicate remaining in drinking water at equilibrium.

Now we must consider how fast hydrolysis takes place. In the 1970s, Plakhotnik conducted studies into the effects of lithium and calcium cations on the rate of hexafluorosilicate (and tetrafluoroborate) hydrolysis.[23-24] Based on Plakhotnik's results, we calculated[4] that the hydrolysis would be 99 mol% complete in 12 minutes *if carried entirely by the uncatalyzed pathway*. That

notwithstanding, natural water supplies do contain calcium and other divalent metals as well as trivalent metal cations (*e.g.*, Al^{3+}, Fe^{3+}); hence, the actual hydrolysis rate would be even faster so that equilibrium is reached long before water reaches the consumer's tap.

Based on the above information on both the thermodynamics of the hydrolysis reaction and its kinetics, we can safely conclude that there is essentially no (« 1 part per trillion) hexafluorosilicate remaining in drinking water at equilibrium and that equilibrium is rapidly reached from the combined uncatalyzed and metal-catalyzed reactions.

2. Can F^- or residual SiF_6^{2-} complex with Pb^{2+} and make it more bioavailable?

Another way to ask this is: Do fluoro-species complex with Pb(II), promoting permeation of the gastric mucosa and absorption into the bloodstream? Even though we have demonstrated that there is no hexafluorosilicate remaining by the time water reaches the consumer's tap, the following scenarios nicely illustrate the magnitude of the effects on lead(II).

To produce 1.0 mg/L fluoride requires an initial hexafluorosilicate concentration of 8.8 µM. The hydrolysis reaction (eqn 2) is a reversible equilibrium, and in the most acidic gastric conditions, the pH could be as low as 1.5 so that $[H^+] = 10^{-1.5}$ M (assuming unit activities). Using this hydrogen ion concentration, we calculate the ratio $[Si(OH)_4]/[SiF_6^{2-}] = 4.5 \times 10^5$. This means that only 0.00022% of the total silicon(IV) is present as the hexafluorosilicate ion so that $[SiF_6^{2-}] = 1.9 \times 10^{-11}$ M = 19 picomolar (pM).

Haque and Cyr[25] showed that hexafluorosilicate anion forms complexes with several metal cations: Cu^{II}, Ni^{II}, Co^{II}, and Fe^{III}. The largest stability constant they obtained was for the reaction with ferrous ion, with $K = 1.2 = 10^{0.08}$. Let us assume that the lead(II) ion forms a stabler complex and set the stability constant for eq 18 to an arbitrarily high value of 100. In addition, we shall *pretend* that the hydrolysis computed above has not occurred, that all 8.8 µM of the silicon(IV) remains in the form of hexafluorosilicate ion. In this worst case, only 0.088 mol% of the total lead(II) would be in the form of a hexafluorosilicate complex. The µ-fluoro ligand would serve as a link between the silicon(IV) and lead(II).

$$Pb^{2+} + SiF_6^{2-} \text{ ⚬ } PbSiF_6(aq),\ K = 100 \text{ (hypothetical)} \qquad (8)$$

Now consider the actual case where $[SiF_6^{2-}] = 19$ pM. If the equilibrium constant for eqn (8) were 10,000 times larger, say 10^6, so that the reaction could be treated as going to near completion, there would still be less than 19 pM Pb–µ-F–SiF_5. Because of the magnitude of the equilibrium constant for eqn (6), the equilibrium constant for eqn 8 would have to exceed 10^{25} in order to have a quantitatable effect by preventing hexafluorosilicate hydrolysis. There is no basis in fact for such an assertion. As a final point, we note that the national primary drinking water standards are intentionally predicated on the assumption that all lead is bioavailable, and the water utilities should be complying with these standards.

What about the effect of the fluoride itself? Can it promote lead(II) bioabsorption? Is there an association between lead(II) and fluoride? Therefore, dosing the chemical NaF does affect pH indirectly via eqn (9) because HF is a weak acid with a pK_a of 3.17.[19] However, its effect can generally be neglected since the pH of drinking water is controlled by many different buffering species,[26] which will be discussed later.

$$F^- + H_2O \rightleftharpoons HF + OH^-, K_b = 10^{-10.5} \quad (9)$$

There is only a very small association[27-28] between Na^+ and F^- (Table 2, eqn F1). The magnitude of this stability constant is so small as to be negligible; however it can still be calculated. On the other hand, other cations present in reasonably high concentrations, most notably aluminum, bind to fluoride much more strongly. Table 2 summarizes these chemical equilibria and their stability constants.

Table 2. Cumulative stability constants for formation of fluoro-complexes*

Fluoro-complexation	Eqn	log $
$Na^+ + F^- \rightleftharpoons NaF(aq)$	(F1)	–0.24†
$Al^{3+} + F^- \rightleftharpoons AlF^{2+}$	(F2)	7.0†
$Al^{3+} + 2\ F^- \rightleftharpoons AlF_2^+$	(F3)	12.7†
$Al^{3+} + 3\ F^- \rightleftharpoons AlF_3(aq)$	(F4)	16.8†
$Al^{3+} + 4\ F^- \rightleftharpoons AlF_4^-$	(F5)	19.4†
$Al^{3+} + 5\ F^- \rightleftharpoons AlF_5^{2-}$	(F6)	20.6†
$Al^{3+} + 6\ F^- \rightleftharpoons AlF_6^{3-}$	(F7)	20.6†
$Al^{3+} + H_2O + F^- \rightleftharpoons AlOHF^+$	(F16)	0.0§
$Al^{3+} + H_2O + 2F^- \rightleftharpoons AlOHF_2°$	(F17)	20.6§
$Fe^{3+} + F^- \rightleftharpoons FeF^{2+}$	(F8)	5.2‡
$Fe^{3+} + 2\ F^- \rightleftharpoons FeF_2^+$	(F9)	9.1‡
$Fe^{3+} + 3\ F^- \rightleftharpoons FeF_3(aq)$	(F10)	11.9‡
$Ca^{2+} + F^- \rightleftharpoons CaF^+$	(F11)	0.94†
$Mg^{2+} + F^- \rightleftharpoons MgF^+$	(F12)	1.82†
$Cu^{2+} + F^- \rightleftharpoons CuF^+$	(F13)	1.2‡
$H^+ + F^- \rightleftharpoons HF(aq)$	(F14)	3.18†
$H^+ + 2\ F^- \rightleftharpoons HF_2^-$	(F15)	3.76†
$2H^+ + 2\ F^- \rightleftharpoons H_2F_2°$	(F18)	6.77§

*These stability constants are used for the construction of Figures 1-4 with the exception of Equations (F8)-(F10) and (F13). †Values taken from reference 28. ‡Values taken from reference 19. §Values taken from reference 32.

There are many metal cations competing for the fluoride; therefore, the free fluoride available to complex with the lead(II) ion is very small. In addition, most, if not all, of the competing metal cations are in greater abundance than lead(II) by orders of magnitude. Further reducing the lead(II) are such ligands as hydroxide, chloride, carbonate, bicarbonate, and sulfate, all of which compete with fluoride for the lead(II) and are present in far greater concentrations. Table 3 summarizes these equilibria and their stability constants. For pH > 6, the free lead(II) concentration drops off dramatically from hydroxo- and (bi)carbonato-complexation. That drinking water contains a substantial fraction of fluoroaluminum complexes rather than free fluoride was highlighted by Pitter as a concern because free fluoride is more effective in protecting against tooth decay.[29] We shall take these and other factors into account in speciating the lead(II).

Table 3. Lead(II) equilibria and constants*

Equilibrium	Eqn	log $
$Pb^{2+} + H_2O$ ° $PbOH^+$	(L1)	–7.22
$Pb^{2+} + 2\ H_2O$ ° $Pb(OH)_2(aq) + 2\ H^+$	(L2)	–16.91
$Pb^{2+} + 3\ H_2O$ ° $Pb(OH)_3^- + 3\ H^+$	(L3)	–28.08
$Pb^{2+} + 4\ H_2O$ ° $Pb(OH)_4^{2-} + 4\ H^+$	(L4)	–39.72
$2\ Pb^{2+} + H_2O$ ° $Pb_2OH^{3+} + H^+$	(L5)	–6.36
$3\ Pb^{2+} + 4\ H_2O$ ° $Pb_3(OH)_4^{2+} + 4\ H^+$	(L6)	–23.86
$Pb^{2+} + CO_3^{2-}$ ° $PbCO_3(aq)$	(L7)	7.10
$Pb^{2+} + 2\ CO_3^{2-}$ ° $Pb(CO_3)_2^{2-}$	(L8)	10.33
$Pb^{2+} + H^+ + CO_3^{2-}$ ° $PbHCO_3^+$	(L9)	12.59
$Pb^{2+} + SO_4^{2-}$ ° $PbSO_4(aq)$	(L10)	2.73
$Pb^{2+} + 2\ SO_4^{2-}$ ° $Pb(SO_4)_2^{2-}$	(L11)	3.50
$Pb^{2+} + Cl^-$ ° $PbCl^+$	(L12)	1.6
$Pb^{2+} + 2\ Cl^-$ ° $PbCl_2(aq)$	(L13)	1.8
$Pb^{2+} + 3\ Cl^-$ ° $PbCl_3^-$	(L14)	1.7
$Pb^{2+} + 4\ Cl^-$ ° $PbCl_4^{2-}$	(L15)	1.4
$Pb^{2+} + F^-$ ° PbF^+	(L16)	2.06
$Pb^{2+} + 2\ F^-$ ° $PbF_2(aq)$	(L17)	3.42
$Pb^{2+} + H_4SiO_4(aq) + 4\ H^+ + 6\ F^-$ ° Pb-F-$SiF_5(aq) + 4\ H_2O$	(L18)	35.18†

*Values derived from Table 4-16 in reference 30 at 25 EC and zero ionic strength. These equilibria are used in the construction of Figures 1-4.

†Computed from combining the dissociation constant for the reaction $Si(OH)_4 + 4\ H^+ + 6\ F$ ° $SiF_6^{2-} + 4\ H_2O$, log K = 30.18 (from reference 28) with Equation (8), but using an extremely exaggerated hypothetical value for K in equation (8). We believe this value to be an intentional overestimate by a factor of at least 1000-2000 over the likely value of the true stability constant, which has not been measured.

One might logically inquire whether PbF_2 can precipitate under drinking water or physiological conditions. The solubility product expression for plumbous fluoride is:

$$PbF_2(s) \text{ ° } Pb^{2+} + 2\ F^-,\ K_{sp} = 10^{-7.44} \quad \text{(reference 18)} \qquad (10)$$

One way to estimate the minimum amount of lead in solution needed to precipitate lead fluoride would be to back-calculate from the concentration of the solubility of the aqueous uncharged difluorolead(II) coordination complex, $PbF_{2(aq)}$, the concentration of which can be obtained by combining equations (L17) and (10). This yields: $[PbF_{2(aq)}]_{max} = 9.5 \times 10^{-5}$ M. *Pretending that there are no competing metal cations and no competing coordinating ligands*, the total Pb(II) concentration is given by eqn (11):

$$[Pb^{II}]_T = [Pb^{2+}] + [PbF^+] + [PbF_2] = [Pb^{2+}] (1 + \beta_1[F^-] + \beta_2[F^-]^2) \quad (11)$$

where β_1 and β_2 come from eqns L16 and L17, respectively. In 1.0 mg L^{-1} *free* fluoride (5.3×10^{-5} M) solution, the approximate fractional speciation is as follows:[4] $f_{Pb^{2+}} = 99.904\%$, $f_{PbF^+} = 0.096\%$, and $f_{PbF_{2(aq)}} = 0.000099\%$. We draw attention to the fact that, in fluoridated water, the number 5.3×10^{-5} M really refers to the total fluoride, which is expressible as (12):

$$[F^-]_T = [F^-] + [PbF^+] + 2\,[PbF_2] = 5.3 \times 10^{-5}\ M \quad (12)$$

Nevertheless, because $[F^-]_T \approx [F^-]$ (less than 0.1% difference), there is no point in distinguishing between these two concentrations. However, in a real water, there are other metals competing for fluoride and other ligands competing for lead(II). The competition of other metal cations for fluoride as a ligand substantially reduces the free fluoride concentration. Thus, the required concentration of lead(II) in solution would be impossibly high:

$$[Pb^{II}]_T \geq \frac{[PbF_2(aq)]}{f_{PbF_2(aq)}} \geq \frac{9.5 \times 10^{-5}\ M}{0.000\ 000\ 99} \approx 9.6\ M \quad (13)$$

To make the situation even more extreme, aluminum, iron(III), calcium, magnesium, and copper(II) all compete with lead(II) for fluoride. Meanwhile hydroxide, carbonate, phosphate, and sulfate compete with fluoride for lead(II). The net result of these simultaneous competitions is that PbF_2 cannot precipitate as a solid. Even with an extremely high 90th percentile lead(II) level of ~210 µg L^{-1} (~ 1 µM), plumbous fluoride would be orders of magnitude from precipitating.

The formation of soluble fluoro-complexes of Pb(II) is governed solely by the stability equilibria, and no simple stoichiometric ratio exists among the concentrations of lead(II), fluoride, and the fluor-complexes. If ligand availability alone were the determining factor, chloride itself would usually be far more important than fluoride. Considering the relative stability constants for the complexes given in Table 3, a chloride concentration of 50 mg L^{-1} (= 1.4 mM = 1400 µM) is about 26 times the normal fluoride concentration. In the vast majority of all cases in drinking water, concentrations of lead(II) complexes with chloride (and even sulfate) considerably exceed those of fluoride.

3. Do fluoridation additives significantly affect the acidity and pH of consumed drinks constituted with tap water?

How does the acid contribution from hexafluorosilicate hydrolysis compare with that from other sources of acidity? Deionized water treated to contain 1 mg L^{-1} fluoride would contain 53 µM HF(aq). If one were to drink this solution of 53 µM HF(aq), which is 93 mol% dissociated to hydrogen and fluoride ions, it would contain 49 µM H^+ and its solution would have a pH of 4.3. We will show later that drinking water contains buffering components that essentially neutralize even this effect. Meanwhile, the high extreme for stomach pH (lowest acidity) is about 3 (1000 µM H^+); the lowest stomach pH is about 1.5 (for optimal pepsin enzymatic activity in the digestion of protein). At pH 3, roughly half of the HF will not ionize since it is a weak acid. Meanwhile, some foods are equally or more acidic, for example, apple juice (pH 2.9).

If this reasoning were correct, consuming soft drinks made with unbuffered tap water should be high risk, given the high concentrations of complexing organic acids (*e.g.*, citric and tartaric acids in powdered fruit drink mixes) or inorganic acids (*e.g.*, phosphoric and carbonic acids in colas). In fact, Coleman *et al.* showed that chelating organic bases (*e.g.*, citrate, ascorbate, EDTA) promote the transport of lead(II) in the small intestine.[31] The acidic components of these beverages completely overwhelm the contribution from HF in the water used to prepare them. Whether any of these other species is present in sufficient concentration to influence bioavailability is unknown. Regardless, acid from hexafluorosilicate-based fluoridation is negligible compared to other dietary sources. Consequently, one cannot demonstrate that an increase in blood lead(II) ion levels can be linked to acidity from SiF_6^{2-} hydrolysis any more than one can demonstrate it results from consuming soft drinks.

In the small intestine, bile (produced by the gall bladder) and bicarbonate (secreted by the pancreas) raise the pH and effectively buffer against pH change. Partly digested food in the chyme also acts as a buffer. Moreover, normal gastric biophysiology resists changes in acidity by a mechanism involving gastrin secretion and activity for which a detailed description is beyond the scope of this work. In conclusion, the production of acid from fluoridation of water is insignificant when compared to other acids and bases supplied by a normal diet or physiological mechanisms.

4. What are the concentrations of the lead(II) and fluoride species in a typical drinking water?

To test different hypotheses about the impacts of fluoride ligands on lead solubility, several aqueous solutions were modeled with MINEQL+.[32] The effect of background ions such as CO_3^{2-}, HCO_3^-, and PO_4^{3-} and water quality parameters such as pH have been extensively investigated and reported in the water treatment literature.[26,30,33,35-43] Free lead(II) ion, Pb^{2+}, is a very small fraction of the soluble lead in most drinking water systems.

We have taken into account equilibria of lead(II), aluminum, calcium, and other metals for such ligands as carbonate, chloride, hydroxide, sulfate, and, of course, fluoride. These were given earlier in Tables 2 and 3. Other necessary equilibria and their constants that we have used for this modeling exercise are shown in Table 6.

Table 6. Other equilibria used to calculate the fractional distribution of aqueous species*

Equilibrium	Eqn	log $
$CO_2(g)$ ° $CO_2(aq)$	(E1)	–1.468
$CO_2(aq) + H_2O$ ° $HCO_3^- + H^+$	(E2)	–6.352
HCO_3^- ° $CO_3^{2-} + H^+$	(E3)	–10.329
$Na^+ + HCO_3^-$ ° $NaHCO_3(aq)$	(E4)	–0.25
$Na^+ + CO_3^{2-}$ ° $NaCO_3^-$	(E5)	1.27
$Ca^{2+} + HCO_3^-$ ° $CaHCO_3^+$	(E6)	1.106
$Ca^{2+} + CO_3^{2-}$ ° $CaCO_3(aq)$	(E7)	3.224
$Mg^{2+} + HCO_3^-$ ° $MgHCO_3^+$	(E8)	1.07
$Mg^{2+} + CO_3^{2-}$ ° $MgCO_3(aq)$	(E9)	2.98
$Si(OH)_4(aq)$ ° $SiO(OH)_3^- + H^+$	(E10)	–9.83
$Si(OH)_4(aq)$ ° $SiO_2(OH)_2^{2-} + 2\ H^+$	(E11)	–23.0
HSO_4^- ° $SO_4^{2-} + H^+$	(E12)	–1.988
$Ca^{2+} + SO_4^{2-}$ ° $CaSO_4(aq)$	(E13)	2.30
$Mg^{2+} + SO_4^{2-}$ ° $MgSO_4(aq)$	(E14)	2.37
$Al^{3+} + SO_4^{2-}$ ° $AlSO_4^+$	(E15)	3.02
$Al^{3+} + 2\ SO_4^{2-}$ ° $Al(SO_4)_2^-$	(E16)	4.92
$Al^{3+} + HSO_4^-$ ° $AlHSO_4^{2+}$	(E17)	0.46
H_2O ° $H^+ + OH^-$	(E18)	–14.00
$Na^+ + H_2O$ ° $NaOH(aq) + H^+$	(E19)	–14.18
$Ca^{2+} + H_2O$ ° $CaOH^+ + H^+$	(E20)	–12.78
$Mg^{2+} + H_2O$ ° $MgOH^+ + H^+$	(E21)	–11.44
$Al^{3+} + H_2O$ ° $AlOH^{2+} + H^+$	(E22)	–5.00
$Al^{3+} + 2\ H_2O$ ° $Al(OH)_2^+ + 2\ H^+$	(E23)	–10.1
$Al^{3+} + 3\ H_2O$ ° $Al(OH)_3(aq) + 3\ H^+$	(E24)	–16.9
$Al^{3+} + 4\ H_2O$ ° $Al(OH)_4^- + 4\ H^+$	(E25)	–22.7
$Al^{3+} + SiO(OH)_3^-$ ° $AlH_3SiO_4^{2+}$	(E26)	–0.785

*Values taken from reference 29, except (E26) from reference 32. These equilibria are used in the construction of Figures 1–4. Equations (59)-(60), (67)-(68), and (75) are used for Figure 5.

Calculations were performed for the following hypothetical water solutions, as a means to test some plausible limits on when fluoride or fluorosilicate complexes might be of consequence with respect to solubility. Conditions are summarized in Table 7. The lead(II) concentration used represents the regulatory 90th percentile action level (AL) for public water supplies under the Lead and Copper Rule.[44-48] Because the general distribution of lead levels in residences and buildings is widely accepted to follow a lognormal pattern according to virtually all published research, this assumption will provide an example that is very biased towards the highest lead occurrence.[49-50] For all modeling, temperature was set to 25 °C, and an ionic strength of 0.005 M was assumed. Stability constants for important chemical species of lead are not available for other temperatures, but temperature effects on the speciation of other metals have not been reported to be so dramatic as to

affect the speciation by the orders of magnitude that would be necessary to change the conclusions of this investigation.

This background water is characteristic of many areas of the United States where geological and hydrological conditions create soft waters of low carbonate content (dissolved inorganic carbon, DIC) and ionic strength. These are the most susceptible waters for any effect of fluoridation on lead speciation, because strong competitive complexation by carbonate in higher DIC waters reduces the amount of lead complexed by fluoride, sulfate and chloride.[4] Small differences in hardness, silica concentration, or other major ions will have mimimal impact on the aqueous lead speciation.

The concentration of aluminum in Table 7 represents a moderate to somewhat high residual carried over from coagulation with aluminum sulfate (alum), as commonly occurs with surface water treatment plants. The point of addition of fluoride varies widely, from before coagulation to the clearwell or entry point to the distribution system. The final effluent pH is often adjusted for corrosion control and the Lead and Copper Rule, ofsetting acidity created by fluoride chemical addition.

Table 7. Water quality parameters for speciation modeling

Species	Concentration, mg L^{-1}	Concentration, mol L^{-1}
$[SiO_2]_T$†	5.0	8.3×10^{-5}
$[Pb^{2+}]_T$‡	0.015	7.2×10^{-8}
$[F^-]_T$+	1.0	5.3×10^{-5}
$[CO_2]_T$° as C	5.0	4.2×10^{-4}
Ca^{2+}	5.0	1.2×10^{-4}
Mg^{2+}	2.0	8.2×10^{-5}
Na^+	10.0	4.4×10^{-4}
Al^{3+}	0.20	7.4×10^{-6}
Cl^-	10.0	2.8×10^{-4}
SO_4^{2-}	5.0	5.2×10^{-5}

†$[SiO_2]_T$ = total silicon(IV) concentration, expressed as silicon dioxide.
‡$[Pb^{2+}]_T$ = total lead(II) concentration, all species.
+$[F^-]_T$ = total fluoride concentration = $[F^-] + [HF] + \sum n[MF_n^{(q-n)}]$.
°$[CO_2]_T$ = DIC = $[CO_2(aq)] + [H_2CO_3] + [HCO_3^-] + [CO_3^{2-}]$ (dissolved inorganic carbon). Mass-based concentration is expressed as C not CO_2).

As noted previously, for these calculations we intentionally exceedingly overestimated the highest conceivable value for the equilibrium constant for equation (8), using a value of 10000. The summary plots (Figures 1-4) clearly show that hexafluorosilicate and fluoride complexes are minuscule contributors to lead(II) in a drinking water matrix. Figure 1 shows the computed relative

amounts of the associations of fluoride with hydrogen ion, dissolved silica, aluminum, lead and the group calcium+magnesium+sodium. Figures 2 and 3 show that Pb^{2+}(aq) is the dominant species only at low pH, with the bicarbonate and carbonatolead(II) complexs already beginning to dominate soluble lead speciation by pH 7. By pH . 7.2, the hydroxolead(II) ion also exceeds the free lead(II). As pH increases to - 8.4, only - 1% of the total lead is the free aquated ion. In Figures 2 and 3, we see that the mono- and difluorolead(II) complexes always account for less than 0.3% of the total lead(II). Note that the species $PbSiF_6^0$ is present at such low concentrations that we would expect to find *only one molecule of this complex in more than 1000 liters of tap water* at pH 6, which of course, far exceeds the volume possible for water consumption and the human stomach. Note the broken ordinate.

In Figure 4, we show the minor species, including the sulfato-, fluoro-, and chloro-complexes of lead for this hypothetical but realistic water. The carbonato-complexes of lead(II) are much stronger than the halo-complexes—as reflected by their stability constants, which are 5-8 orders of magnitude higher than those of the comparable halide complexes, combined with the higher molar concentration of total DIC ($[CO_2]_T$). Since the increase in lead complexation by carbonate has been shown elsewhere,[26,30] we did not repeat the calculations here. Clearly, at the higher $[CO_2]_T$ concentrations, the fluoro-complexes become even less significant. We note that there is less than one molecule of $PbSiF_6$ per liter of water even with the extreme exaggeration of the possible value for the stability constant with the fluorosilicate anion.

The insignificance of any SiF_6^{2-} can be logically determined another way. Even if the formation constant for a hypothetical $PbSiF_6$(aq) complex were ten times higher than the strongest complex found by Haque and Cyr,[25] it would have a similar stability to PbF^+. Assuming all of the fluoride present in drinking water were SiF_6^{2-}, Figures 1-4 show that it would still be approximately 3 (pH 6) to more than 6 (pH 10) orders of magnitude lower than the soluble lead level, which is governed by the concentrations of other Lewis bases. Because complexation with carbonate and bicarbonate dominates aqueous lead speciation at drinking water pH, any increased $[CO_2]_T$ level makes contribution of the fluoro-complexes to $[Pb^{II}]_T$ even less significant. The bar graphs in Figures 3 and 4 clearly illustrates how free lead(II), hydroxo-, and (bi)carbonato-complexes dominate the speciation of lead(II) at all drinking water pH values while fluoro-complexes are always in the minority.

5. Can fluorosilicates or fluoride affect the pH of a finished water?

We have previously stated that naturally occurring buffers have a significant impact on drinking water chemistry. At this point, we will quantitatively illustrate the magnitude of this impact. Figure 5 shows the buffer intensity (capacity) $\mathscr{B}$ as a function of pH.[e] The buffer intensity is a

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[e]Buffer intensity is usually represented by the symbol $, which we find to be an unfortunate coincidence as it leads to confusion between this quantity and cumulative stability constants, for which $ is often used simultaneously. As a result, here we have broken with convention and use the symbol $\mathscr{B}$ to stand for buffer intensity.

quantitative description of a solution's resistance to changes in pH upon addition of acid or base and is defined as (14):

$$\mathscr{B} = -dC_a/d(pH) = dC_b/d(pH) \quad (14)$$

where C_a is the *formal* concentration (formality) of added acid and C_b is the formality of added base. Because we are discussing infinitesimal (differential) quantities of added acid or base, the effects are equal and opposite for added acid versus added base. Note that the derivative described in eqn 14 is the slope of a curve of C_b versus pH, which is the inverse of a "titration curve" where base molecules are added directly to a solution of acid so that the titer is zero and there is no change in volume, only changes in concentration.

Virtually all potable waters contain some dissolved inorganic carbon, represented here as $[CO_2]_T$; therefore, the buffer intensity will be controlled by the simultaneous conjugate acid-base equilibria of the carbon dioxide-carbonic acid-bicarbonate-carbonate system, unless it is very low in concentration relative to orthophosphate or other weak acid/base systems. Although not conceptually difficult to understand, the derivation of a quantitative definition of the buffer intensity $\mathscr{B}$ for a given system can be cumbersome. We have previously written about the importance of buffer capacity and we and others have derived the formulae for a diprotic species, such as carbonic acid.[26,51-53]

$$\mathscr{B} = (\ln 10)\left[\frac{K_w}{[H^+]} + [H^+] + [CO_2]_T\left(\frac{\beta_1[H^+] + 4\beta_2[H^+]^2 + \beta_1\beta_2[H^+]^3}{(1 + \beta_1[H^+] + \beta_2[H^+]^2)^2}\right)\right] \quad (15)$$

where $\mathscr{B}$ has units of M (pH unit)$^{-1}$ when all concentrations are expressed in molarities. Upon inspection of (15), it can be seen that the buffer intensity can readily be divided into contributions from $[OH^-]$, $[H^+]$, and $[CO_2]_T$.

Figure 5 shows an illustration of a water with a $[CO_2]_T$ of 5 mg L^{-1} as C and 3 mg L^{-1} as PO_4 of an orthophosphate corrosion inhibitor at 25°C. It can clearly be seen that much of the buffer intensity is derived from the carbon dioxide-bicarbonate-carbonate system, and that contribution gets more and more significant as $[CO_2]_T$ increases. For the example water used in these calculations, the minimum buffer intensity contributed by the $[CO_2]_T$ and water is approximately $\mathscr{B}$ = 0.25 mM (pH unit)$^{-1}$. The contribution of acid from undissociated SiF_6^{2-} can again be proved negligible from the following extreme example. Even if 10% of the total fluoride input were as SiF_6^{2-} at pH 7, the acid input would be $C_a = 5 \times 10^{-6}$ M. I = 0.005 M, T = 25 °C. Thus, we can compute the change in pH directly using the buffer intensity calculated above: $\Delta(pH) = -C_a/\mathscr{B}$ = –0.0050 mM/[0.25 mM (pH unit)$^{-1}$] = –0.020 pH unit. This value is within the limits of a linear approximation of buffer capacity. Such a small effect on pH is analytically undetectable and inconsequential with respect to other sources of variability in factors affecting lead release from plumbing materials. Masters and Coplan assert without any field data or simple calculation, that slow dissociation of silicofluoride in distribution systems can increase acidity and increase lead release.[2] However, to the contrary, the concepts of chemical equilibria are well-established and measured equilibrium constants are

sufficiently accurate and precise to show that fluoride and fluorosilicate essentially do not affect the solubility distribution of lead(II) species under potable water conditions. Many water systems are also compelled under the Lead and Copper Rule to conduct distribution system pH monitoring, and dangerous pH decreases from any cause would usually be uncovered and treatment adjustments made.

6. Can additives be responsible for contaminants in the water supply?

Water treatment chemicals are subject to National Sanitation Foundation specifications, which require that additives contain a *maximum allowable level* (MAL) less than or equal to 10% of the *maximum contaminant level* (MCL) for any regulated contaminant in the national primary drinking water standards.[54] It is worth reviewing how fluoridation chemicals are made to see whether there are any steps where contamination could occur. Most hexafluorosilicate and fluorosilicic acid are derived from the processing of phosphate rock by the fertilizer industry.[55] In this process, apatite and fluoroapatite (which can be thought of as a blend of fluorite and apatite for this purpose) are decomposed with sulfuric acid. HF and SiF_4 are removed as gases so that there is little chance of lead contamination from the crushed rock. The resulting 23% w/w hexafluorosilicic acid is a strong acid and quite corrosive, but no evidence has been put forth to suggest that this additive has become contaminated prior to use. Moreover, testing either the water at the plant or the stock fluoridating agent itself would also be sufficient to rule this out.

7. What is required to have a valid sampling scheme for measuring lead(II) intake from tap water? How do regulation-required samplings relate to exposure?

The total lead in a first draw sample mostly reflects the nature of the building plumbing system. A one liter sample volume dominantly represents the metals picked up in contact with the last approximately 17 to 26 feet of plumbing material before the consumers' tap, presuming " ½-inch" pipe of commonly-used materials. Comprehensive water sampling for epidemiological and other health effects studies for lead(II) is logistically complicated and expensive; therefore, it is very tempting to try to use available regulatory tap water monitoring data for this purpose. The temptation must be resisted, however as the monitoring program specified in the United States drinking water regulations is both statistically and physically invalid for this purpose, and *was never intended to be an exposure assessment* sampling program.[44,48-50] The regulatory targeting scheme is intentionally biased towards reducing the highest lead exposures through central water treatment. It does not capture the highest copper exposures. It does not give any information on the levels of metals to which the general population is exposed from old non-lead plumbing materials, or many other corrosivity-related characteristics too numerous to list.

As previously noted, the vast preponderance of the lead(II) in nearly all tap waters originates from the plumbing materials located between the water distribution mains and the end of the faucet used by the consumer. Individuals consume water under innumerable combinations of volumes of water, interior plumbing system configurations and ages, and lengths of stagnation of the water in the plumbing between uses. Data reported from many tap water sampling experiences throughout

the US and Europe indicate tap water lead levels tended to follow a log-normal distribution, and both within-site and between-site variability tended to be large relative to the lead(II) concentrations.[49,50] Keeping this in mind, the American standard for lead in drinking water was crafted to focus on the lowering of lead(II) levels by central water treatment for the plumbing configurations most likely to represent nearly the worst cases for the most vulnerable humans, *i.e.*, infants, children, pregnant women (see Q6). Some attempts have been made to define reasonable statistical bases for comparing soluble metal release from parallel pipe loops used for corrosion control testing, and the required number and frequency of samples directly relates to the intrinsic variability of the metal release and the confidence levels one wishes to place in the characterization of the metal levels.[56,57] After a cursory examination of the requirements for a statistically valid sampling program accounting for needed levels of predictive confidence across all sources of variability observed, one realizes that it would take literally hundreds or thousands of samples at great frequency from cities of all sizes to try to adequately characterize tap water lead levels for even a single uniformly applied national sampling protocol.

Obviously, the water chemistry at the point the distributed finished water enters the domestic or commercial building plumbing system plays a very significant role in affecting lead release into the water, but many other physical factors also operate.[26,30,33-36,49,50,56,58,59] The water at this point may have undergone chemical changes during its passage through the distribution system from the treatment plant or well, and changes in treatment or changes in water sources may also cause the chemical characteristics of the water to change periodically, especially in such important aspects as pH, and concentrations of alkalinity, natural organic matter, oxidant levels, and a variety of potentially aggressive anions. Even the season may influence lead levels in complicated ways, by changes in ground temperature, or temperatures in buildings where pipes run through basements, unheated crawl spaces, concrete slabs, or nearby heating or air conditioning ducts. A single snapshot sampling event cannot capture this.

The drinking water literature is full of papers that show how difficult it is to correlate lead levels with any one or even a mix of several water quality parameters (even when frequently sampled and sophisticated statistics are applied[60]), and a complete discussion of the matter is beyond the scope of this article. There may be countless other physical or chemical quantities that may be statistically correlated with lead(II) levels but nonetheless be totally unrelated mechanistically. Clearly, aggregate measures such as a small number of first-draw or fully-flushed water samples taken infrequently from an intentionally biased relatively small pool of sampling sites throughout a water system cannot quantitatively and precisely predict the exposure of any individuals to lead from drinking water. To accurately determine lead(II) intake, sampling schemes using diverters or proportional sampling devices that capture a representative fraction of the water actually drawn at the faucet by the consumer seem to be the only feasible approach.[30] Interestingly, the bibliographies of the Masters and Coplan study most strongly asserting the adverse effects of silicofluoride shows only a single reference related to sampling of drinking water or the control of lead or other metals by water treatment, so the level of awareness in the design of the studies and interpretation of the data is highly questionable. By not measuring or statistically testing numerous other water and plumbing characteristics that could correlate with lead(II) levels with equal to or greater statistical significance than those relationships that were put forth, the studies of Reference 2 are intentionally biased towards what appears to be a preconceived conclusion. Even simple analytes that are known

to affect lead mobility, such as pH or alkalinity, or analytes known to play important dietary roles in health, such as calcium, sodium or magnesium, were not reported to be measured in their study, so possible confounding variables are conspicuously excluded from evaluation.

Needless to say, tap water intake is highly variable with beverage preferences of individuals, and that factor needs to also be taken into account in any assessment of exposure and behavioral implications.

8. How is lead(II) concentration measured in blood?

The best methods that analytical chemistry has to offer are spectroscopic in nature: AAS, ICP-MS, or ICPS. Quick screening tests, as we have pointed out previously,[4] have several weaknesses in terms of precision and accuracy. What is appropriate to screen children for exposure prior to a more expensive and more elaborate test is not necessarily appropriate for investigating overall lead exposure from drinking water. Acceptable uncertainty in a yes-no screening test, for instance, would not be appropriate when seeking a quantitative relationship.

Masters and Coplan did not give the total lead concentrations in the first draw water samples, so we cannot directly compare blood lead levels with water lead levels.[2] They did give blood lead levels divided up by those water systems where first draw samples were divided by a cut-off of 15 μg L^{-1} of lead(II); see Table 5.[f]

Table 5. $[Pb^{II}]_{blood}$ (μg L^{-1}) for fluoridation processes*

$[Pb^{II}]_{water}$	none	NaF	Na_2SiF_6	H_2SiF_6
<15 μg L^{-1}	19.7	21.1	23.7	23.1
n =	86	31	6	26
>15 μg L^{-1}	21.8	19	43.8	32.7
n =	29	8	1	25

*Taken from reference 2.

Because the sodium hexafluorosilicate data are based on one system with $[Pb^{II}]_{water}$ > 15 μg L^{-1}, it is impossible to treat that value as significantly supporting any hypothesis. Without some estimation of the uncertainties of $[Pb^{II}]_{blood}$, we also cannot be assured that 23 μg L^{-1} is distinct from 33 μg L^{-1}. Reporting 3 significant digits in the blood lead(II) concentrations seems suspect. We expect that the numbers are probably good to about 10–15%. Masters and Coplan also failed to include the possibility of naturally occurring fluoride and silicates in the unfluoridated water systems, which

[f] It is unclear from where these numbers originated. Reference 2 mentions averaged 90th percentile values. We take this to mean that 90th percentile values from two or more rounds of regulatory testing were averaged. Given that the normal distribution of monitoring data contains many non-detects, and the number of samples varies somewhat with system size so that the number and extent of values above the reported 90th percentile is unpredictable, the meaning of this table is very difficult to determine.

would be necessary to substantiate their thesis, as naturally occurring silica and fluoride should chemically react to produce the same effects.

In the Masters and Coplan studies that most strongly assert to implicate drinking water fluoridation in lead neurotoxicity,[1,2] there is no report of efforts to obtain appropriate exposure data and then attempt to correlate the consumed water quantity and quality from an individual building or house with the blood lead levels of individuals residing or spending significant time there. There is no indication that there is any connection between sampled taps and sampled persons. In other words, nothing indicates that a person living in the sampled house had his blood drawn. Instead, the authors rely on quartile divisions of both water lead levels and blood lead levels. For there to be a correlation between the lead(II) levels in blood and water, there must be a link between the samples. It is possible that the highest blood levels of lead(II) are closely linked to other exposures, such as paint, soil, or mine run-off. The authors appeared to notice and somewhat acknowledge the [unsurprising]lack of correlation of the water lead levels with blood lead levels in one part of their paper,[2] even after postulating earlier in the paper that slow dissociation of the fluoridation chemical would cause more acidity and more lead release. To overcome this, they then invoke a kind of black-box "...biochemical effects..." to justify their conclusion. The postulation of some "...chemical effects that maintain lead in suspension..." is contradictory, because that would be reflected in higher lead levels at the tap using regulatory analytical procedures. Another interesting apples-*versus*-oranges comparison is made where they attempt to see if silicofluoride could enhance lead uptake for exposure to lead paint and dust by looking at old housing (pre-1940 and post-1940) combined with other data that includes 90th percentile lead levels. Of course, the targeting scheme for the sampling sites under the Lead and Copper Rule have only the most indirect of relationships to housing age, and houses can easily be remodeled, repainted and re-landscaped making those differentiations very problematic.

Thus, when all of these methodological problems are coupled with the failure to account forthe quantitative level of fundamental chemical interactions, the relationships posed between any lead(II) speciation and water fluoridation become unjustifiable.

9. What are the routes of lead exposure besides drinking water?

Exposure routes have been the subject of multiple studies. The problem is further complicated by incomplete understanding of subacute toxicity and dose-response.[61] Much of the exposure to lead occurs through dust, air-borne particulates, soil, paint, ceramic glazes, and sundry other sources, including drinking water.[62-65] One of the special concerns for drinking water is that the lead(II) appears to be far more bioavailable.[62] This is probably because aqueous lead(II) is far more likely to pass through mucous membranes than insoluble plumbous minerals. However, there is some evidence to suggest that even insoluble minerals can release lead(II) when ingested under the right conditions.[66] A number of studies[g] have concentrated on other factors affecting bioavailability and bioabsorption, including other nutrients, alcohol, cigarettes, water hardness, plumbing, and

[g]The references cited here include a representative sampling over the last two decades of the kinds of work that have been done. These references are not intended to comprise a complete listing or review of the studies in this area.

lifestyle.[37,67-72] At least one study has also shown some data indicating that lead associated with orthophosphate (either ingested as particles or simultaneously ingested in solution may be less bioavailable through the intestinal system, because of the higher levels of phosphate present in that organ causing the formation of insoluble lead phosphate particles that would not be readily absorbed. This is very interesting from a drinking water perspective, because of the widespread use of orthophosphoric acid or orthophosphate-containing corrosion inhibitor chemicals. The main conclusion that can be drawn from these studies is that the biological availability, absorption, and accumulation of lead and its compounds depend on a wide variety of factors, making this a very complicated puzzle to solve. [39]

Conclusions

Recent reports[1,2] that purport to link certain water fluoridating agents, such as hexafluorosilicic acid and sodium hexafluorosilicate to human lead uptake are inconsistent with accepted scientific knowledge. The authors of those reports fail to identify or account for these inconsistencies, and mainly argue on the basis of speculation stated without proof as fact. The sampling scheme employed in the studies is entirely unrelated to any credible statistically-based study design to identify drinking water lead and fluoride exposure as a significant source of blood lead in the individuals. The authors use aggregated data unrelated in space and time and then attempt to selectively apply gross statistical techniques that do not include any of thousands of other possible water quality or exposure variables which could show similar levels of correlation utterly by accident. Many of the chemical assumptions are scientifically unjustified, are contradicted by known chemistry data and principles, and alternate explanations (such as multiple routes of Pb^{II} exposure) have not been satisfactorily addressed. The choice in water fluoridation approach is often made for economic, commercial or engineering reasons that may have a regional component that could also be related to various community socio-economic measures, and so should not be considered to be a purely independent variable without investigation.

At present, the highly-promoted studies asserting enhanced lead uptake from drinking water and increased neruotoxicity still provide no credible evidence to suggest that the common practice of fluoridating drinking water has any untoward health impacts via effects on lead(II) when done properly under established guidelines so as to maintain total water quality. Our conclusion supports current EPA and PHS/CDC policies on water fluoridation.

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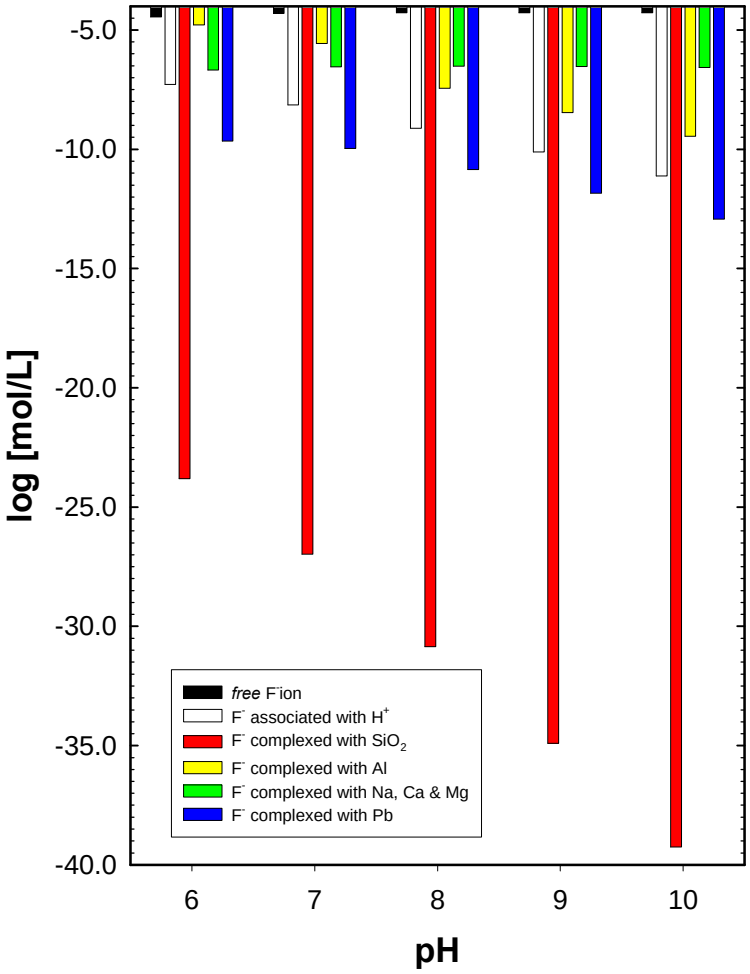

Figure 1. Illustration of concentration of fluoride species bound to different metal groups for hypothetical low-DIC water, assuming 15 µg L^{-1} Pb and background ion concentrations given in Table 7 of the text.

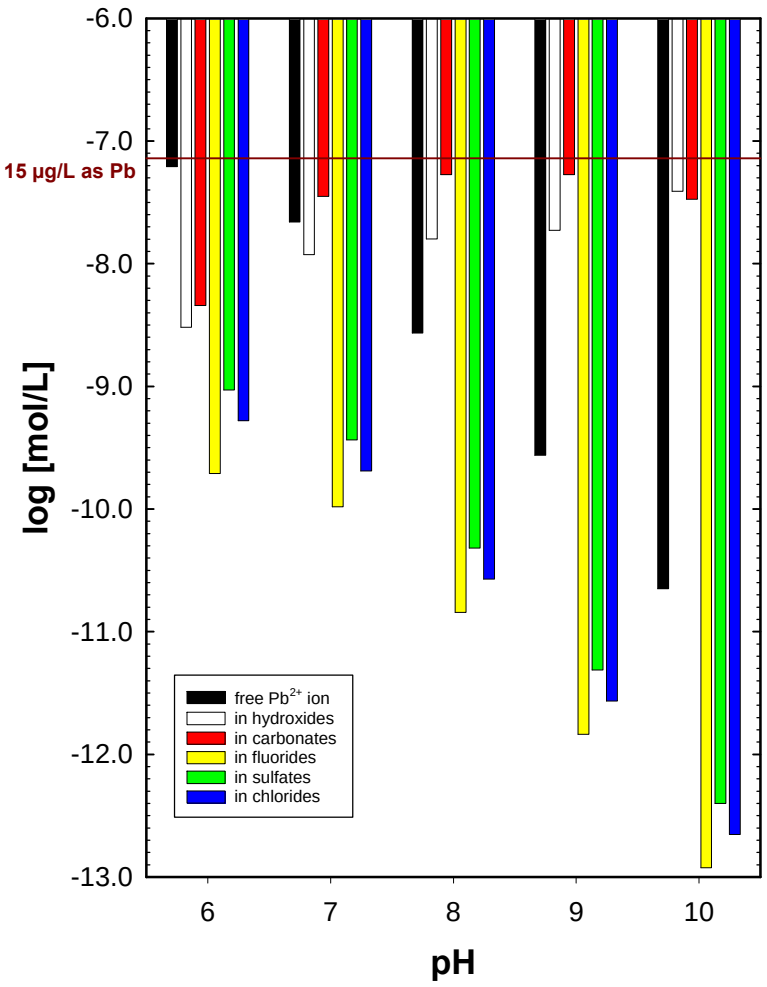

Figure 2. Illustration of concentration of soluble lead bound to different ligand groups for hypothetical low-DIC water, assuming 15 µg L^{-1} Pb and background ion concentrations given in Table 7 of the text.

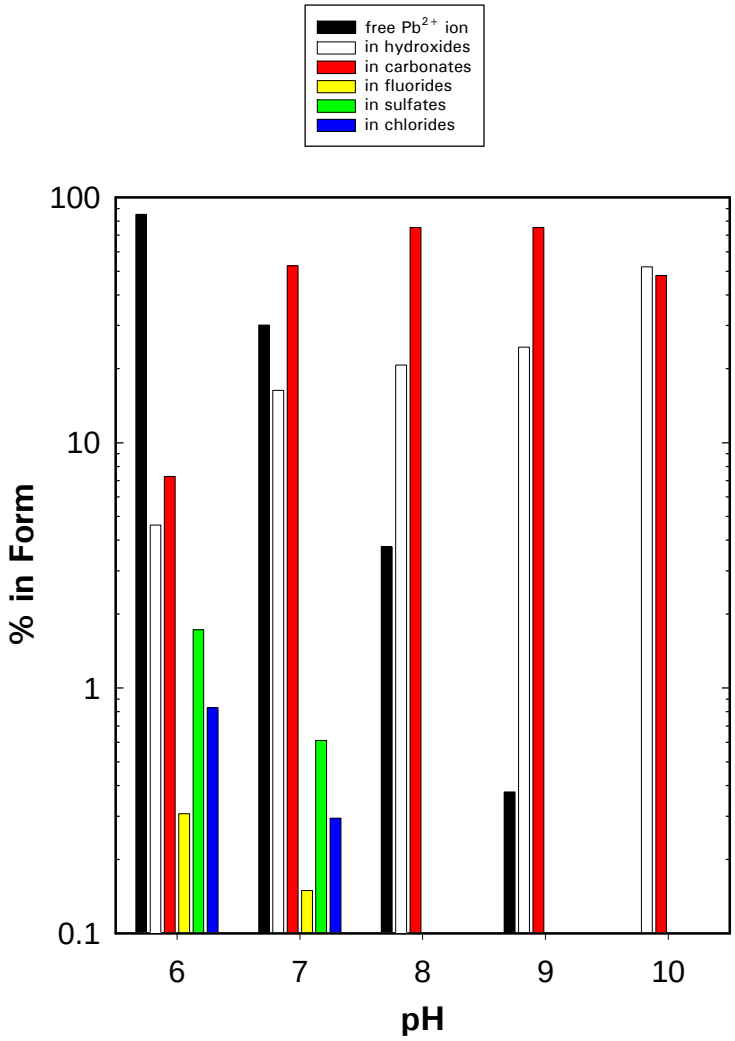

Figure 3. Illustration of fractions of soluble lead bound to different ligand groups for hypothetical low-DIC water, assuming 15 µg L^{-1} Pb and background ion concentrations given in Table 7 of the text. Note logarithmic scale for "% in Form.."

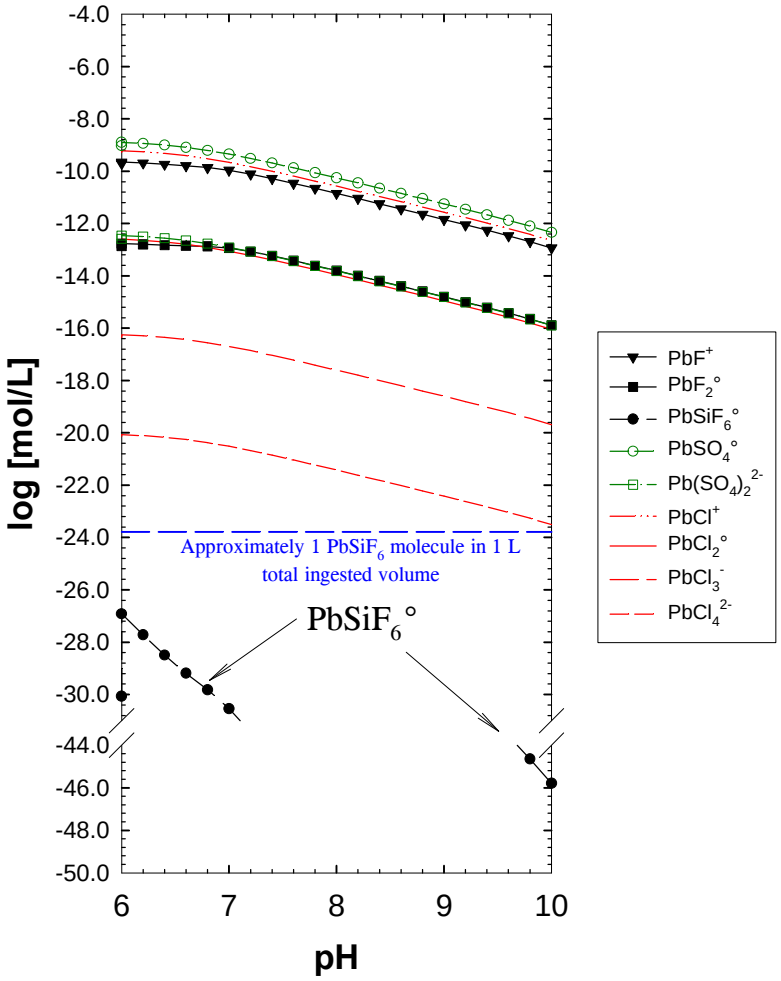

Figure 4. Minor lead species distribution in hypothetical water described in Table 7. Computations were done for 25°C, I=0.001. PbSiF6° complex was included in the model, assuming of log β = 5.

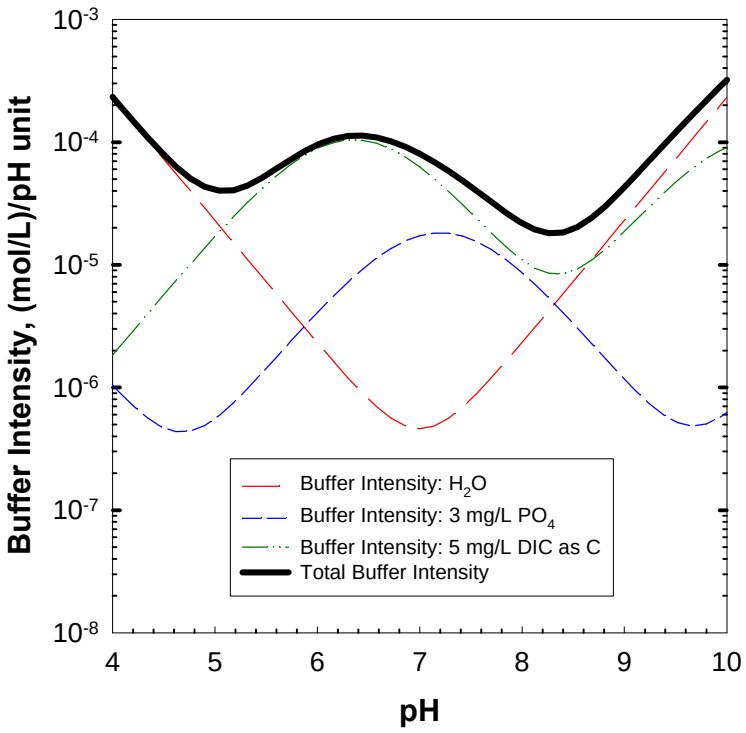

Figure 5. Components of buffer intensity for a hypothetical water with DIC = 5 mg L^{-1} as C (4.16×10^{-4} M), and orthophosphate at 3 mg L^{-1} as PO_4 (3.16×10^{-5} M) at 25°C.