

## Chemical Analysis of Poisoning from Fluoridated Public Water

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**Abstract.** The mechanism by which fluoride from unnatural sources exerts adverse effects in man and animals is examined. Low level artificial fluoridation of municipal water can cause well known alterations in teeth and bone structure with incorporation of fluoride into a wide array of tissues and increased hip fracture tendency, depending on years of exposure and water hardness. High accidental levels cause acute lethal poisoning and are responsible for fluoride listings on poisons registries and for their major industrial use as rodenticides, insecticides or pediculicides. Solubility calculations indicate that blood fluoride concentrations required to decrease calcium below normal physiological levels compare to those present in the tissues of poisoned victims and to those causing decreased beat rates in isolated heart cells in culture. The effects of calcium ion and pH over broad ranges on the free fluoride ion concentration were determined. Acute lethal poisoning with heart attack, and also many of the chronic 'low' level effects of fluoride, are mediated by calcium binding by fluoride ion. At a pH typical of gastric juice, approximately 50% of fluoride is protonated as hydrofluoric acid HF, with 50% remaining the free fluoride ion. The significance of these observations is discussed in terms of potential hazards, both short and long term, associated with consumption of artificially fluoridated waters of varying calcium content.

**Introduction.** All artificial fluoride compounds lack calcium and are listed toxic substances<sup>1,2,3,7</sup>. Calcium fluoride is found in nature and is not considered a toxic compound because of a comparatively very high lethal acute dose ( $LD_{50} = 3,750$  mg/kg). The fluoride compounds sodium fluoride and fluosilicic acid, used as water injectables, are synthesized artificially by industrial reaction and are classed as rodenticides, insecticides and pediculicides, with acute lethal doses in experimental animals comparable to arsenic and lead<sup>3</sup> ( $LD_{50} = 125$  mg/kg). Ocean salt water contains fluoride at 1 mg/kg (1 ppm) accompanied with extremely high calcium and magnesium levels that minimize toxicity.

Waters in the Southwestern U.S. can contain natural calcium fluoride along with other calcium salts.<sup>22</sup> Although fluoride has been the focus of involvement in structural alterations of teeth of consumers living in this region of the U.S., very high calcium levels always accompany the ion that are most likely involved in any positive effects originally discovered. The principal effect of fluoride on teeth incorporated from blood is to alter the structure of teeth interiors in a process known as fluorosis that can lead to mottling unsightly spots that are permanent on developing teeth<sup>9</sup>.

An array of scientific findings indicate that the decision made by many cities since World War II to inject artificial fluorides into municipal drinking water, as long as the dose is below a certain level (usually 1-4 part per million, 0.05-0.20 mM), to decrease the incidence of tooth decay, has caused significant unwanted biological effects, the extent determined by the acidity and the calcium and magnesium levels in the water and the duration of exposure<sup>1,8,13,18,19,21,24</sup>.

Municipal water supplies in the United States have been found to contain fluoride levels ranging from nearly absent to a record lethal accidental 7.5 mM<sup>8</sup>. Associated biologic effects have been diverse over

this dose range. In spite of much literature reporting toxic effects of municipal water fluoridation programs, the Public Health Service retained its mandate, now through the Oral Health Division of the Centers for Disease Control, to artificially fluoridate most major U.S. cities as soon as possible indefinitely and to recommend this to other cities throughout the world. Although fluoridating the bloodstream was thought to minimize tooth decay (as an alternative to topical fluoride or addition of fluoride to one's own consumed water) without adverse biologic effects to outweigh it, this has been the subject of much contention, since all artificial fluoride compounds are toxic calcium chelators.

Unfortunately, in 1992 at the mouth of the Yukon River in Hooper Bay, Alaska the unthinkable occurred. In what is considered an accident, an entire village was poisoned by its own fluoridated water supply when the system malfunctioned. Blood samples measured for incorporated fluoride and calcium ion, coupled with observed symptoms, provided useful pathologic information on the effects of high doses of fluoride assimilated from municipal drinking water supplies<sup>8</sup>. 296 residents were severely poisoned with one fatality. Most had heart malfunction-associated symptoms and severe gastrointestinal pain.

It is suspected that the conversion of fluoride ion into HF, hydrofluoric acid, occurs in the stomach due to the gastric acid HCl at pH 3 that caused the observed intense pain. Blood calcium levels dropped to 1/3 of normal in one victim, causing a heart attack and the loss of his life. Although the authors of the study were uncertain whether the fluoride itself caused the effect directly, or rather was due to its known ability to precipitate magnesium or calcium ion, reported here are computations that indicate low blood calcium is responsible for the lethal effect of acute fluoride poisoning.

In the absence of such overfeeds, consumption of fluoride treated water for two or more years leads to substantial precipitation of fluoride into bone<sup>13</sup> at thousands of times the level in the water and also into tendons and ligaments<sup>9</sup>. The condition known medically as fluorosis can be associated eventually with bone fragility<sup>2</sup> and can lead to hospitalization due to bone pain<sup>14</sup>, the severity depending on the fluoride level present, the hardness of the water and length of consumption.

Interestingly, in children raised on fluoridated water, teeth themselves can become more crumbly and subject to mottling or fluorosis, but no such effect on adult teeth occurs<sup>1</sup> (chap. 39, p. 896). Thus fluoridation of adult blood is unnecessary and indeed useless for this purpose.

The dean of Tulane University in New Orleans indicated that fluoridated water consumption at certain doses can eventually cause gum disease and for this reason New Orleans water was not fluoridated at the time Chicago and New York and other cities approved it<sup>1</sup>. Also, in 1960 under oath in Chicago, the individual for the Public Health Service who largely contributed to instituting the fluoridation idea admitted that his data constituting the scientific basis for fluoridation were invalid, shattering its foundation<sup>1</sup>. The original observation that people consuming water in Texas that happened to have 1 ppm

fluoride also had whiter teeth than usual was insufficient to justify mass fluoride addition to other public water supplies, because of adverse effects that also occurred, and the positive effects on teeth structure were explained by the accompanying high calcium levels in the hard water, in some regions to 500 ppm, rather than the fluoride.

The effects of fluoride in moderate soft water can be subtle enough to go unnoticed for many people for long time periods at the levels of fluoridation used currently in many U.S. municipal water supplies such as 0.05 mM. But since fluoride is converted in the stomach to hydrofluoric acid<sup>5</sup>, it is likely that consumption of fluoride at levels used in some cities is involved with ulceration of gastric and duodenal tissue (where the pH has yet to return to basic values that occur in the middle intestine) which are commonplace. Many report evidence in rats that it eventually causes cancer<sup>1</sup> and that in humans bone cancer in the U.S. is significantly higher in fluoridated cities in exposed young boys. In addition, approximately 1% of people are allergic to artificial fluoride exposure.

Some argue these effects are unimportant if a municipal supply maintains very low levels of fluoridation; but the longer the consumption occurs for an individual and the more elderly the person with less cell division occurring in the gastric mucosa, the more overt symptoms could potentially become. In addition, half of all ingested fluorides are retained permanently, mostly in bone. Whether accumulated fluoride in brain and other tissues is reversible or not is unknown.

We here determine whether and to what extent blood levels of calcium may be affected by various fluoride doses that are known to occur in the blood of fluoridated water consumers. Calculations shown are consistent with the notion that fluoride's lethal effects are due to low blood calcium subsequent to saturation of body fluids with fluoride at its known low solubility in the presence of physiologic levels of calcium that causes interruption of the heart beat. The effects of pH and calcium ion level in the medium on the free fluoride ion concentration were determined with a fluoride specific electrode. At gastric acidity levels, approximately 50% of ingested fluoride is hydrofluoric acid, and calcium but not monovalent cations substantially reduce the activity of the fluoride ion. The significance of these data are discussed in relation to hazards associated with artificially fluoride-drugged water

### **Analytical Results and Discussion**

Sublethal poisoning with artificial non-calcium-based fluorides occurs at 0.1-0.2 mM fluoride in blood<sup>3,7</sup> and lethal poisoning occurs in the 0.2 to 0.6 mM range due to heart failure (3). It is possible that this margin of safety is so slight between unnoticed effects (0.02-0.05 mM) to sublethal (0.1-0.2 mM) and lethal poisoning (0.2-0.6 mM) because at levels below the critical concentration of fluoride at which calcium fluoride precipitates, only chronic, unnoticed effects would occur. Much like being near a hot electrical wire, one can do so for a lifetime without difficulty, but one false movement too close to the wire could be disastrous.

With this in mind, we calculated the concentration of fluoride that would cause calcium fluoride precipitates to first form, from the known solubility product constant ( $K_{sp}$ ) for calcium fluoride ( $K_{sp} = 3.4 \times 10^{-11}$ )<sup>6</sup> and the known concentration of calcium ion in normal human blood (3 mM)<sup>5</sup>. The computed dose is 0.1 mM. Here the concentration of fluoride is:  $[F^-] = (K_{sp}/[Ca^{2+}])^{1/2}$  from the definition of the solubility product constant for insoluble salts where  $CaF_2 \rightarrow Ca^{2+} + 2F^-$  and  $K_{sp} = [Ca^{2+}][F^-]^2$  (see Table I). The concentration of blood fluoride where the blood calcium level would be lowered to the lethal low level of about 1 mM is 0.2 mM fluoride.

In Table I the calculated calcium levels that would coexist in fluid with a given fluoride level from solubility considerations are compared with actual measurements of blood levels of calcium and fluoride ion in the lethal poisoned human victim from Hooper Bay, Alaska. Note the good agreement between theoretically calculated fluoride levels, that should lower blood calcium ion to levels below normal, with the actual calcium and fluoride ion levels measured in the blood of this victim poisoned with fluoridated municipal water in Hooper Bay.

Unlike skeletal muscle, cardiac muscle requires extracellular calcium ion from the bloodstream to couple electrical excitation of the cell membrane with contraction of cardiac muscle fibers<sup>11</sup>. Each time the heart contracts, calcium fluxes into the heart cells from the extracellular fluid (at 3 mM calcium ion normally). When the heart relaxes, the calcium is pumped back out of the cell, allowing the fibrils to relax. Lowered extracellular calcium ion levels block contraction of the heart.

Indicated also are the below-normal calculated calcium ion level that would coexist with fluoride doses found to slow heart cell beat rates in detailed in vitro experiments<sup>10</sup>. Isolated mammalian beating heart cell preparations exhibit beat rates that are proportional to the calcium ion level in the incubation medium from 0.3 - 3 mM. Calcium chelating agents EGTA and EDTA and the calcium binding site competitor  $La^{3+}$  ion completely block excitation-contraction coupling, both in intact beating hearts and in isolated cell preparations<sup>11</sup>. Further, addition of fluoride to beating heart cell preparations slows beat rates in a dose-dependent manner that  $K_{sp}$  calculations indicate would lower calcium ion levels in the incubation medium (see Table I).

**Table I**  
**Inverse Relation Between F<sup>-</sup> and Ca<sup>2+</sup> Concentration\***

**Blood**

[Ca<sup>2+</sup>] [F<sup>-</sup>] (mM)

- 3.0 0.10 (F<sup>-</sup> calculated from K<sub>sp</sub>, for first precipitation of blood Ca<sup>2+</sup>)
- 1.3 0.48 (human blood measurements, Hooper Bay, Alaska victim)
- 1.2 0.18 (human blood calcium from Hooper Bay deceased victim\*\*)

**Incubation media**

- 1.4 0.15 (Ca<sup>2+</sup> calculated from K<sub>sp</sub> for F<sup>-</sup> added dose lowering heart cell beat rate 17%)
- 1.0 0.20 (F<sup>-</sup> calculated from K<sub>sp</sub> to lower blood Ca<sup>2+</sup> to 1 mM)
- 0.4 0.30 (Ca<sup>2+</sup> calculated from K<sub>sp</sub> for F<sup>-</sup> added dose lowering heart cell beat rate 27%)

\*Some cities recommend 0.1 - 0.2 mM fluoride be added to drinking water. Typically 1/5 or so of the water fluoride level is the consumers' blood fluoride level (as long as there are no accidents, equipment malfunction as in the Hooper Bay disaster, or miscalculated doses added); pharmacologic studies indicate that for soluble fluoride compounds, such as sodium fluoride, the ingested fluoride ion is fully absorbed from the gastrointestinal tract into the blood and in the kidneys about 90% of the glomerular filtrated fluoride is reabsorbed by the renal tubules. Perhaps differences between blood level and drinking water level are age dependent because ingested fluoride that is en route for deposition in bone, teeth and other locations cause a flux until such sites are more saturated at which time blood fluoride might then become as high as that prevailing in the drinking water itself.

As for any insoluble precipitate, the K<sub>sp</sub> solubility product constant determines the concentration in solution of the ions that dissolve from the salt. For calcium fluoride where  $\text{CaF}_2 \rightarrow \text{Ca}^{2+} + 2\text{F}^-$ ,  $K_{sp} = [\text{Ca}^{2+}][\text{F}^-]^2 = 3.4 \times 10^{-11}$ . This relation was used to calculate F<sup>-</sup> levels for a given Ca<sup>2+</sup> level or Ca<sup>2+</sup> levels for a known F<sup>-</sup> level. Other measurements in the table were from actual blood samples drawn from Hooper Bay, Alaska victims where fluoridated municipal water, for which machinery malfunctioned, poisoned 296 residents. Not mentioned is the increased thirst associated with heavily fluoridated water, a biologic response to this insult that was up to that time unknown.

The solubility of calcium fluoride changes somewhat with temperature and pH. It is slightly more soluble at body temperature of 37°C (about 5%) but less soluble with increasing basicity (very slight for pH 7.4 of blood); we here estimate the solubility with the K<sub>sp</sub> for calcium fluoride at pH 7 at 25°C since these offsetting effects are opposites.

\*\*For unexplained reasons the blood fluoride was not measured in the victim who died, but blood calcium was. Urine fluoride was measured, which for all other subjects was about 50 times the blood level. The blood fluoride level entered with the known calcium level was computed from K<sub>sp</sub> considerations. Dividing the urine fluoride by 50 produces a dose somewhat below this amount.

The K<sub>sp</sub> for magnesium fluoride at room temperature is  $6.4 \times 10^{-9}$ . Intracellular magnesium levels range from 2.5 to 15 millimolar. The plasma concentration is 0.75 to 1.1 millimolar, with one third bound to protein, the rest the diffusible free cation. 1.1 millimolar fluoride would thus precipitate intracellular magnesium and could be the mechanism by which fluoride blocks glycolysis in red blood cells. Intracellular calcium is in the micromolar range. Average blood plasma concentration of calcium is 2.5 millimolar but 1/3 of this is complexed with protein and one tenth is complexed as citrate and phosphate. The remaining fraction is the diffusible ionic physiologically active calcium ion, about 1.3 millimolar.

The calculated doses in Table I are fully consistent with other published data indicating that tissue levels of fluoride in acutely fluoride poisoned people are in the 0.2 - 0.4 mM range<sup>5</sup>. Also the known human lethal dose is 1-5 grams per adult taken at one time<sup>3,5</sup>. Since the average adult contains about 43 liters of body fluid, this corresponds to a fluoride concentration of approximately 0.5 mM in such a case of instant acute poisoning.

Wang, Zhang and Wang also found the heart cell beat rate in cultured cells in well-controlled experiments progressively slows with increasing fluoride levels in a regular, concentration-dependent manner<sup>10</sup>. These data taken together suggest that the mechanism by which fluoride ingestion is acutely lethal is by causing hypocalcemia and blockage of heart contractions. Fluoride levels in blood below 0.1 mM do not lower calcium ion below normal, and no precipitate yet forms in the blood at this or lower doses. But the instant fluoride approaches this amount, calcium ion precipitates, the blood level is lowered and is unable to support heart function.

Fluoride also is an enzyme inhibitor, for all enzymes requiring calcium for function, by binding the ion and is used routinely to block sugar metabolism in red blood cells for clinical laboratory analyses of blood specimens. Fluoride also attaches to calcium anywhere this ion is concentrated throughout the body, including teeth, bones, ligaments, skeletal muscle and brain. But the most crucial function requiring calcium that is fluoride-sensitive appears to be the contraction mechanism in the beating heart.

That extracellular calcium is an obligatory requirement for heart cells to undergo contraction after electrical excitation is well known. Heart cells do not have well-developed sarcoplasmic reticulum to store calcium for this purpose as does all skeletal muscle, which does not exhibit this extreme sensitivity to changes in blood calcium level. The cellular uptake of calcium occurs during the plateau phase of the cardiac action potential and extracellular calcium is necessary for the development of contractile force<sup>11</sup>. The strength of contraction (inotropic state) of the heart depends on calcium, where half maximal contractility occurs at 0.5 mM calcium outside cells<sup>12</sup>.

It is also possible that chronic 'low' level biologic effects of fluoride are also mediated exclusively by binding and sequestration with calcium since calcium-rich regions are localized in many histologic areas. Prior to levels of calcium in the blood being lowered directly by precipitation (below 0.1 mM fluoride), regions in the body enriched in calcium would still precipitate calcium fluoride, as in bone, teeth, ligaments and brain. For example, the mechanism by which fluoride from blood at desired 'low' levels irreversibly accumulates in bone does not involve precipitation of ionized calcium because fluoride is below the  $K_{sp}$  for direct precipitation. Instead an ion exchange mechanism occurs at extremely minute fluoride levels, where the fluoride ion merely by diffusion exchanges with hydroxide on bone hydroxyapatite. An introductory discussion of the pathologic alterations in fluoridated bone was presented by Fagin<sup>24</sup>. The physiologic response to such an insult is to increase levels of hormones such as calcitonin

to mobilize calcium from bone to fight the sequestration and also to increase bone cell reproduction<sup>14</sup>. This maintains normal blood calcium levels, allowing normal heart function but must also compromise, to a finite degree, overall calcium homeostasis.

The current level of fluoride in many municipal water supplies is regulated at 0.25 mg per liter or 0.012 mM (1 ppm). The blood level equilibrates in consumers typically about 1/5 the water level. This is below the solubility for calcium fluoride at normal body pH, temperature and prevailing extracellular fluid calcium levels, and it is easy for many to assume the information in this manuscript is irrelevant. But some cities use up to 1.5 mg/L (0.05-0.075 mM) or the Federal allowed ceiling of 2-4 mg/L (0.1-0.2 mM) and are near or at the maximum level that would just begin slight precipitation of calcium, with redistribution of calcium stores to maintain normal blood calcium, unless the city water happened to have so much calcium in it that it minimizes fluoride assimilation substantially, preventing the fluoride added from entering one's blood at that level.

Fluoridated cities commonly have increased per capita heart attack rates<sup>18</sup>. Our data suggest that this may involve alterations in calcium distribution, as much as high population density increasing stress in fluoridated cities. The Hooper Bay disaster contained its own internal control, since part of the cities' water was on a different fluoridated system that did not malfunction at the time. Obviously the heart attack rate per capita was greater on the fluoridated system's water because of the fluoride, not because lives were more stressful in this section of Hooper Bay. Consumption of artificial fluoridated water has been reported to have various adverse direct effects on heart function<sup>19</sup>.

Please understand that a fluoride ion solution made in soft or distilled pure water has a very high chemical activity, or chemical potential, compared to the activity of the ion at the same concentration when accompanied also by calcium or magnesium ion in solution. Although much less sensitive and exquisite than an actual biological cell membrane, a fluoride specific electrode senses such a difference. In the following graph for example are fluoride electrode measurements of a solution of sodium fluoride fixed at 0.8 mg/L (ppm) (0.042 mM) actual concentration, in pure de-ionized water, and at various calcium levels that mimic a range of water hardness and ionic strength. 30 mM calcium (Figure 1) causes substantial inter-ionic interactions with fluoride that significantly lower diffusion or Brownian motion of the fluoride ion because of the relatively massive divalent positive charge on the compact calcium ion. This phenomenon applies to Group II cations including magnesium ion, prevalent in all foods and natural hard waters.

In contrast, fluoride accompanied in solution with Group I metal cations, such as sodium or potassium, exhibit no decline in activity over a broad range of cation concentration, because these ions are only monovalent in charge (Figure 2.) Notice that a 1 ppm fluoride solution in pure water has as a function of added potassium ion has an activity not significantly decreased until 200 mM, a concentration at which

fluoride activity would be already reduced a massive 50% by calcium ion (Figure 1). Magnesium ion, or in particular calcium and magnesium together, as in hard natural U.S. waters, decreases fluoride mobility efficiently.

The physiologic behavior of a given concentration of fluoride ion is mostly determined by the prevailing water hardness. Soft water states in the U.S., devoid of divalent cations, are higher in fluoride chemical activity, particularly when added from artificial fluorides without calcium. The ratio of calcium ion molarity (around 0.12 mM) to added fluoride molarity (0.05 mM) in soft water states, particularly in the Pacific Northwest, in an artificially fluoridated city, is very low. In hard water states the ratio is typically about 80-100 to one or more, but still insufficient to prevent blood levels from reaching 0.21 ppm<sup>14</sup> (p. 70). Hard water states are thus more protected from fluoride ion than soft water states in the U.S., since assimilation of fluoride is more marked in the latter due to fluoride distribution according to the extent to which a given side of a biologic membrane is more enriched in calcium or magnesium. Extracellular calcium levels are in the several millimolar range, while intracellular calcium is in the micromolar range. Magnesium levels however are millimolar levels both inside and outside cells.

Activity coefficients for the fluoride ion are substantially reduced in the presence of calcium and magnesium divalent cations<sup>26</sup>. This effect may be compared to the well known phenomenon of attraction between fluoride ion and hydrogen atoms in water known as hydrogen bonding which decreases the Brownian motion and diffusion of the ion. These factors determine the overall biologic effect of fluoride ion for living organisms, by affecting assimilation through the gastrointestinal tract, and other events not well understood because membranes exhibit such complex structural and functional features<sup>20</sup>. Fluoride tends to remain concentrated in a solution containing calcium ion, even though far below the level required for binding as calcium fluoride. The higher the calcium concentration of a region, the less fluoride is able to diffuse away from it. This electrical attractive force is also responsible for the fact that fluoride, even at levels far below the known solubility constant  $K_{sp}$  for forming calcium fluoride precipitate, is trapped into bone, with an ion exchange mechanism due to directed collisions.

The actual, LIVING importance of the chemical differences between sodium fluoride or fluosilicic acid versus calcium fluoride has been amply demonstrated in biology: the precise dose at which lethal fluoride poisoning occurs in tested animals, the LD<sub>50</sub>, for calcium fluoride is a safe 3,750 mg/kg single dose, whereas lethality for sodium fluoride or fluosilicic acid, as expected, compares to that for arsenic at 125 mg/kg single dose (Merck Index, 7<sup>th</sup> edition). Hooper Bay is not the only soft water city treated with calcium-absent fluoride to experience disaster. It is well publicized<sup>21</sup> that horses were slaughtered in Pagosa Springs, Colorado after only 9 years drinking artificial silicofluoridated soft water, deficient in calcium and magnesium from nearby snowmelt. These animals drink their body weight in water every few days and all suffered severe skin reactions, crumbled hooves and browned, pitted, cracked, destroyed



teeth, muscle weakness, and were eventually killed by skeletal fluorosis with severe associated tumors. The owners were newcomers who assumed city water was acceptable for hoofed animals. If natural calcium fluoride had been the agent employed in these cities, the quick lethal reactions could not have existed. For example, fluoride water levels even 10 fold higher than this from natural sources exist in areas in India. Natural fluorides are always accompanied with other calcium and magnesium salts from the natural erosion from which the fluoride also erodes. This hardness prevents acute lethality, and instead these people, with lifelong drinking, exhibit bone deformities.

No bone cancer is known to be induced by lifetime drinking of natural calcium fluoride, whereas substantial proof exists that lethal untreatable bone cancer is produced from artificial fluoride when exposures are continued for a sufficient time<sup>14,22</sup>. For the cancer-resistant and fluoride uptake-resistant rat, this usually requires about ½ of the normal lifespan of the animal. Shorter duration exposure does not significantly increase osteosarcoma, which led some to suggest that data are ‘conflicting’, but all prospective experimental data with proper controls are consistent when times of exposure are considered. Bessin at Harvard published that kids raised on fluoridated U.S. water are experiencing 5 fold increases in lethal bone cancer<sup>23</sup>.

Artificial fluoride, but not natural calcium fluoride, during water district overfeeds have severely poisoned and killed in the U.S. This is because artificial fluorides, unlike natural calcium fluoride, are all fully soluble in water to extremely high concentrations without precipitation. The Hooper Bay artificially fluoridated water caused pump corrosion and an overfeed. Natural calcium fluoride is unable to corrode metals as artificial fluorides do in neutral or acidic waters, which base-hydrolyze to form small amounts of hydrofluoric acid HF, the most corrosive substance in the universe, where  $F^- + H_2O$  produces  $HF + OH^-$ . Natural calcium fluoride also does not require neutralization with sodium hydroxide prior to injection into water, which now is a common practice for U.S. water districts.

In conclusion, fluoride ion from artificial fluorides are not biologically or even physico-chemically the same as fluoride ion from natural calcium fluoride, for otherwise identical concentrations of ionized fluoride ion. Inexperienced or amateur chemists often feel this way, but this provides a false defense for CDC to continue artificial fluoridation in states not having very hard water.

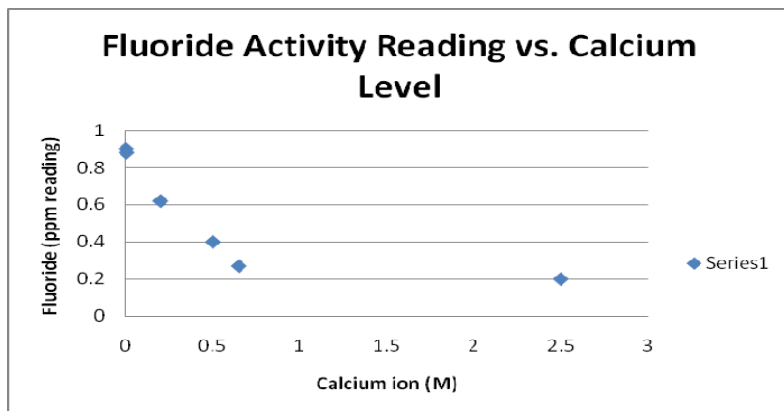
The Osmundsen data indicate that ranking states according to % of the population receiving fluoridated water correlates with increased incidence of per capita heart attack, mental retardation, cancer and other effects (personal communication). When these data are plotted as a function of water hardness for the state (Figure 4) the correlation is even more striking, as toxicologists have long expected.

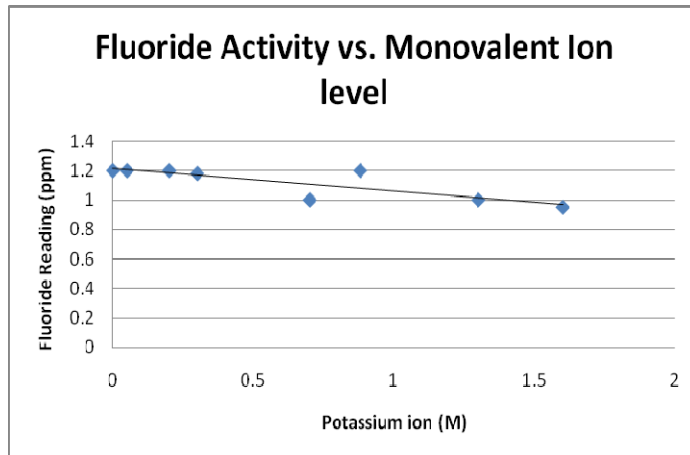
Incorporation of fluoride ion into blood, bone and other tissues is variable<sup>13</sup> due in large part to wide ranges in water hardness. In any event, the National Research Council Report of Fluoride in Drinking Water 2006 clearly proved that 1 ppm fluoridated water accumulates to about 5,000 ppm fluoride in

bones lifetime and far higher levels are expected for type II diabetics, reaching levels associated with severe bone pain requiring hospitalization<sup>14</sup> (p. 35, 179) and of course with weakened bones that resist healing after fracture. The U.S. currently has a well recognized epidemic of hip fractures in the elderly and this author holds water fluoridation as the most significant cause.

Water with 1 ppm fluoride causes uptake averaging 0.03 mg/kg body weight fluoride ingested daily, so after 60 years, half enters the bones to 4,000 ppm, permanently stored in the bone as a non-mobilized fraction. This causes bone cells to undergo cell division, since a crucial function of bone is to provide mobilize-able calcium ion into the blood to maintain the normal heart beat. Indeed, deceased bone from such fluoridated regions measure fluoride in this expected, calculated range<sup>13</sup>.

The graph below demonstrates how the fluoride ion level is lowered as the calcium concentration is increased (Figure 1). The initial level of fluoride in the pre-made solution was 0.90 ppm at room temperature. Addition of calcium from 0.1 to 2.5 molar causes progressive decreases in the free ion level due to precipitation of calcium fluoride particles that the electrode cannot detect. The calcium level calculated to first begin fluoride precipitation at 0.90 ppm fluoride is 0.03M, from solubility product constant published values of  $0.34-1.7 \times 10^{-10}$ <sup>17</sup>. The accompanying graph indicates that only subtle effects on fluoride ion mobility occur over a wide range of concentrations of added monovalent ion from potassium nitrate (Figure 2).



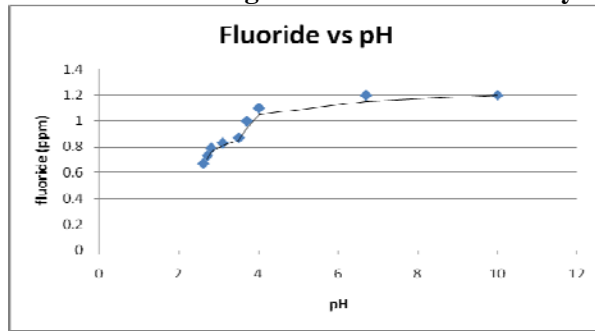


**Figures 1 and 2.** A 0.9 ppm fluoride solution in distilled water was measured for fluoride level with a LaMotte fluoride specific electrode calibrated with 1.00 ppm sodium fluoride in distilled de-ionized water at room temperature. Calcium ion was adjusted over a wide range by addition of aliquots of calcium biphosphate. Fluoride readings progressively decrease with increasing calcium concentration over the range 20 mM to 3 M. A 1.2 ppm sodium fluoride solution in distilled water at room temperature was measured for fluoride readings as a function of added potassium nitrate over a broad range. Readings were unaffected until above 1 M potassium.

The fluoride level that would precipitate calcium from Southern California water (where calcium ion is about 2 mM) would be 0.14 mM fluoride. Before reaching this fluoride level, approaching the Federal ceiling, in water it would precipitate calcium from our drinking water first. To maintain a higher level of fluoride than 0.14 mM would be expensive, requiring addition of enough to precipitate the calcium in the water first. Fortunately this would be unlikely even in accidental overfeed.

Figure 3 indicates the effects of pH on the concentration of the free fluoride ion. Notice that the ion is calculated to exist substantially as the intact hydrofluoric acid molecule at approximately pH 3.5, indeed where fluoride free ion concentration readings begin to dramatically decline. At gastric levels of acidity in a 1 ppm fluoridated city notice that the HF level in the stomach would be expected to be 50% of the total fluoride level, each at approximately 0.05 ppm. In the Hooper Bay disaster the presence of this % as HF could fully explain the severe gastric pain experienced in the hundreds of victims. Substernal pain that compares to this gastric pain can occur from angina pectoris of the heart associated with chronic ischemia, but the direct effect of the HF acid itself on the stomach mucosa is the most likely mechanism by which gastric pain occurred on such a mass scale in the entire affected population at Hooper Bay.

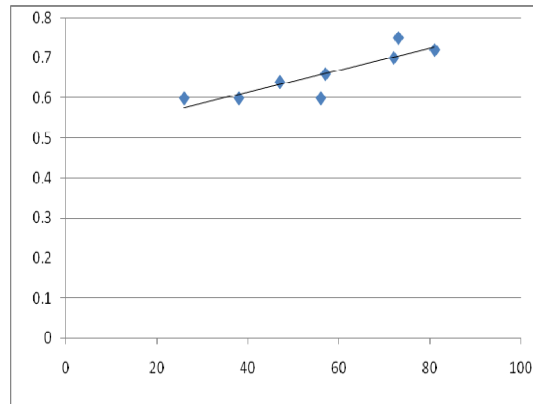
**Figure 3 Fluoride Readings as a Function of Acidity**



All readings were from a LaMotte fluoride specific electrode (calibrated with a 1.00 ppm sodium fluoride standard solution in distilled deionized water at room temperature). Readings for the 1.2 ppm true concentration solution in distilled water progressively decrease as pH decreases. Acidity was adjusted with dilute acetic acid. At stomach acid pH readings the fluoride is about 50% protonated, as hydrofluoric acid HF, and 50% free fluoride.

Figure 4 presents data selected from the study of Osmundson (personal communication) for U.S. states known to have low water hardness (below 100 ppm calcium), to help eliminate the protective effect of calcium in blocking assimilation of fluoride. Notice the progressive increase in per capita heart attack incidence in those states with progressively increased percentages of populations using artificially fluoridated water.

**Figure 4** **Heart Attack Incidence in U.S. States**  
**Correlated with % Fluoridation of Population over Several Years**



From U.S. data sources please notice the correlation between heart attack per capita rate (per 100,000) with % of state population using fluoridated water for those U.S. states having soft drinking water with relatively low calcium levels (less than 100 ppm calcium).

Although all states have not been fluoridated for the same number of years the correlation is highly significant between heart attack per capita incidence and the percent of the population receiving fluoridated water. Soft water states were used to eliminate the known effect of calcium in minimizing assimilation of water fluoride into blood. All data were from U.S. government websites.

Fluoride incorporates readily into intracellular locations, which means the data conducted in vitro that demonstrating fluoride inhibits at least 100 examined enzymes, at concentrations well below 1 ppm, is highly significant. Many of these enzymes are involved in DNA repair and prevention of cancer from any cause, including enzyme repair systems inhibited as much as 50% at only 1 ppm artificial fluoride<sup>15,16</sup>. The fact that these DNA repair enzymes are dramatically inhibited at blood levels found in 1 ppm fluoridated cities (0.21 ppm blood fluoride in 1 ppm fluoridated cities<sup>14</sup>) is fully consistent with the common findings of chromosomal aberrations and genetic damage that is so commonly induced by fluoride in vast numbers of published studies<sup>14</sup>.

Injecting artificial fluorides into public water is paid for by taxpayer adults who themselves reap no measurable benefit. It requires resources, time, chemicals and machinery to continue to add it to drinking water. It is putting the water district in charge of drugging the public and for something as innocuous as a cavity rather than for serious effects such as infectious illness for which chlorination has been properly chosen, a much less electronegative halogen.

It is not in keeping with a free society or with proper health care practice to impose these risks associated with fluoridating the blood of people, livestock, and pets, and also all agricultural products, lawns and gardens, compared to the less significant problem of perhaps having tooth decay. Tooth decay should be minimized more efficiently and safely by simply brushing more vigorously and regularly.

Maintenance of normal calcium exchange mechanism in the bloodstream is far more important than concern for cavities. This is demonstrated not only with the Hooper Bay mass poisoning event but also with several children that have been killed with heart attacks in dental chairs when fluoride gel was swallowed. Teeth are replaceable but lives are not. In keeping with the Hippocratic oath, no physician medicates anyone without their permission, and all patients must remain free to withdraw from drug or other treatment programs at any time. Forced fluoridation in public water supplies ironically constitutes a reversal of these Public Health Service policies. The easy way - fluoridate through the bloodstream by drinking - is unnecessary (since topical application is possible), potentially hazardous, and exerts over lifetime consumption significant alterations in bodily processes and structure. Proper dental hygiene is much safer to achieve the desired result. The notion recently publicized that 'antifluoridationists' are similar to earlier critics of smallpox vaccination is inconsistent with the facts that smallpox is lethal and only prevented with blood vaccination. Cavities are not lethal and can be prevented with proper hygiene and if necessary the bacteria that cause caries in the first place can be quickly destroyed with simple methods such as hydrogen peroxide washings, etc. without loss of life.

Hard waters in the U.S. Southwest typically contain fluoride from natural calcium fluoride. In Texas the structural effects on teeth prompted U.S. dental schools to support raising fluoride levels artificially in other U.S. cities. Unfortunately, fluoride assimilated into the blood is trapped in dentine inside teeth at ten times higher levels than in tooth enamel, making teeth interiors crumbly (National Institutes of Health). Texas Dentist Dr. Heard first promoted natural fluoride consumption since enamel presented temporarily with fewer cavities, but he later found teeth after long-term consumption to be more crumbly and then fought against artificial fluoride in water. We now know long-term consumption of either natural or unnatural fluoride leads to thousands of times higher levels in bones than in water, that is irreversible and pathologic, weakening bones<sup>14</sup>.

From the National Academy of Sciences report note that 1) fluoride accumulation is linear, rather than saturable which proves it is a pathology, not normal physiology, where mineral nutrients always exhibit curved, saturable dose dependence and are fully reversible; 2) long-term consumption of fluoridated water at only 1 ppm incorporates fluoride to 4,000 mg/kg into bone after about 20 years typically that is often associated with severe bone pain requiring hospitalization, and 3) subjects moving to non-fluoridated water source cities maintain their accumulated bone fluoride level for 25 years because this unnatural, pathologic accumulation is irreversible, as mentioned in Chapter 11 of that reference<sup>14</sup>.

Artificial fluoridated water fares even worse than natural fluoride. The experience at Grand Rapids, Michigan indicated artificial fluoride injected into water caused no greater decrease in cavities than that also noted over an equal time period in the non-fluoridated control city of Muskegon. In Newburgh, N.Y. artificial fluoridation delayed teeth eruption and after teething developed, children had a cavity rate

comparable or somewhat worse than the control non-fluoridated city of Kingston. Since assimilation of artificial fluoride occurs at a higher fluoride to calcium ratio than for natural fluoride, assimilation of the ion into skeleton, brain and teeth dentin is higher than that for natural fluoride at the same water concentration.

In 2000, there were 2.6 million medically treated non-fatal fall related injuries<sup>25</sup>. Direct medical costs totaled 19 billion dollars for non-fatal injuries. Of the non-fatal injury costs, 63% (12 billion dollars) were for hospitalizations, 21% (4 billion dollars) were for emergency department visits, and 16% (3 billion dollars) were for treatment in outpatient settings. Medical expenditures for women, who comprised 58% of the older adult population, were 2-3 times higher than for men for all medical treatment settings. Fractures accounted for just 35% of non-fatal injuries but 61% of costs. Fall related injuries among older adults, especially among older women, are associated with substantial economic costs. In cities fluoridating at higher levels, 12,000 ppm bone fluoride is found post mortem. This is accompanied with fluoride accumulation into soft tissues, after bone sites are filled. The observed increased fractures are consistent with the fact that 90% of 'fluoridated' cities use artificial fluorides instead of natural calcium fluoride. 60% of Americans now live with fluosilicic acid in municipal water.

Long-term drinking causes fluoride to also accumulate into hydroxyapatite of the brain's pineal gland and decreases IQ in children<sup>14</sup>. Mullenix first reported in extensive studies with large populations of animals that fluoride blood levels comparable to that in human blood in a 1ppm fluoridated city cause accumulation of fluoride into various regions of the brain that correlated with alterations in behavior observed with nonbiased computer controlled cameras. Ranking the 50 U.S. states according to % of the population receiving treated water correlates strongly not only with modest increases in per capita heart attack rate but also with mental retardation and infant mortality, while cavities were not decreased ( Osmundson study, personal communication).

Ironically, in the original studies with 1 ppm artificial fluoride in water in Newburgh, N.Y., delayed teeth eruption occurred compared to the control city of Kingston. After teeth are fully erupted, cavity rates are not significantly different between the two cities. Since missing teeth were counted as absence of cavities, the conclusion that fluoridated water decreases cavities was premature. In fact, worldwide data indicate that cavity reduction occurred over recent history in non-fluoridated counties that compares with that for fluoridated.

Water districts most commonly now inject artificial fluosilicic acid into water to increase fluoride. Clinical trials have never been done, so the U.S. Food and Drug Administration has never approved artificial fluoride in public water supplies and label it as a drug, not a mineral nutrient and consider artificial fluoridation to be an uncontrolled use of a drug. In Pagosa Springs, Colorado, horses died from

skeletal fluorosis after only 9 years drinking 1 ppm fluosilicic water<sup>21</sup>. Salmon spawning is obliterated by artificial fluoride in water at only 0.3 ppm, but returns after discontinuing fluoride dumping<sup>23</sup>.

Adding a high toxicity halogen, fluoride, to alter human tissues, is a radical, pre-emptive action, based on the assumption that future bacterial caries will occur that will have required such action that were outside one's own ability to control. It considers irrelevant the common, complete absence of cavities in hygienic minded persons, and that some households have zero access to another bathing and/or drinking water source. Based on information in this manuscript, the author is concerned that the fluoridated city of Seattle, Washington, where North West Pacific water is very low in calcium and magnesium ions, has the highest per capita heart attack rate in the country. It had been formerly believed that this was due to a deficiency in calcium and magnesium in the diet, but the possibility is much more likely to be due to the use of artificial soluble fluoride compounds injected into the soft water, where insufficient calcium is present to buffer the effects of added fluoride. Also, a first suspected, not last suspected, phenomenon involved in the presence of heart attacks in children under 13<sup>24</sup> would be fluoride use, because it is so easy to become overexposed to it. Many children like to swallow mint flavored toothpaste and in fluoridated cities this is contra-indicated. Finally, it is likely that adverse effects of fluoride long-term exposure is determined by the number of total fluoride binding sites that are available in the bony skeleton. Those having the least bone mass for a given body weight would be the earliest to exceed the total body burden of consumed fluoride during lifelong drinking.

It is unfortunate that the former Public Health Service and now the U.S. Centers for Disease Control fail to measure existing calcium and magnesium levels in water prior to recommending mineral additives; forcing such action to alter the natural waters of the United States is outside the mission and scope of the CDC and in fact is in contradiction to the Federal Water Pollution Control Act, section 101a, which explicitly mandates the maintenance and protection of the natural chemistry of all U.S. waterways. Fluosilicic acid  $H_2SiF_6$  is not at any strength found in natural water supplies. Both the FDA and National Research Council correctly maintain that fluoride has no nutritive value of any kind, and no studies are available to prove that silicic acid or its derivatives are of zero adverse health effect lifetime regardless of prevailing water acidity or hardness.

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