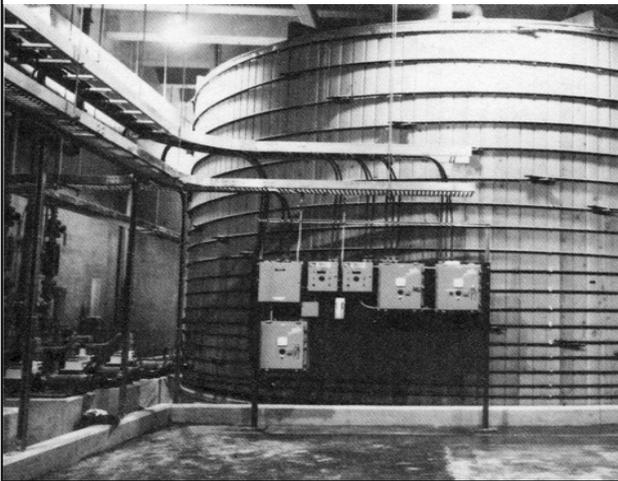




Fluorosilicate Toxicity

Hydrofluorosilicic Acid Tank (Bears paw, Alberta, Canada)



Any implication that the water treatment facility is merely adjusting the amount of fluoride that is found naturally in the water supply is misleading.

Fluoride concentrations that are found naturally in water usually contain a proportionately high level of calcium, which is not represented in hydrofluorosilicic acid used in the fluoridation process. **The presence of calcium is able to alter fluoride absorption and protect against fluoride toxicity.**

Hydrofluorosilicic acid is neither “natural” nor passive. It is anthropogenic, meaning made by man (in the smoke stacks of the phosphate fertilizer industry), and extremely toxic.

Inhalation: Corrosive! Product may cause severe irritation of the nose, throat and respiratory tract. Repeated and/or prolonged exposures may cause productive cough, running nose, bronchopneumonia, pulmonary oedema and reduction of pulmonary function. Mist or spray may be irritating to the nose, throat and respiratory tract.

Skin Contact: Corrosive! May cause pain and deep and severe burns to the skin. Prolonged and repeated exposure to dilute solutions often causes irritation, redness, pain and drying and cracking of the skin. Toxic effects may be delayed.

Skin Absorption: Skin absorption is a secondary concern to the continual destruction of tissue while the product is in contact with the skin.

Eye Contact: Extremely Corrosive! This product causes corneal scarring and clouding. Glaucoma, cataracts and permanent blindness may occur.

Ingestion: Corrosive! This product, causes severe burning and pain in the mouth, throat and abdomen. Vomiting, diarrhea and perforation of the esophagus and stomach lining may occur.

Other Health Effects: Corrosive effects on the skin and eyes may be delayed, and damage may occur without the sensation or onset of pain. Strict adherence to first aid measures following any exposure is essential.

May cause pulmonary oedema, fluorosis, exostoses, hypocalcemia, shock, central nervous system (CNS) depression, coma and death. CNS depression is characterized by headache, dizziness, drowsiness, nausea, vomiting and incoordination. Severe overexposures may lead to coma and possible death due to respiratory failure. Pulmonary oedema is the build-up of fluid in the lungs that might be fatal. Symptoms of pulmonary oedema, such as shortness of breath, may not appear until several hours after exposure and are aggravated by physical exertion.

Fluoride is a bone seeker, and excessive amounts will produce weakening and degeneration of the bone structure. Chronic exposure may cause excess accumulation of fluorine (fluorosis) in the teeth and bones. Severe fluorosis in children weakens tooth enamel resulting in surface pitting. After prolonged high intake in adults bony changes occur characterized by hardening or abnormal density of bone (osteosclerosis), benign bony growths projecting outward from the surface of the bone (exostoses) and calcification of ligaments, tendons, and muscle attachments to bone. Ingestion and skin contact may cause an abnormal reduction of blood calcium (hypocalcemia) and kidney damage since fluorides precipitate calcium stored in the body. There may also be heart, asthma, nerve, intestinal and rheumatism problems.“

Brenntag Canada Inc. Current Hydrofluorosilicic Acid (HFSA) Safety Precautions

Burns caused by hydrofluoric acid can be life-threatening. Of special significance is the often underestimated local and sometimes delayed deep action of the highly diffusible free fluoride ions and the accompanying systemic toxicity. The specific antidote calcium gluconate can be topically applied, injected into tissue or infused intra-arterially. Because of the extreme danger of systemic toxicity even after seemingly trivial injuries, monitoring in the intensive care station, especially by measuring the calcium concentration in blood and electrocardiography, and therapy is recommended.

Richter H, Hollenberg S, Sachs HJ, Oeltjenbruns J, Weimann J. 2005 Hydrofluoric Acid Burns: A rare chemical emergency situation. *Anaesthesist* Feb;54(2):123-6. [in German].

Accidental hydrofluoric acid (HF) splashes often occur in industrial settings. HF easily penetrates into tissues by initial acid action allowing fluoride ions to penetrate deeply, chelating calcium and magnesium. Resultant hypocalcemia and hypomagnesemia can be fatal.

Soderberg K, Kuusinen P, Mathieu L, Hall AH. 2004 An Improved Method for Mergent Decontamination of Ocular and Dermal Hydrofluoric Acid Splashes. *Vet Hum Toxicol* Aug;46(4):216-8.

A 37-year-old male laboratory technician was performing acid digestion of sedimentary rock samples with 70% w/w concentrated hydrofluoric acid ...he knocked over a small quantity (100–230 mL of the hydrofluoric acid onto his lap, splashing both thighs. He sustained burns to 9% of his body surface area, despite washing his legs with water from a makeshift plumbing arrangement that supplied water at low pressure...Following flushing, because he was still in severe pain and shock, he immersed himself in a chlorinated swimming pool at the rear of the workplace, where he remained for approximately 35–40 min before ambulance help arrived. At that time he was hypothermic and hypocalcaemic on admission to an intensive care unit at a nearby hospital, and soon became unconscious. His condition continued to deteriorate despite subcutaneous injections of calcium gluconate and administration of intravenous calcium and magnesium. His right leg was amputated seven days after the incident. He subsequently died from multi-organ failure 15 days after the spill.

Muriale L, Lee E, Genovesef J, Trend S. 1996 Fatality due to acute fluoride poisoning following dermal contact with hydrofluoric acid in a palynology laboratory. *Annals of Occupational Hygiene* 1996; 40(6):705-10.

A quick look at the **Periodic Table** will show you that the halogen family holds a distinct position. It is a group that is chemically very reactive (group 7). Fluorine is the smallest of the halogens, the most reactive of all of the halogen family and the most reactive non-metal element known to man. It has a propensity to combine with anything in its path. Fluoride combines with essential nutrients like calcium and magnesium, interfering with tissue uptake. Fluoride can help facilitate the uptake of aluminum and lead into tissues where these metals would not otherwise go. [Gilman 1987, Wittinghofer 1997, Mahaffey 1976, Allain 1996, Varner 1998, Li 2003, Sternweiss 1982, NRC 2006]

It interferes with hydrogen bonding [Emsley 1981, NRC 2006] and interferes with more than 100 enzymes. [See: partial bibliography below or visit WHO website]

Virtually every vital function of the body is dependent on enzymes. The same enzyme in different tissues/organs exhibit different sensitivities to fluoride; e.g. **Esterase** in liver – fluoride inhibits; **Esterase** in pancreas and bowels – fluoride does not inhibit.

Some enzymes are inhibited, others are activated by fluoride. For example, fluoride inhibits glycolysis (breakdown of glyucose into energy for cells) by inhibiting the enzyme enolase. Fluoride inhibits energy metabolism through the tricarboxylic acid cycle by blocking the entry of pyruvate and fatty acids and by inhibiting succinic dehydrogenase.

Voluntary and involuntary muscle action is stimulated by **acetylcholine** (ACh) which is cleaved by the enzyme **acetylcholinesterase** (AChE) to end the stimulation. Without AChE, muscle excitation would persist as spasm with potentially lethal effect, as caused by a nerve gas. Acetylcholine modulated by acetylcholinesterase also induces saliva flow. Intense salivation is a symptom of fluoride poisoning. Fluoride interferes with acetylcholinesterase, but silicofluorides are a much more potent inhibitor of this essential enzyme. [Westendorf 1975]

Alkaline phosphatase, an enzyme involved in growth of bones and liver function, on the other hand is often elevated in chronic fluoride poisoning. [Merz 1970, Srikantia 1965, Waldbott 1962]

Hypothesized mechanisms of action for fluoride include the inhibition of enzymes vital for the survival of bacteria in mouth which are involved in acid dissolution. This begs the question; if fluoride destroys the enzymes in the bacteria, what does it do to the enzymes in other parts of our body? It has been demonstrated that fluoride destroys enzymes involved in the construction of teeth and bones at very low doses in rats at 0.1ppm. [Takei et al. 2007]

Table 7-1 and 7-2 From: Waldbott G. 1968 Fluoridation – The Great Dilemma p 88

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CHAPTER 7

Table 7-1

Comparative Toxicity of Inorganic Fluorides⁶

Extremely Toxic

Hydrogen fluoride (anhydrous)	HF
Silicon tetrafluoride	SiF ₄
Hydrofluoric acid (aqueous)	HF
Hydrofluorosilicic acid	H ₂ SiF ₆

Very Toxic

Easily soluble fluorides and fluorosilicates

Sodium fluoride	NaF
Potassium fluoride	KF
Ammonium fluoride	NH ₄ F
Sodium fluorosilicate	Na ₂ SiF ₆
Potassium fluorosilicate	K ₂ SiF ₆
Ammonium fluorosilicate	(NH ₄) ₂ SiF ₆

Moderately Toxic

Poorly soluble (almost insoluble) fluorides

Cryolite	Na ₃ AlF ₆
Calcium fluoride	CaF ₂

Table 7-2

Lethal Dose of Fluorides in Adult Guinea Pigs⁷

<i>Compound</i>	<u>Oral (mg/kg)</u>	<u>Subcutaneous (mg/kg)</u>
NaF	250	400
→ CaF ₂	>5,000	>5,000
AlF ₃	600	3,000
HF (aqueous)	80	100
→ H ₂ SiF ₆	200	250
Na ₂ SiF ₆	250	500
Al ₂ (SiF ₆) ₃	5,000	4,000

Industry Fluoride

vs.

Natural Fluoride

Hydrofluorosilicic Acid (H₂SiF₆)

is 20 times more toxic than

Calcium Fluoride (CaF₂)

Table 7-2 shows the acute toxicity of the most important fluoride salts to guinea pigs, expressed in milligrams per kilogram of body weight. It also demonstrates the difference in the lethal dose following oral and subcutaneous administration.

Table 7-2 from: Simonin P, Pierron A. 1937 Toxicite brute des derives fluores. C.R. Seances Soc. Biol. Fil. 124: 133-134.