

# **Flouride Toxicology and Health Information**

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### **Abstract**

A review of toxicology and health information of fluoride is given. Historically, industrial and natural processes have released large amounts of fluoride into our environment and as a result, routes of exposure to fluoride are numerous. Fluoride is present in almost all food and water to some degree. Lack of fluoride in the diet can lead to dental caries (tooth decay). The acute toxic fluoride dose is reported as 0.1 to 8 mg F/kg body weight (*Akiniwa, 1997*). Symptoms of acute fluoride poisoning include vomiting, abdominal pains, diarrhea, convulsions, nausea, and fever. Chronic overexposure of fluoride can lead to severe dental fluorosis (a.k.a. "mottled teeth") and bone fractures. Though some fluoride gasses are extremely poisonous, chronic exposures to fluoride in the air are typically considered negligible. Fluoride in drinking water has an maximum contaminant level (MCL) of 4 ppm. US Public health service recommends the "optimal" of fluoride in the drinking water range for preventing tooth decay and dental fluorosis as 0.7 to 1.2 ppm.

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## 1. Introduction

Fluorine is estimated to be the 13th most abundant element in our Earth's crust (*Cholak, 1959*). As the most electronegative element, Fluorine is almost exclusively found combined with other elements. A compound which includes fluorine is referred to as a fluoride (*Waldbott et al., 1978*). Fluorides occur as organic and inorganic compounds both naturally and as industrial products and by-products. Commercial uses of fluorides are wide ranging; some example are rocket fuel, brewery sanitation, toothpaste and dielectrics. Fluoride is also a common additive to drinking water. As a result of its abundance in our environment and industry, methods of exposure are numerous. Fluoride induces a variety of responses in humans, both beneficial and harmful, and as a result is carefully regulated. This paper will discuss various routes of exposure, human responses to exposure and regulations of fluoride. This paper will not attempt to take a side in the ongoing fluoridation debate.

## 2. Exposure

A common route of exposure to fluoride is through drinking water but this is by no means the primary route of exposure (*Waldbott et al., 1978*). Levels of fluorine are typically measured through fluoride concentration in urine. This section discusses the main routes of exposure including air, water, food and soil. The National Research Council estimates the daily intake of fluoride at less than 1 mg (*McClure, 1943*).

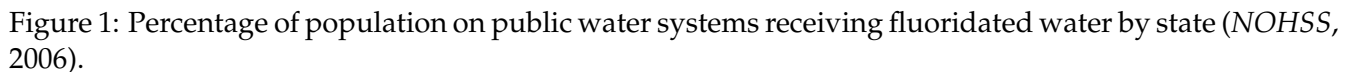
### 2.1. Air

The primary source of fluorides in the air is industrial processes such as "grinding of fluoride-containing minerals, smelting, kiln firing of brick and other clay products, combustion of coal and in the aluminum and steel industries" (*NHDES, 2008*). Typically, concentrations in air average less than 0.0625 parts per billion (ppb) fluoride which is generally considered negligible (*Yunghans and McMullen, 1970*). Though in areas near where any of the above industrial processes occur, fluoride intake from air may exceed intake from water (*Balazova et al., 1969*). Typical exposures in the air would be classified as chronic. Acute exposures to fluoride gas is possible though very rare.

### 2.2. Water

The average concentration of fluoride in surface water varies depending on the amount of naturally occurring fluoride in the local rocks. Typical ranges are 0 to 0.2 parts per million (ppm) though rivers containing industrial contamination have recorded fluoride levels as high as 46 ppm (*Johnson, 1961*). The average concentration of fluoride in groundwater is 0.4 ppm (*NHDES, 2008*). The average concentration of fluoride in ocean water is 1.3 ppm at depths up to 2500 meters (*Wilson, 1975*). Oceans serve as a source of fluoride to the atmosphere through evaporation. Rainwater from this evaporation can contain up to 0.025 ppm fluoride (*Kester, 1971*).

Fluoride is a common additive to drinking water. This practice is known as fluoridation. After fluoridation, the range of fluoride concentrations found in drinking water is 0.7 to 1.2 ppm (*NHDES, 2008*). As of 2002 67% of the US population on public water supplies received fluoridated water which accounts for 60.5% for the total population (*NOHSS, 2006*). Figure 1 shows the distribution of fluoridation by state. *Waldbott et al. (1978)* estimates the fluorine intake has increased by a factor of 2 to 10 because of food and drinks prepared with fluoridated water. Fluoride from the water is absorbed at up to 90% efficiency where most is incorporated onto the bones and teeth (*NHDES, 2008*). Exposures from water would be classified as chronic.



Fluoride enters the soil by precipitation, eroding rocks, fertilizers, and waste runoff (Waldbott *et al.*, 1978). Clay soils tend to retain more fluoride than sandy soil. Typical soils range from 100 to 300 ppm (Waldbott *et al.*, 1978). Typical exposures from the soil would be considered chronic but are generally considered negligible compared to exposures in food and water.

Nearly all food contains some amount of fluoride (NHDES, 2008). Fluoride intake from food depends on the type of plants and animals we consume. Seafood is known to be a rich source of fluoride, 1.37 to 5.21 ppm in the flesh and 5 to 20 times more in the bones and skin (Oelschlager, 1970). The concentration of fluoride in toothpaste is 900 to 1,100 ppm (NHDES, 2008). There exists a risk of acute fluoride poisoning if toothpaste is ingested. According to IOM (1997), “intake from fluoridated dental products adds considerable fluoride often approaching or exceeding intake from the diet, particularly in young children who have poor control of the swallowing reflex.” Section 3 will address this further. Intakes from food are less soluble than intakes in water and only a small fraction is absorbed into the blood (Waldbott *et al.*, 1978). Normal exposures from food would be considered chronic.

This section will discuss the beneficial effects of fluoride intake and responses to acute and chronic doses of fluoride.

Concentrations of fluoride in the drinking water in concentrations in the range of 0.7 to 1.2 ppm are known inhibit the the formation of dental caries (tooth decay) (NHDES, 2008). according to NHDES (2008) usual range of fluoride in the drinking water “provides optimal cavity prevention while keeping

the occurrence of dental fluorosis at about 10 percent of the population, with the degree of fluorosis mostly in the 'very mild' category" (Table 1). The proposed mechanisms for this includes the inhibition of the lactic acid producing bacterium *Lactobacillus Acidophilus* and enhancing calcification of tooth enamel. For these reasons fluoridation of the drinking water supply is a common practice in the US.

Table 1: Dental Fluorosis Classification (*Dean, 1942*)

| Classification | Description of Enamel   |
|----------------|---|
| Normal         | Smooth, glossy, pale creamy-white translucent surface                                   |
| Questionable   | A few white flecks or white spots   |
| Very Mild      | Small opaque, paper-white areas covering less than 25% of the tooth surface             |
| Mild           | Opaque white areas covering less than 50% of the tooth surface                          |
| Moderate       | All tooth surfaces affected; marked wear on biting surfaces; brown stain may be present |
| Severe         | All tooth surfaces affected; discrete or confluent pitting; brown stain present         |

### 3.2. Acute Response

The acute toxic fluoride dose is reported as 0.1 to 8 mg F/kg body weight (*Akiniwa, 1997*). Acute fluoride responses are typically different whether the fluoride is organic or inorganic (*Waldbott et al., 1978*). The most toxic organic fluorides are salts of fluoroacetic acid. In a 22 pound dog, 1 mg of such a substance produced no effect for 8 to 10 hours followed by "fatal convulsions" caused by blockage of the citric acid cycle (*DeEds, 1933*). When heated above 300 °C (572 °F) Teflon breaks down releasing the highly toxic gas perfluoroisobutene which has been the cause of human deaths (*Waldbott et al., 1978*). Of inorganic fluorides, fluoride gasses are the most toxic. The two most toxic fluoride gasses are hydrogen fluoride (HF) and silicon tetrafluoride (SiF<sub>4</sub>) (*Roholm, 1937*). Fluoride gas typically dissolves quickly in water and therefore acute poisoning is rare. Table 2 shows lethal doses in adult guinea pigs of various inorganic fluorides. The most toxic aqueous inorganic fluoride is hydrofluoric acid (HF).

Toothpaste as a source of fluoride poses a threat of acute toxicity in children (*Shulman and Wells, 1997*). A typical tube of toothpaste contains 900 to 1100 ppm F. According to *Shulman and Wells (1997)* a 10 kg child who ingests 50 g of 1000 ppm fluoride toothpaste "will have ingested a probably toxic dose."

There is limited evidence that fluoride exposures in the air have been the cause of symptoms in incidents such as Donora, Pennsylvania, 1948 (*Waldbott et al., 1978*). This evidence is controversial.

Symptoms of fluoride poisoning include vomiting, abdominal pains, diarrhea, convulsions, nausea, and fever (*Waldbott et al., 1978*).

Table 2: Lethal dose of inorganic fluorides in adult guinea pigs (*Simonin and Pierron, 1937*).

| Compound   | Oral (mg/kg) | Subcutaneous (mg/kg) |
|--|--------------|----------------------|
| NaF  | 250          | 400                  |
| CaF <sub>2</sub>                                 | >5000        | >5000                |
| AlF <sub>3</sub>                                 | 600          | 3000                 |
| HF   | 80           | 100                  |
| H <sub>2</sub> SiF <sub>6</sub>                  | 200          | 250                  |
| Na <sub>2</sub> SiF <sub>6</sub>                 | 250          | 500                  |
| Al <sub>2</sub> (SiF <sub>6</sub> ) <sub>3</sub> | 5000         | 4000                 |

### 3.3. Chronic Response

The most common chronic response to fluoride poisoning is dental fluorosis or “mottled teeth” (Figure 2). Dental fluorosis is caused by chronic ingestion of high levels of fluoride from the ages of 6 months to 5 years (Waldbott *et al.*, 1978). The minimum dose suggested to cause dental fluorosis is 2 ppm fluoride in drinking water and children exposed to 4 ppm or more are at risk of developing severe dental fluorosis *BEST* (2006). Dental fluorosis is not positively correlated with tooth decay, in fact just the opposite is true, individuals with dental fluorosis are less likely to develop dental caries (Waldbott *et al.*, 1978). Figure 3 shows the increase in prevalence rate of severe and the decrease in normal dental fluorosis as the concentration of fluoride in the drinking water increases. Increased risk of dental caries may occur with less than 0.5 ppm intake of fluoride from drinking water *BEST* (2006).



Figure 2: “Mottled” teeth of a 56 year old female from Northern Sicily. Water in this area naturally contains 3-6 ppm of fluoride (Waldbott *et al.*, 1978).

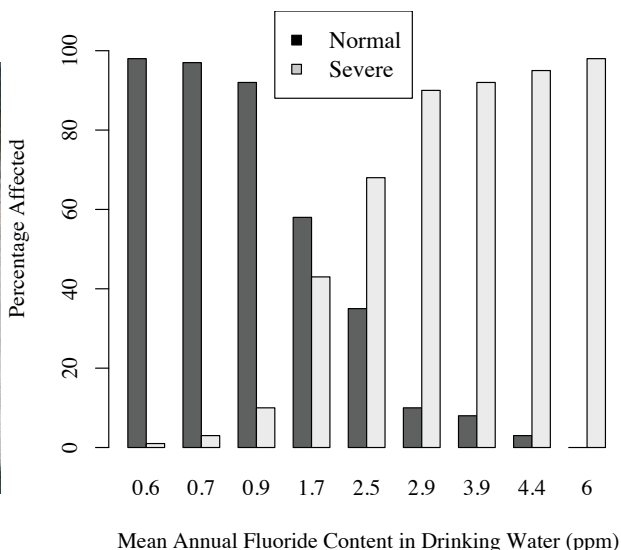


Figure 3: Relationship between natural fluoride concentration and prevalence rate of dental Fluorosis (Muhler and Hine, 1959).

Bone fractures are another response to chronic fluoride exposure. *BEST* (2006) suggests that individuals who drink water containing 4 ppm or more of fluoride over a lifetime are likely at increased risk for bone fractures.

## 4. Control and Regulation

The Environmental Protection Agency (EPA) regulates fluoride in drinking water. Fluoride in drinking water has an maximum contaminant level goal (MCLG) of 4 ppm (*BEST*, 2006). The maximum contaminant level (MCL) is also 4 ppm in drinking water. Put in place for cosmetic effects of dental fluorosis, the secondary maximum contaminant level (SMCL) is 2 ppm. These regulations were formulated based on the previously mentioned chronic responses to fluoride. Outbreaks in the US have primarily been due to pump failure (Akiniwa, 1997). Aside from the EPA's regulated levels the US Public health service recommends the “optimal” range for preventing tooth decay and dental fluorosis as 0.7 to 1.2 ppm depending on climate (*USPHS*, 1991). Estimates indicate that 1.4 million people drink public water with 2.0 to 3.9 ppm fluoride and 200,000 people drink public water with over 4 ppm fluoride (*BEST*, 2006).

No national regulations exist for fluoride in food or soil. The only nationally regulated fluoride gas is hydrogen fluoride which is listed as a hazardous air pollutant under the 1990 clean air act (EPA, 2007).

The author agrees with (BEST, 2006) that the EPA MCL should be lowered to reduce the risk of severe dental fluorosis in a portion of the population. A more stringent standards could help to reduce the number of children affected by dental fluorosis and balance the cost of increased treatment.

## 5. Conclusions

The exposure routes of fluoride are numerous and include food, water, air and soil. Fluoride is present in almost all food and water to some degree. Lack of fluoride can lead to dental caries (tooth decay). The acute toxic fluoride dose is reported as 0.1 to 8 mg F/kg body weight (Akiniwa, 1997). Symptoms of acute fluoride poisoning include vomiting, abdominal pains, diarrhea, convulsions, nausea, and fever (Waldbott *et al.*, 1978). Chronic overexposure to fluoride can lead to dental fluorosis and bone fractures. The only nationally regulated fluoride gas is hydrogen fluoride which is listed as a hazardous air pollutant under the 1990 clean air act (EPA, 2007). Fluoride in drinking water has an MCLG of 4 ppm (BEST, 2006). US Public health service recommends the “optimal” range for preventing tooth decay and dental fluorosis as 0.7 to 1.2 ppm (USPHS, 1991). Estimates indicate that 1.4 million people drink public water with 2.0 to 3.9 ppm fluoride and 200,000 people drink public water with over 4 ppm fluoride (BEST, 2006).

Future directions for this work include research on the economics of fluoridating our public water supply and a more formal justification for the lowering of the current MCL.



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