SILICOFLUORIDES AND FLUORIDATION

Water fluoridation trials began in 1945 using sodium fluoride (NaF). In 1947, without formal announcement, the use of silicofluorides ("SiFs" – fluosilicic acid or hexafluorosilicic acid, H_2SiF_6, and sodium fluosilicate or silicofluoride, Na_2SiF_6) began. At the time, even though their stability in concentrated form was not in question, the nature and extent of their dissociation in dilute aqueous solution was uncertain. On the basis of laboratory studies on dental fluorosis effects and net skeletal retention, SiFs were proposed to undergo essentially complete dissociation at low concentration and would therefore be physiologically equivalent to NaF at one-sixth the molar concentration of NaF in dilute aqueous solution.[1] As early as 1935, however, SiFs and NaF were known to behave differently in the body, with SiFs producing significantly greater excretion of fluoride in urine than NaF.[2] Nevertheless, virtually all the extensive laboratory research on the biological properties and effects of fluoride in water has been performed using NaF rather than SiFs, on the premise that the latter are equivalent to NaF in their behavior and effects.

Unnoticed until recently is a report from the University of Hamburg, Germany, that is materially at odds with the notion that the biological behavior of SiFs does not differ significantly from that of uncomplexed NaF. This work was part of the 1975 PhD chemistry dissertation of Johannes Westendorf[3] under the direction of Professor Adolf Knappwost, who was searching for ways to increase the anti-caries activity of fluoride in saliva, based on the assumption – now known to be incorrect – that ingested fluoride confers significant protection against dental caries of the permanent teeth. This research, as published,[4] showed that, even at the low concentrations employed in fluoridation, one molar equivalent of SiF was substantially more potent as an inhibitor of acetylcholinesterase activity under physiological conditions (37°C and pH 7.4) than six molar equivalents of uncomplexed NaF.

Westendorf found that hydrolytic dissociation of SiF apparently does not go to completion but stops after loss of four fluoride ions to produce a difluorosilicate species like $[SiF_2(OH)_4]^{2-}$ and its protonated forms. He proposed that this difluorosilicate complex was responsible for a noncompetitive enzyme-inhibiting effect which, together with the competitive inhibition by the uncomplexed fluoride that is released, produces a significantly greater inhibition of cholinesterase activity than an equivalent nominal concentration of NaF alone. In studying the hydrolysis of other fluoro complexes, he found a similar dissociation of four fluoride ions from Na_3AlF_6 (cryolite), a loss of five from K_2GeF_6, and six from K_2SnF_6, but none from KPF_6 and KBF_4, both of which are reported to be physiologically inert.[5,6]

In regard to the already-mentioned 1935 report,[2] that work, conducted at the Ohio Agricultural Experiment Station, was primarily concerned with comparing the health effects on farm animals of various forms of fluoride-containing mineral supplements such as rock phosphate. In one of the ancillary experiments on rats, with equal dosage and equal amounts of fluoride retained, three times as much non-retained fluoride was excreted in the urine from sodium SiF as from NaF, from which more fluoride was eliminated in the feces. Apparently about three times as much fluoride had crossed the gut/blood barrier into the bloodstream from SiF than from NaF.

In another early report, in this case from researchers in the US Public Health Service,[7] urinary excretion of fluoride was compared in boys and men drinking water fluoridated with either SiF or NaF. For the boys, who were growing and therefore adding bone mass during the several years of the study, the time required for them to reach a steady-state output of urinary fluoride from either source of waterborne fluoride was longer than for the men. An even more revealing finding was that it took longer for the urine fluoride level to reach equilibrium in the younger males from SiF than from NaF, in agreement with the similar excretion pattern difference noted above in the rat experiment.[2]

In still another study, conducted at the Yerkes Primate Research Center in Atlanta, Georgia, about the same time as Westendorf's research, commercial fluosilicic acid was added to the distilled drinking water of squirrel monkeys for 14 months at a concentration equivalent to 1 and 5 ppm (mg/L) of F ion.[8] Morphological and cytochemical disturbances were found in the liver, kidney, and nervous system. Although exposure to NaF was not included, the report emphasized the fact the kidneys of monkeys ingesting SiF treated water "showed significant cytochemical changes, especially in the animals on 5 ppm fluoride intake in their drinking water."

This report also cited previous work by others showing that "fluoride has an inhibitive effect on the activity of succinate dehydrogenase. These studies indicate that under the effect of fluoride intake, a serious metabolic distress may develop in the kidneys." It was further noted that earlier workers had found that "inorganic fluorides have a strongly adverse effect on the activity of some enzymes and of these, mitochondrial enzymes, acid and alkaline phosphatases and ATP-utilizing enzymes and aldolase may be the most affected." Increased thirst and polyuria were also observed in the monkeys drinking SiF fluoridated water, as has also been observed in human clinical studies.[9]

If the authors of the foregoing work on squirrel monkeys had known of Westendorf's research, their investigation might well have taken a different turn and included a comparison of the effects of NaF in the drinking water with those of SiF. Even so, two of the three American experiments did com-

pare SiF and NaF and showed that they do not produce identical effects. Moreover, it is noteworthy that all three studies found that the strongest physiological effect of SiF was in the kidney, a point to consider in light of increased rates of kidney failure during recent decades.

As pointed out in a recent comprehensive review,[10] among the many different enzymes that initiate, control, and terminate various chemical changes in the body, acetylcholinesterase is one of the most fundamental. Therefore, in view of the extensive use of SiFs for water fluoridation (estimated to be 200,000 tons per year in the United States), Westendorf's seminal findings take on added importance in that they reveal that fluorosilicates are more potent in interfering with acetylcholinesterase activity than uncomplexed fluoride. These SiFs are industrial grade materials derived from HF and SiF_4 emissions that are collected in water as toxic by-products in the manufacture of phosphate fertilizers from fluoride-bearing rock phosphate. During that step concentrated aqueous solutions of fluosilicic acid, H_2SiF_6, are formed containing residual HF and SiF_4, together with variable low concentrations of contaminants like lead, arsenic, cadmium, beryllium, and heavy-metal radionuclides. Neutralization with soda ash then precipitates sodium fluosilicate, Na_2SiF_6, which is used as an alternative SiF to fluosilicic acid for water fluoridation.

Unfortunately, and as surprising as it may seem, neither of these commercial-grade SiFs have been properly (or officially) tested for safety in fluoridating drinking water. Indeed, their use in water fluoridation has even been called an "ideal solution to a longstanding problem"[11] as a way to dispose of a highly toxic by-product that is otherwise an enormous health hazard to the local environment. Meanwhile, our own research has revealed[12] and recently confirmed[13] a statistically significant association between silicofluoride-treated water and elevated blood lead levels, which, in turn, have disturbing implications in relation to their very unwelcome neurological and sociological consequences.

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Editor's note: The foregoing guest editorial was adapted from the foreword by the authors to their translation of Dr Westendorf's PhD dissertation cited in ref. 3 above.