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**OVERVIEW**

The paper by Richard Sauerheber, titled Physiologic Conditions Affect Toxicity of Ingested Industrial Fluoride (*Journal of Environmental and Public Health*, Vol 2013, Article ID 439490) contains several errors and misconceptions that will be discussed below. More importantly the author implies that there is the possibility of acute fluoride toxicity from drinking fluoridated water that contains little or no calcium. That is a widely recognized impossibility regardless of the amount of calcium in the water.

The author begins by emphasizing the well-known effect of high concentrations of calcium in reducing the acute toxic potential of fluoride when ingested in large amounts. This effect is due to the formation of insoluble and poorly absorbable calcium fluoride which forms when the concentrations of calcium and fluoride are high enough to exceed its solubility product. In this context the issue is acute toxicity caused by those large amounts of fluoride and not the comparatively miniscule amounts ingested from drinking fluoridated water. The author is “mixing apples and oranges” and therefore, this has led to confusion. As seen later in his article, the author is aware of this fact by acknowledging that it is not possible to cause acute toxicity by drinking water containing fluoride at about 1.0 mg/L (ppm). However, in the Introduction he does not make that distinction but instead leaves it to the casual or uninformed reader to infer that ingestion of water without calcium, regardless of its fluoride concentration, may lead to acute fluoride toxicity.

The fact is that both calcium and fluoride in either of the two kinds of water, “natural” or not, are free and in the ionic state. This is true because the concentrations of calcium and fluoride present in virtually all surface waters (rivers, lakes, streams, etc.) and all fluoridated public water supplies are not high enough to reach the solubility product of calcium fluoride (3.4 x 10^{-11} at 18°C). For example, even in extremely “hard” water containing calcium at 200 mg/L the ion product is 1.4 x 10^{-11} when the fluoride concentration is 1.0 mg/L. Thus, the calcium and fluoride will not combine to form insoluble calcium fluoride and precipitate. Instead they will exist in their ionic states and in solution. This is also illustrated in Figure 1a of Sauerheber’s paper. Figure 1a shows that there is no calcium

Restricting the safety of water fluoridation to Sauerheber’s “natural” drinking water is clearly misleading. For many decades fluoride has been added to public drinking water supplies as sodium fluoride, fluorosilicic acid or sodium silicofluoride without adverse health effects.
Sauerheber claims that “pure pristine fresh drinking water” contains calcium but not fluoride and calls it “natural” water. Presumably such water is found in mountain streams, lakes, etc. However, he provides no reference or data to document the unlikely existence of “natural” or any other kind of surface water devoid of fluoride. Contrary to his assertion, fluoride has been a measurable component in every substance, animate or inanimate, that has ever been analyzed including sea water, polar ice caps and the atmosphere. This is consistent with the fact that fluoride is the 13th most abundant substance in the crust of the earth. Fluoride is a natural part of the biosphere. The author then refers to the compounds most commonly used to add fluoride to public drinking water supplies, sodium fluoride and fluorosilicic acid, as “synthetic industrial” compounds. Since these compounds do not contain calcium, Sauerheber implies that fluoridated water contains no calcium and therefore is toxic. That concept is erroneous as made clear by the facts that (1) over 200 million U.S. citizens safely consume fluoridated tap water daily throughout their lives and (2) all fluoridated public water supplies contain calcium. This is true because calcium is present in the source water before reaching the water treatment plants and is not removed during processing. For example Moor et al. (HHS J, 130-135, September 2006) reported calcium concentrations ranging from 1 to 135 mg/L in the tap waters of 39 major cities throughout the U.S. and Canada many of which fluoridate their public water supplies with sodium fluoride or fluorosilicic acid. The overall averages for U.S. and Canadian cities were 50.6 mg/L and 48.8 mg/L, respectfully.

Sauerheber briefly discusses the fact that, in the presence of hydrogen ions, fluoride forms hydrofluoric acid, HF. HF is a weak acid whose pKa is 3.4, similar to that of several important human biochemical metabolic intermediates. Since water always contains some concentration of hydrogen ions, HF is invariably present in all aqueous solutions. Its concentration depends on the pH of the solution. At pH 3.4, 50% of the total amount of fluoride in solution is ionic (F-) and 50% is HF. The percentage of HF increases at lower pH values and decreases at higher pH values as shown in the author’s Figure 2b. For example, fluoridated drinking water with a total fluoride concentration (ie, ionic F- plus HF) of 1.0 mg/L and a pH of 7 has an HF concentration of only 0.00025 mg/L. In the acidic environment of the stomach most of the fluoride is instantly converted to HF. If the pH were 2.0 then the HF concentration would increase from 0.00025 mg/L to 0.96 mg/L. Of course, the HF doesn’t remain at that concentration very long due to absorption into the systemic circulation and passage into the early small intestine. In these fluids the pH is close to or above 7 so HF is instantly converted back into ionic fluoride.

The question is: At what concentration and duration is HF likely to cause structural and/or functional effects to the gastric mucosa? Sauerheber stated that exposure to 20 mg/L HF for 15 minutes produced “structural damage” but did not provide a reference. We found that prolonged exposure of the canine gastric mucosa to 19 mg/L HF (1.0 mmol/L NaF in 0.1 mmol/L HCl, pH ~1.6) for 30 minutes caused no or only minor changes in the gross and histological appearances or to the functional characteristics of the tissue. At an HF concentration of 95 mg/L each of these variables showed measurable changes (Whitford et al, Digestive Diseases and Sciences 42: 2146-2155, 1997). It was concluded that the threshold HF concentration for structural and functional damage to the mucosa was more than 19 mg/L but less than 95 mg/L.

**SPECIFIC COMMENTS**

**Introduction**

- The typical plasma fluoride concentration in persons who drink fluoridated water is approximately 0.019 mg/L, not 0.2 mg/L as stated by the author.
- Contrary to the author’s claim, fluoride is a normal constituent of the mammalian bloodstream. This simply reflects the fact that fluoride is a normal constituent of the environment and all foods that mammals (and all other species) eat.
- Contrary to the author’s claim, fluoride is a normal constituent of the blood and therefore it is present in all tissues of the body including the hydroxyapatite of enamel.
- The author states that dental fluorosis is “unsightly as best” and afflicts ~5 million U.S. teenagers. In fact, the vast majority of cases are classified as very mild or mild so that the condition is unseen or just barely noticeable.
- The fluoride in bone is exchangeable (by hetero- and isoionic exchange) and therefore its uptake by bone is reversible, not irreversible as stated by the author. It is also mobilized from bone during the normal process of remodeling.
- The author states that “although large populations are reported to safely consume 1 ppm fluoride in water for long periods of time, this is when it exists naturally at this level.” Restricting the safety of water
fluoridation to Sauerheber’s “natural” drinking water is clearly misleading. For many decades fluoride has been added to public drinking water supplies as sodium fluoride, fluorosilicic acid or sodium silicofluoride without adverse health effects.

Results and Discussion

- Section 3.2: “Kidney dialysis patients have frequently been killed from fluoridated water during accidental fluoride overfeeds …..” Sauerheber cites Gessner et al. (1994) to support that statement. In fact, Gessner states that there were only four such deaths which, in view of the huge number of patients who were undergoing dialysis at that time, does not justify the term “frequently” as used by Sauerheber. It should be replaced by “rarely.” Gessner, in fact, stated that the safety of water fluoridation “is supported by the extreme rarity of incidents of over-fluoridation.” The author has failed to mention to note that tap water is not used for dialysis.

- Section 3.3: “The EPA currently allowed levels (of fluoride) in drinking water were found by the NRC to not be protective of human health.” The only health issue identified by the NRC in its 2006 report was the possibility of an increased risk of dental caries in cases of severe dental fluorosis with pitted enamel. The NRC committee therefore recommended that the EPA lower its maximum contaminant level goal (MCLG) for fluoride in drinking water from 4 mg/L to a lower level. At this writing the EPA has not changed the MCLG.

- Section 3.3: It is not true that “more human studies correlate 1 ppm fluoride ingestion with bone weakening than studies that do not.” Sauerheber cites no source to support that statement. The abundant literature dealing with the risk of bone fractures associated with long-term ingestion of fluoridated water was carefully examined and evaluated by the NHS Centre for Reviews and Dissemination at the University of York (2000). As shown on page 47 of that report, the consumption of fluoridated water had little or no effect, positive or negative, on the incidence of bone fractures.

- Section 3.3: Contrary to Sauerheber’s assertion, but in agreement with the 2006 NRC report, fluoride does not accumulate in the thyroid. Further, fluoride does not accumulate in the aorta, kidney or pineal gland unless the tissue contains ectopic calcifications.

- Section 3.3: The author claims that direct contact of the teeth with water-borne fluoride does not have an effect on dental caries. This may be true regarding the enamel per se. However, the fluoride concentrations in dental plaque are directly related to intraoral fluoride exposures including that from fluoridated water. Fluoride in dental plaque tends to inhibit bacterial acid production, inhibit demineralization and promote remineralization of the enamel.