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<http://www.fluoridealert.org/epa-sf.htm>

Written Objections and Request for Hearing in the matter of:

Sulfuryl Fluoride; Pesticide Tolerance. Final Rule.

Docket control number OPP-2003-0373

Submitted to

Office of the Hearing Clerk (1900C),
Environmental Protection Agency,
1200 Pennsylvania Ave., NW., Washington, DC 20460-0001

James Hollins, Information Resources and Services Division (7502C),
Office of Pesticide Programs, Environmental Protection Agency,
1200 Pennsylvania Ave., NW., Washington, DC 20460-0001

Public Information and Records Integrity Branch,
Information Resources and Services Division (7502C),
Office of Pesticide Programs, Environmental Protection Agency,
1200 Pennsylvania Ave., NW., Washington, DC 20460-0001

Submitted by

Paul Connett, Michael Connett, Ellen Connett,
Chris Neurath, and Phil Allen M.D. (Russian translation)
on behalf of

Fluoride Action Network

82 Judson Street, Canton New York 13617
Tel: 315-379-9200.

Email: pesticides@fluoridealert.org

Fax: c/o Connett at: 315-229-7421

and

Jay Feldman, Executive Director
Beyond Pesticides/National Coalition Against the Misuse of Pesticides

701 E Street, SE, Washington, DC 20003

Tel: 202-543-5450

Fax: 202-543-4791

Email: jfeldman@beyondpesticides.org

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SUMMARY:

We wish to appeal the US EPA's granting of a Final Rule to Dow for residue tolerances of inorganic fluoride and residue tolerances of Sulfuryl fluoride. We are submitting these Objections and a Request for a Hearing based upon the following:

RELIEF:

In terms of the relief sought, the submitters ask US EPA to rescind the Final Rule granted for Sulfuryl fluoride residue tolerances.

FEE WAIVER:

Fluoride Action Network (FAN) requests a fee waiver as it is dedicated to working in the public interest. FAN is currently in the process of obtaining non-profit status.

Under 40 C.F.R. § 180.33 (m) The Administrator may waive or refund part or all of any fee imposed by this section if the Administrator determines in his or her sole discretion that such a waiver or refund will promote the public interest or that payment of the fee would work an unreasonable hardship on the person on whom the fee is imposed. A request for waiver or refund of a fee shall be submitted in writing to the Environmental Protection Agency, Office of Pesticide Programs, Registration Division (7505C), Washington, DC 20460. A fee of \$1,700 shall accompany every request for a waiver or refund, except that the fee under this sentence shall not be imposed on any person who has no financial interest in any action requested by such person under paragraphs (a) through (k) of this section. The fee for requesting a waiver or refund shall be refunded if the request is granted.

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Appendix B – Fluoride & Bone Damage: Published Data

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Appendix C – Translation of Chinese Fetal Bone Study

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Appendix D – FAN's response to EPA's criticisms of submitted health studies.

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Appendix F – A comparison of a review of animal studies on fluoride's reproductive effects by Stan Freni (1994) and the DHHS (1991).

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Critique of EPA's Risk Assessment for Sulfuryl Fluoride

1. EXECUTIVE SUMMARY

On January 23, 2004, the US Environmental Protection Agency (EPA) issued a Final Rule that allows the highest residue tolerances on food commodities for inorganic fluoride in EPA's history. These residue tolerances are for Dow AgroSciences (DOW) use of Sulfuryl fluoride as a fumigant for over 40 food commodities. The Fluoride Action Network (FAN) together with Beyond Pesticides formally objects to EPA's Final Rule and requests a public hearing for adjudication on the following issues relating to the ruling:

- a) EPA has failed to wait for the National Research Council (NRC) to review its Maximum Contaminant Level (MCL) for fluoride in drinking water, which underpins the risk assessment used in its approval of DOW's petition (see Section 2).
- b) EPA has failed to re-examine the basis for, and derivation of, the MCL (see Section 2).
- c) EPA has failed to provide a rational examination of fluoride's damage to bone (see Section 2 and Appendices A & B).
- d) EPA has inexplicably developed a "safe chronic risk assessment dosage" for infants and children which is five times less stringent than the reference dose used for adults, and normally used for all age groups (see Section 3).
- e) EPA has failed to meet a key requirement of the Food Quality Protection Act (FQPA) - the extra protection needed for children (see Section 4).
- f) EPA has underestimated the daily doses of fluoride for children, particularly the exposure from fluoridated toothpaste (see Section 5).
- g) Based on recently published data (Levy 2003) it can be shown that some children will exceed both the previously accepted, and newly derived, "chronic risk assessment dosage", and thus EPA's final ruling granting DOW's petition for sulfuryl fluoride use must be overturned.
- h) EPA has inappropriately dismissed the literature submitted by FAN which documented FAN's health concerns (see Section 6 and Appendix D).
- i) EPA has failed to consider reasonable alternatives to DOW's proposal to substitute Sulfuryl Fluoride as a fumigant in place of ozone damaging methyl bromide (see Section 7).

j) EPA has inadequately analyzed the threat that Sulfuryl Fluoride poses to those applying this fumigant, other workers in facilities where it is used, and residents who live in the vicinity (see Section 7).

k) EPA may have underestimated fluoride exposure if it didn't include Sodium fluoride as a "List 4 Inert" in pesticide products used on food commodities (see Section 8).

l) EPA has not informed the public of the total inorganic fluoride levels estimated for Raisin and Dried Fruit. By not doing so, FAN cannot estimate total fluoride exposure levels from this Final Rule (see Section 9).

m) EPA has issued Residue Tolerances for 6 commodities that were not petitioned for (see Section 10).

n) EPA has approved excessive Residue Tolerances compared to what Dow petitioned for (see Section 11).

o) EPA has failed to adhere to statutes and guidelines (see Appendix K).

1.1. Final Rule predicated on an inadequate and outdated standard

EPA's MCL of 4 ppm, established in 1985, cannot be defended scientifically - a fact which possibly explains why EPA has rushed to approve DOW's petition before waiting to hear from the NRC panel which is currently reviewing the fluoride MCL (at EPA's request!).

EPA's decision to let DOW add another source of fluoride to the food supply is heavily predicated on the adequacy on the 1985 MCL standard. Therefore, FAN has provided an analysis of this standard. The analysis reveals critically important inadequacies with the MCL, including:

a) The MCL assumes crippling skeletal fluorosis is the only adverse bone damage that fluoride can cause, and ignores other bone damage (arthritic symptoms, reduced bone strength, mineralization defects, and exacerbation of bone disease in people with kidney disease) fluoride can cause *before* it cripples the skeleton.

b) The Lowest Observed Adverse Effect Level (LOAEL) (20 mg/day) selected for crippling fluorosis was derived from a limited set of data which provided inadequate information on how this LOAEL may vary across the range of the population, and across the range of pertinent exposure durations.

c) The scientist responsible for establishing the 20 mg/day LOAEL (Harold C. Hodge) ended up revising his estimate to 10 mg/day. EPA, however, ignored

this revision - even though it was made in 1979, 6 years before EPA issued the MCL.

d) The 20 mg/day LOAEL has been further undermined by data published since 1985, especially recent research on fluoride and bone strength.

e) The safety factor underpinning the MCL is dangerously low compared with other comparable contaminants in the environment.

f) The MCL is based on a misrepresentation of a large body of epidemiological data on skeletal fluorosis.

g) The EPA has openly acknowledged that the MCL can not be considered safe for people with kidney disease, thus violating EPA's mandate to protect sensitive subsets of the population.

1.2. EPA's new Final Rule has made a bad standard even worse

In its risk assessment for Sulfuryl Fluoride, EPA has managed to make a bad standard (the 1985 MCL) even worse. For, in granting DOW the right to use Sulfuryl Fluoride, EPA has actually increased the MCL (reference dose) for children.

By using non-conventional, poorly justified, and highly questionable assumptions, EPA has created a "reference dose" (or "chronic safe risk assessment dosage") for children which is up to FIVE times higher than the equivalent dose for adults.

EPA's use of a higher reference dose for infants and children than adults constitutes a sudden change in policy. In all recent risk assessments issued by EPA over the past few years, EPA has applied the same reference dose for both children and adults. This may not have been ideal, but any adjustments made to recommended maximum exposure levels for infants and children should have been in the direction of lowering them, not raising them.

EPA's sudden change in policy is as suspicious as it is unacceptable.

It is suspicious because, had EPA not increased the reference dose for children, then EPA's own data would show that children are already being exposed to doses which exceed EPA's previously estimated safe level. The implications are obvious: if children are already receiving too much fluoride, then there is no room for additional fluoride exposures, and EPA would be compelled to reject DOW's request to use Sulfuryl Fluoride which is known to leave fluoride residues on food.

EPA's manipulation of the safe fluoride dosage for children moves in exactly the opposite direction from the intentions of the Food Quality Protection Act (FQPA)

which requires EPA to issue standards that are particularly protective of children. In this case infants are less protected by a factor of 5 instead of more protected by a factor of 10X an overall swing against the FQPA recommendations to protect children by a factor of 50. This change also violates the EPA pesticide division's own requirements for appropriate risk factors.

1.3. EPA's exposure analysis underestimates current fluoride exposure among children

Along with increasing the allegedly safe dosage for children, EPA has also demonstrably underestimated the level of fluoride exposure among US children. FAN's analysis of EPA's exposure estimates reveals that:

- * EPA's supposedly conservative estimates of total fluoride exposure among children in 2 ppm areas have recently been shown to be exceeded by the Iowa Fluoride Project which studied children in 1 ppm areas!
- * EPA's supposedly conservative estimates of total fluoride exposure from toothpaste severely underestimate the contribution from this source. Indeed, EPA's estimate of the maximum daily exposure from toothpaste is a dose that is actually lower than the average daily exposure reported in most studies on the subject.
- * Despite EPA's assurance that children will not receive more fluoride than the agency's newly revised MCLs, recent data from the Iowa Fluoride Project shows that this claim is incorrect, and that some children are already receiving more fluoride than EPA's newly revised MCLs.
- * In assessing the exposure burden among US populations, EPA has ignored critical data on bone fluoride levels, serum fluoride levels, and urine fluoride levels. These omissions represent serious weaknesses in EPA's analysis.

1.4. EPA's detailed scrutiny of papers cited by FAN stands in stark contrast to its unquestioning endorsement of the MCL

In its response to papers submitted by FAN, the EPA displayed a level of intense scrutiny which is noticeably absent in its acceptance of the 1985 MCL.

Indeed, if the concern at the EPA was for the "adequacy of the current MCL," some of EPA's time would have been more wisely spent reviewing the "scientific literature" that underpins this standard with at least the same attention to detail which they used in analyzing the FAN submission. Had they done so they would have found it absurdly deficient.

There is clearly a double standard operating here. On the one hand, EPA blindly accepts the current MCL based upon the flimsiest of evidence for safety,

especially with respect to children, while nitpicking numerous articles which have been peer-reviewed and published in mainstream journals and indicate that the current MCL is unsafe . The use of a double standard is often very revealing. It usually indicates a body "defending" a position, rather than truly investigating an issue objectively.

EPA's reasons for dismissing the papers submitted by FAN are often inaccurate, inappropriate and occasionally cavalier. Remember - we are not discussing the standard for an additive to motor oil, but the standard for a substance which is currently causing between 30% to 50% of American children to be impacted with dental fluorosis, a condition caused by a fluoride-induced toxic effect on the enamel-forming cells.

While the EPA appears content to dismiss this as a "cosmetic effect," at the very least this condition is a biomarker of over-exposure to a physiologically active and cumulative toxin.

While US health authorities, unlike those in Europe, give little credence to the Precautionary Principle, one would have expected a little more caution from EPA when it comes to published research which has found, among other things, that fluoride accumulates in the human pineal gland, increases the uptake of aluminum into rat brain, lowers children's IQ in several studies, alters thyroid function in hyperthyroid patients, causes symptoms very similar to arthritis, and triples hip fractures rates in China at levels in drinking water close to the MCL.

While we accept that there are unresolved questions and uncertainties remaining in the literature concerning some of these health concerns, we believe that EPA's use of the uncertainties (to dismiss the concerns) is inconsistent with its mandate. We would argue, that the existence of uncertainty - especially considering the seriousness of the health effects under discussion - is not unusual, and should not be reason in and of itself to forego the need for precaution. We fear also that waiting for more and more epidemiological studies to quantify these dangers will subject our population to unnecessary risks and possibly irreversible harm.

2. EPA's RISK ASSESSMENT IS BASED ON AN OUTDATED AND UNDEFENDABLE MCL

EPA's Final Ruling in favor of DOW's use of sulfuryl fluoride as a fumigant on food is based upon a standard (the 1985 MCL for fluoride of 4 ppm) which is currently being reviewed by the NRC. The nearly 20 year old MCL does not factor in dozens of newer studies currently under review by NRC. EPA's scientist in charge of the sulfuryl fluoride assessment (Dennis McNeily) informed FAN, at the August 12, 2003 meeting of the NRC, that EPA would not issue its Final Ruling until after the NRC reported back to the EPA. Yet, EPA has done exactly the opposite. This undue haste is puzzling and unseemly, as it was EPA who

requested the NRC to review its own 1985 standard. It was an unwise and undefendable decision to give the DOW approval before the NRC review has been completed and it can only indicate to the public that EPA's ruling is based on other non-scientific considerations.

Even without the NRC's ongoing review of the literature in place, had EPA analysts simply applied the same level of scrutiny to the derivation of their 1985 MCL standard as they applied to the papers FAN presented to indicate the dangers of fluoride exposure, it would become clear to any objective analyst that the current MCL is woefully inadequate and unprotective of the public health.

2.1 A re-examination of EPA's 1985 MCL

Since EPA's current risk assessment is so heavily predicated on the adequacy of its 1985 MCL, we feel compelled to highlight the problems and limitations with the standard. As will be seen, this standard is so profoundly inadequate that any risk assessment which uses this standard as its scientific basis will be inherently flawed.

2.2 1985 MCL is designed to only protect against crippling skeletal fluorosis.

The most egregious problem with EPA's 1985 MCL is that it was crafted to only protect against a very advanced form of human fluoride toxicity - crippling skeletal fluorosis. As common sense alone should indicate, and as the scientific literature confirms, fluoride causes damage to bone long before it cripples the spine. Indeed, crippling skeletal fluorosis represents the final, most severe stage of fluoride's damage to bone. An adequate MCL for fluoride, therefore, would seek to protect against the bone damage which can occur prior to the final crippling stage. These pre-crippling skeletal effects include:

- Arthritic symptoms which mimic rheumatoid and osteoarthritis
- Reduced bone strength
- Reduced bone density
- Increased mineralization defects
- Exacerbation of bone disease in people with kidney disease

The current 4 ppm MCL fails to protect against any of these effects.

2.3 LOAEL for EPA's MCL based on a limited, inappropriate set of data

Before discussing the pre-crippling bone effects which EPA's MCL ignores, it is important to note that EPA's MCL for crippling skeletal fluorosis was based on an inappropriately derived LOAEL.

The LOAEL (20 mg/day) was derived from Kaj Roholm's research on a small group of cryolite workers working in Denmark in the 1930s (Roholm 1937; Brun 1941). Based on Roholm's research, however, it is not possible to reach firm conclusions about the LOAEL for crippling fluorosis. This is because:

A) Roholm was not able to determine the dose that would *not* cause fluorosis (e.g. he was not able to determine a NOAEL).

B) The subset of the population Roholm studied (adult male workers) is not representative of the population at large, particularly those subsets of the population known to be more vulnerable to fluoride, including people with kidney disease, people with chronic malnutrition, and children. Thus, based on Roholm's research, it is simply not possible to determine the LOAEL for people with kidney disease, for people with varying degrees of malnutrition, and for children.

C) The workers Roholm studied who developed crippling fluorosis had only worked at the plant for 10 to 25 years. Since skeletal fluorosis is dependent both on dose *and* duration of exposure, it is not possible - based on Roholm's research - to determine the LOAEL for people exposed to fluoride for longer periods of time than the workers in Roholm's study. For instance, based on Roholm's work, there is no way of determining what the LOAEL for crippling fluorosis is for a person (healthy or not) exposed to elevated levels of fluoride for 70+ years. It is inappropriate, therefore, for the EPA to base its MCL on a dose that is based on people who had only worked 10 years. Needless to say, someone living their lifetime in a 4 ppm community will be exposed to elevated fluoride for far more than 10 years.

D) While the LOAEL used by the EPA is based on Roholm's research, Roholm himself never concluded that 20 mg/day was the minimum dose that could cause crippling fluorosis (and he certainly didn't conclude that 20 mg/day was the minimum dose for all subsets of the population and for all durations of exposure). Roholm, who better appreciated the limitations in his data, stated that his dose information should be used as a guide for future investigation - not the final word!

E) The person who reached the conclusion that Roholm's research establishes 20 mg/day as the minimum dose that can cause crippling skeletal fluorosis was Harold C. Hodge, a prominent pro-fluoridation scientist. It should be noted, however, that Hodge - who repeated this conclusion in numerous papers for nearly 30 years - ended up revising his estimate towards the end of his career. In 1979, Hodge wrote that the LOAEL for crippling skeletal fluorosis may be as low as 10 mg/day. When the EPA established its MCL in 1985, it ignored Hodge's revised estimate, and stuck with Hodge's earlier claim.

2.4. More recent data highlights inadequacy of 20 mg/day LOAEL for crippling fluorosis

Recent research has further underscored the problem of EPA using 20 mg/day as the LOAEL for crippling skeletal fluorosis.

In 2003, Cao published a careful analysis of the doses causing crippling skeletal fluorosis in Tibet. According to Cao's analysis, the average dose causing crippling fluorosis was just 12 mg/day. Meanwhile, according to a recent study from Bo (2003), the average dose in an area of endemic skeletal fluorosis in China was found to be just 9.4 mg/day.

These findings from Asia are consistent with recent estimates from the National Research Council. In 1993, the NRC estimated that crippling skeletal fluorosis could be caused by exposure to 10 to 20 mg/day, while in 1997, the Institute of Medicine estimated that the milder stages of crippling fluorosis could be caused by 10+ mg/day.

Based on this data, it is completely inappropriate for the EPA in 2004 to still be using 20 mg/day as the LOAEL for skeletal fluorosis. The 20 mg/day LOAEL is an anachronism. It no longer represents the "Lowest Observed Adverse Effect Level" for crippling fluorosis.

At the very least, the EPA should be using the 10 mg/day LOAEL cited by the two most recent NRC reviews (NRC 1993; IOM 1997), and by Hodge himself (Hodge 1979).

2.5. EPA's MCL utilizes an irregular and inappropriate safety factor

Even if one assumed that the 20 mg/day is an appropriate LOAEL (which it is not), the MCL would still be completely inappropriate. This is because EPA applied an irregular, insufficient and unjustifiable safety factor in determining the 1985 MCL for fluoride. Normally, EPA applies a safety factor of 10 when a LOAEL is identified within the human population. However, in the case of crippling skeletal fluorosis, the EPA applied a safety factor of just 2.5.

We see no reason, however, why the EPA should apply a factor less than the standard 10 for this serious endpoint. Indeed, for the following three reasons, we believe it is imperative that if the EPA is to stick with its 20 mg/day LOAEL for crippling fluorosis, that it apply the standard safety factor of 10.

First, as noted above, the 20 mg/day LOAEL was derived from a population that didn't represent susceptible subsets of the population (e.g. people with kidney disease, people with chronic malnutrition, and children).

Second, as noted above, the 20 mg/day LOAEL was derived from workers who

had been exposed to fluoride for only 10 to 25 years. Considering, however, that skeletal fluorosis is both dose- and time-dependent, it is extremely probable that the LOAEL will be lower for people exposed to fluoride for longer periods of time (e.g. 70+ years).

Third, the 20 mg/day LOAEL refers to an extreme effect on bone (crippling skeletal fluorosis) that represents the final stage of fluoride-induced bone damage. There is ample evidence, however, that fluoride can damage bone before it cripples the skeleton, and thus a large safety factor (e.g. at least 10) is essential in order to protect against these pre-crippling effects.

2.6. EPA has misrepresented epidemiological data on skeletal fluorosis

In its recent risk assessment, EPA provides an entirely unacceptable defense of the 2.5 safety factor. According to the EPA, "the typical 100x factor used by the HED to account for inter- and intra-species variability have been removed due to the large amounts of human epidemiological data surrounding fluoride and skeletal fluorosis" (EPA Jan 20, 2004; p. 16).

The problem with this statement is that when the EPA established its MCL in 1985 it ignored and misrepresented most of the epidemiological data on skeletal fluorosis.

In its November 14, 1985 Final Rule, the EPA made a profoundly misleading statement concerning the epidemiological data on skeletal fluorosis. To quote:

"EPA notes that crippling skeletal fluorosis, rheumatic attack, pain and stiffness have been observed in a large number of individuals in other countries chronically exposed to fluoride in drinking water at levels of 10 mg/L to 40 mg/L" (*Federal Register*, Nov 14, 1985, p. 47144).

Anyone reading this statement could reasonably conclude that skeletal fluorosis was only found in communities where the water contained fluoride above 10 ppm. However, as had been documented repeatedly for over 40 years, skeletal fluorosis was known to occur at levels well below 10 ppm. Some examples:

In two of the most frequently cited papers on skeletal fluorosis in India, (Singh 1961, 1963) crippling fluorosis was observed at 1.2 ppm and between 1 and 2 ppm.

In 1970, Siddiqui observed skeletal fluorosis at 1.2-1.4 ppm, while Jolly (1970) reported fluorosis at 1.4 ppm.

Many more studies reported skeletal fluorosis above 2 ppm, but far below 10 ppm, including Pandit (3 ppm; 1940); Siddiqui (5.2 ppm; 1955); Kumar (6 ppm; 1963); and Krishnamachari (3.5-6 ppm; 1973).

Thus, EPA's statement in 1985 implying that skeletal fluorosis only occurred in communities with more than 10 ppm fluoride in the water was incorrect and misleading as it failed to acknowledge the abundant published evidence of skeletal fluorosis at water levels far below 10 ppm .

Meanwhile, many additional studies - published since 1985 - have confirmed the presence of skeletal fluorosis at between 1 and 2 ppm (see Xu 1997; Choubisa 2001, WHO 2002; Bo 2003). Indeed, in China today, any water concentration exceeding 1 ppm is considered a risk for developing skeletal fluorosis, while any concentration exceeding 2.5 ppm is considered a "high risk" (Bo 2003).

It is, therefore, blantly wrong for the EPA, in 2004, to claim that the existence of a large body of epidemiological data on skeletal fluorosis justifies its use of a lower than standard safety factor for fluoride. Indeed, most of the data published directly contradicts and undermines the validity of the 1985 MCL (Pandit 1940; Siddiqui 1955; Singh 1961, 1963; Kumar 1963; Sauerbrunn 1965; Jolly 1970; Siddiqui 1970; Juncos & Donadio 1972; Krishnamachari 1973; Johnson 1979; Xu 1997; Choubisa 2001, WHO 2002; Bo 2003; Cao 2003).

2.7. MCL does not protect people with kidney disease

MCL's are designed to protect the most susceptible subsets of the population from harm. According to the EPA's Final Rule for fluoride's MCL:

"[T]he Agency is acutely aware of sensitive subgroups in the population. Under the SDWA, EPA is charged with setting standards to protect the most sensitive subgroup of a population" (*Federal Register*, Nov 14, 1985, p. 47151).

However, despite being "acutely aware" of the importance of protecting sensitive subgroups, the EPA openly acknowledged in its Final Rule that the 4 ppm MCL could not be regarded as safe for people with kidney disease. To quote:

"The Agency feels that this RMCL provides an adequate margin of safety **except** in those very extreme cases involving severely renally impaired individuals who consume unusually high levels of fluoride due in part to polydipsia and other confounding factors" (emphasis added; *Federal Register*, Nov 14, 1985, p. 47152).

"Except" is the key word here. It shows that the EPA in 1985 ignored its legal obligation under the SDWA to protect a key subgroup of the population vulnerable to fluoride harm: people with kidney disease.

Based on the findings of Juncos & Donadio (1972) and Johnson (1979), it is clear that if the MCL for fluoride is to protect people with kidney disease, it needs to be

set below 1.7 ppm. The findings of Johnson (1979) are particularly important for the EPA to consider. It appears this paper was not considered by EPA in 1985, nor was it considered by the NRC in 1993.

Johnson (1979) reported the Mayo Clinic's findings of fluoride-induced bone damage in people with kidney disease. The conclusion of this paper is that fluoride levels of 1.7 to 2.0 ppm can exacerbate the bone disease of people with kidney disease. We believe this paper is particularly significant as it includes measurements of fluoride levels in the patients' serum and bones. In particular, the levels of fluoride found in the serum were greatly elevated (up to 14.1 $\mu\text{mol/L}$), and, in the person with the severest case of bone disease, these levels exceeded a) the estimated toxic threshold (10 $\mu\text{mol/L}$) for fluoride-induced bone damage (Pak 1989); b) the serum fluoride levels (9-11 $\mu\text{mol/L}$) consistently associated with reduced bone strength in animals (Turner 1995, 1996, 2001); and c) the serum fluoride levels associated with skeletal fluorosis (5+ $\mu\text{mol/L}$) in some humans (see Table 3a in Appendix B).

Indeed, based just on the serum fluoride findings of this study, we believe this paper establishes the lack of safety of 2 ppm fluoride for people with kidney disease. Based on current knowledge, it is simply unacceptable to allow people to attain 14 $\mu\text{mol/L}$ fluoride in the blood.

As such, any MCL for fluoride would need to be below 2 ppm in order to prevent the toxic buildup of fluoride in kidney patients' serum, as documented in the 1979 Johnson study.

2.8. EPA's MCL Undermined by Studies Published since 1985

New research, published since 1985, has highlighted yet additional problems with the EPA's MCL. In particular, the new research has highlighted the severe limitations inherent in EPA's focus on crippling fluorosis as being the only relevant adverse effect that fluoride can have on bone.

Some examples:

In the same year that the EPA approved the 4 ppm MCL, Arnala (1985) reported that fluoride concentrations exceeding 1.5 ppm were associated with an increase in mineralization defects in human bone.

A year later, Sowers (1986) reported a statistically significant increase in bone fractures in a 4 ppm community versus a control community with 1 ppm. In 1991, Sowers updated her findings, and noted that in addition to an increase in bone fractures, there was also a statistically significant reduction in bone mass in the 4 ppm community.

A year earlier, Phipps (1990) reported the results of a separate study which also looked at bone mass in a 4 ppm community. As with Sowers, Phipps found that the 4 ppm community had significantly less bone density than the 1 ppm community in the bone that she measured (the forearm).

While Phipps' study did not investigate bone fracture rates, a later study by Li (2001) did. As with Sowers, Li found a statistically significant increase in bone fracture rates, particularly hip fractures, in communities with excess fluoride. In a community with 4.3-8 ppm, Li found that the hip fracture rate was 3 times higher than the hip fracture rate in the control 1 ppm community. Li also found a doubling of hip fractures at 1.5+ ppm, however this effect was not statistically significant.

2.9. Recent fluoride/bone strength research eviscerates 4 ppm MCL

The likelihood that fluoride weakens bones and promotes fracture has been established by two powerful lines of scientific evidence published since 1985: human clinical trials and animal studies.

2.9a Human clinical trials published since 1985:

Since 1985, a series of well-controlled clinical trials - including the much anticipated NIH-sponsored 4 year double-blind trial (Riggs 1990) - have reported that osteoporotic patients treated with fluoride experience a higher rate of bone fractures (Dambacher 1986; Hedlund 1989; Bayley 1990; Orcel 1990; Riggs 1990; Schnitzler 1990; Gutteridge 2002).

Of particular interest are the clinical trials of Hedlund (1989); Bayley (1989), Orcel (1990), and Gutteridge (2002), as the doses used in these trials were only 21 to 25 mg per day.

While EPA dismissed the relevance of these clinical trials in their recent response to FAN, we find EPA's dismissal entirely unacceptable. Firstly, the EPA made a blatant mistake about the doses used in these trials by failing to convert the dose of sodium fluoride into the corresponding dose of fluoride ion. By failing to make this conversion, and by apparently failing to read the studies in question, the EPA stated that that the doses used in Hedlund (1989) were 50 mg/day when they were really 23 mg/day; that the doses used in Bayley were 60 mg/day when they were really 21 mg/day; and that the doses used in Gutteridge (2002) were 60 mg/day when they were really 25 mg/day.

Secondly, EPA dismisses the relevance of these trials by pointing out that the doses exceed the current LOAEL of 20 mg/day. However, we believe that a more appropriate response would be to calculate the safe dose by applying the standard margin of safety to this data. As noted earlier, the standard margin of safety applied when health effects have been found in humans is 10. If we apply

this standard safety factor of 10 to these trials, we get a reference dose of between 2.3 to 3.3 mg/day. Assuming 2 liters of consumption of water per day, the corresponding MCLG for fluoride (based on bone fractures from clinical trials) would range from 1.15 to 1.65 ppm.

2.9b Animal studies published since 1985:

Complimenting the clinical trials reporting increased bone fractures in humans receiving fluoride has been a series of well conducted animal studies (Mosekilde 1987; Turner 1992, 1993, 1995, 1996a, 1997, 2001; Lafage 1995; Sogaard 1995).

As multiple authors have noted (Sogaard 1995; Turner 1996b), the majority of animal studies investigating fluoride's impact on bone strength have found that fluoride reduces it.

In the extensive series of animal studies conducted by Dr. Charles Turner over the past 12 years, an extremely consistent finding has emerged: When rats are exposed to 50 ppm fluoride in their water, the strength of the bone is reduced. This finding was reported by Turner in multiple groups of animals in 3 separate studies in 1995, 1996a, and again in 2001.

The dose of the rats receiving 50 ppm F in these studies ranged from 2.1 to 3.5 mg/kg/day (Dunipace 1995, 1998), with the estimated average dose ranging from 2.2 to 2.7 mg/kg/day.

Considering the consistency of the finding of reduced bone strength among these rats, it can be reasonably concluded that the LOAEL for reduced bone strength is at, or below, 2.2 - 2.7 mg/kg/day.

If we then apply the standard 2 UFs of 10 to account for inter- and intra-species variations, we arrive at a safe human dose of 0.022 - 0.027 mg/kg/day. This dose is 4.2-5.2 times lower than EPA's current MCL for adults (0.114 mg/kg/day) and up to 26 times lower than EPA's current MCL for infants (0.571 mg/kg/day).

It should be noted, meanwhile, that other recent animal studies have found reductions in bone strength at doses below the doses found by Turner. For instance, Lafage (1995) found a reduction in bone strength in minipigs at an average dose of 0.91 mg/kg/day.

It might be appropriate, therefore, to use Lafage's dose as the LOAEL for reduced bone strength in animals. If we apply the standard 2 UFs of 10 to Lafage's data, we arrive at a safe human dose of 0.009 mg/kg/day, which is 12.7 times lower than EPA's current MCL for adults (0.114 mg/kg/day) and 63 times lower than EPA's MCL for infants (0.571 mg/kg/day).

Hence, whether we use human clinical trials reporting increased bone fractures, or animal studies reporting a fluoride-induced reduction in bone strength, it is clear that the safe dose for protecting against pre-crippling bone damage is well below what EPA currently considers safe.

2.10. The Need for a More Sophisticated Analysis of Fluoride's Impact on Bone

As the above discussion should help indicate, EPA's MCL is based on an exceptionally crude analysis of how fluoride impacts bone. The 4 ppm MCL is a) designed to only protect against the extreme crippling impacts of fluoride, is b) based on an inappropriate LOAEL derived from a small group of cryolite workers from the 1930s, and is c) acknowledged by EPA to be unsafe for people with kidney disease.

FAN has recently produced an analysis of fluoride and bone which we believe is much more meaningful than EPA's antiquated, and indefensible, 1985 assessment (see attached appendix A & B).

3. EPA'S ALTERATION OF MCL FOR CHILDREN IS CONTRARY TO STANDARD PRACTICE AND WITHOUT SCIENTIFIC MERIT

Considering the crude nature of EPA's 1985 MCL, it was shocking to read in EPA's January 20th risk assessment that EPA is actually increasing this MCL for children.

Indeed, in addition to unwisely accepting the 1985 MCL at face value, EPA has now made matters worse by manipulating the 1985 MCL in an unconventional and unscientific manner in order to reach its recent conclusions.

In short, EPA has taken an incredibly weak standard and made it five times less stringent for infants.

3.1. The alteration

Up until 2003, the dosage EPA used for chronic risk assessment for fluoride was 0.114 mg/kg/day. This dosage was obtained in the following way:

Based upon the questionable claim that it takes at least 20 mg per day of fluoride for 10-20 years to cause crippling skeletal fluorosis, 20 mg per day is considered the LOAEL for fluoride-induced health damage. EPA applies an arbitrary safety factor of 2.5 to this figure which in turn generates a purportedly safe dose of 8 mg/day. EPA then divides this daily dose by 70 kilograms bodyweight (the average weight of an adult), to yield a safe daily dosage of 0.114 mg/kg/day. Note the terminology - **dose** refers to mg/day; **dosage** refers to mg/kg-bwt./day.

Even though this dosage was derived from data obtained from adults, it has been used across the whole age range (without lowering it for the extra sensitivity we can anticipate for children, now mandated under FQPA, see introduction). For example, in a proposed new and modified tolerance petition for cryolite which appeared in the Federal Register, April 24, 2002, the following statement is made:

"For the chronic dietary exposure assessment, EPA has determined that the dose to be used for risk assessment for exposure to fluoride is **0.114 mg F/kg/day**, per the 1996 Cryolite RED. **This value is used for all population subgroups ...**" (emphasis added)

<http://www.epa.gov/fedrgstr/EPA-PEST/2002/April/Day-24/p9655.htm>

In the same document this standard is applied to children in a risk assessment very similar to that used for Sulfuryl Fluoride, except in this case the pesticide is cryolite. The authors conclude that:

"... Levels of fluoride in/on food from the agricultural use of cryolite plus fluoride levels in U.S. drinking water supplies, results in a daily intake of fluoride of approximately 0.064 mg/kg/day **for the most highly exposed population subgroup, children 1-6 years old. This is 56% of the dose used for chronic risk assessment (0.114 mg/kg/day)**...(emphasis added)" <http://www.epa.gov/fedrgstr/EPA-PEST/2002/April/Day-24/p9655.htm>

Indeed, this was the methodology accepted by the EPA from DOW prior to FAN's intervention in this matter. In DOW's original petition for tolerances on raisin and walnuts which appeared in the Federal Register, June 15, 2001, they, too, cite the same dosage normally used for chronic risk assessment, but they call it a "Maximum Concentration Limit Goal (MCLG)" :

"EPA (Cryolite RED decision, August 1996) **determined a Maximum Concentration Limit Goal (MCLG) of 0.114 mg/kg/day** for fluoride which provides protection from any known or anticipated health effects (emphasis added)"

<http://www.fluoridealert.org/pesticides/sulfuryl.fluoride.fr.ju2001.htm>

In this same petition DOW uses this same figure when considering exposure to children:

"The sub-population most susceptible to fluoride is children. For this reason a number of studies have attempted to quantify the fluoride intake from a variety of sources. The total daily intake of fluoride from water (used to prepare formula, juices, and other foods) for infants ages birth to 9-months ranged to 1.73 mg with means from 0.29 to

0.38 mg. Assuming a body weight of 10 kg, these amounts are equivalent to 0.03 to 0.04 mg/kg/day. **These levels of dietary exposure in combination with the potential dietary exposures that the proposed uses of ProFume** on stored walnuts and raisins would represent (chronic dietary exposures of 0.002419 mg/kg-bwt/day) **are considerably lower than the USEPA MCLG for fluoride of 0.114 mg/kg-bwt/day** (emphasis added)."

<http://www.fluoridealert.org/pesticides/sulfuryl.fluoride.fr.ju2001.htm>

Dow essentially repeats this same analysis using the same chronic risk assessment dosage, in its petition for fluoride tolerances on 40 foods, Federal Register, Feb 15, 2002:

"The sub-population most susceptible to fluoride is children. For this reason a number of studies have attempted to quantify the fluoride intake from a variety of sources. The total daily intake of fluoride from water (used to prepare formula, juices, and other foods) for infants ages birth to 9-months ranged to 1.73 mg with means from 0.29 to 0.38 mg. Assuming a body weight of 10 kg, these amounts are equivalent to 0.03 to 0.04 mg/kg/day. **These levels of dietary exposure in combination with the potential dietary exposures that the proposed uses of ProFume** would represent (chronic dietary exposures of 0.00004 mg/kg/bwt/day) **are considerably lower than the USEPA MCLG for fluoride of 0.114 mg/kg/bwt/day** (emphasis added)."

<http://www.fluoridealert.org/pesticides/sulfuryl.f.fr.Feb.15.2002.htm>

In the EPA's Final Ruling on DOW's petition for temporary tolerances on raisins and walnuts (Federal Register, Feb 7, 2002) it, too, reiterated the use of this same chronic risk assessment dosage:

"In consideration of the proposed temporary tolerances for walnuts and raisins, **the Agency used the maximum concentration limit goal (MCLG) of 4.0 ppm (0.114 mg/kg/day)**..." (emphasis added) <http://www.fluoridealert.org/pesticides/sulfuryl.f.fr.Feb.7.2002.htm>

Nowhere in the Cryolite, DOW or EPA documents prior to FAN's intervention is there any suggestion that there should be a different chronic risk assessment dosage for fluoride for different age ranges. However, after FAN had intervened and showed that children living in fluoridated areas are already exceeding this dosage, and thus could tolerate NO INCREASED FLUORIDE EXPOSURE from any new source, including residues from DOW's application of ProFume, EPA "changed the rules of the game" and introduced a different chronic risk assessment dosage for different age ranges. Here is the offending passage:

"Using body weight and water consumption estimates, the MCL has been converted from a concentration basis (mg/L) to an exposure basis (mg/kg/day). The resulting values for the population groups addressed in the fluoride risk assessments are as follows:

U.S. Population	0.114 mg/kg/day
Infants (< 1 year old)	0.571 mg/kg/day
Children 1-2 years old	0.308 mg/kg/day
Children 3-5 years old	0.182 mg/kg/day
Children 6-12 years old	0.100 mg/kg/day
Youth 13-19 years old	0.133 mg/kg/day
Adults 20+ years old	0.114 mg/kg/day
Females 13-49 years old	0.131 mg/kg/day"

<http://www.fluorideaction.org/pesticides/sf.jan.20.2004.epa.docket.pdf>

3.2. EPA's alteration is without scientific merit

The result of EPA's manipulation is blatantly absurd.

EPA is suggesting that infants less than 1 year of age can tolerate a fluoride dosage (i.e. mg/kg-bodyweight/day) which is FIVE times higher than the daily dosage deemed safe for adults.

EPA offers no new data or rational analysis to defend this radical change from its standard approach on the matter. Instead, EPA simply refers to the existence of a vague, but purportedly large, body of data that shows fluoride to be safe at these levels. EPA, however, fails to cite any specific studies which make it confident that these sweeping changes are safe.

We challenge the EPA, therefore, to produce the studies on fluoride and children which give the agency justification to increase the MCL dosages. What research is the EPA aware of which shows that children's bones are much less susceptible to fluoride-induced bone damage than adults?

Remember the original MCL from which the chronic risk assessment dosage was derived (0.114 mg/kg/day) came from data from *adult* Danish cryolite workers analyzed in the 1930s. Before such data can be increased for children and infants some clear biological rationale is required.

3.3. EPA's alteration is contrary to standard EPA practice

Without convincing data, the assumption that an adult-derived standard will be safe for everyone, including infants, is wishful thinking at best. EPA should not be creating protective regulatory standards for the whole population based on wishful thinking. It also violates the mandate in the Federal Register on the Food Quality Protection Act (FQPA) (http://www.epa.gov/opppmsd1/PR_Notices/pr97-

[1.html](#)) which demands a greater protective standard be applied for infants and children (see Section 6).

If one would want to make distinctions for different age ranges, *in the absence of animal, clinical or epidemiological data*, the correct procedure in a risk assessment would be to be conservative and LOWER the acceptable dose level for children, using an extra margin of safety to protect the young and most vulnerable.

3.4. Current evidence indicates children impacted by the same, or lower, levels which impact adults.

If we consider just the single end point of fluoride toxicity used by the EPA - damage to bone - there is every reason to believe that children's bones are damaged at the same, or even lower, levels of fluoride than adult bones.

The work of Alarcon-Herrera et al (2001) points in this direction. They found that the incidence of bone fracture increased linearly in children with the severity of dental fluorosis, a condition which impacts at least 1/3 of children living in communities with artificially fluoridated water (1 ppm fluoride) and nearly all children drinking water with levels of 4 ppm.

Furthermore, research in China has shown that bones from aborted fetuses show damage at levels of fluoride in bone (e.g. 300-400 ppm) far lower than the bone levels associated with comparable damage in adults (see appendix C).

In addition, as noted in our earlier submission, Schlesinger (1956) observed that children in a fluoridated area had a higher rate of cortical bone defects than children in a control unfluoridated area.

The possibility that children's bones may respond differently than adult bone to fluoride is extremely probable. The probability stems from the fact that the bones of children undergo a much greater turnover rate than in adults. One consequence of this fact is that children's bones accumulate fluoride at a much higher rate than adult bone (up to 87% versus an average of 50% in adults). Moreover, recent findings suggest that bones with high turnover rates are more susceptible to fluoride toxicity than bones with lower turnover rates. Kierdorf (1997, 2000), in studying the detrimental effects of fluoride on antler bone, has concluded that antler bone is more susceptible to fluoride toxicity due precisely to its rapid rate of mineralization.

Kierdorf's conclusion may help explain why fetal bone was damaged at fluoride bone levels (e.g. 300 to 400 ppm) below the levels associated with bone damage in adults. It also underscores the crudeness of EPA's rationale to increase the MCL for children. According to the EPA, a higher MCL for children is acceptable

because skeletal fluorosis is a chronic effect requiring prolonged exposure to fluoride. To quote:

"Skeletal fluorosis is an effect that requires (15-20 years) high exposures in order to be manifested. As such, infants and children will not exhibit this effect and an additional factor to account for potential enhanced sensitivity is not necessary" (EPA, Jan 20, 2004, p. 17).

The problem with EPA's rationale (besides being extremely brief for such a significant and sweeping change) is multifold:

First, it assumes (without scientific backing, and in contradiction to above-mentioned findings) that the only adverse effect fluoride can have on a child's bone is skeletal fluorosis.

Second, it assumes (without scientific backing, and in contradiction to above-mentioned findings) that children's bone responds to fluoride in an identical manner as adult bone.

And, third, it contains a significant error concerning the minimum duration of exposure which can cause skeletal fluorosis in humans. According to Roholm, crippling fluorosis can be caused after just 10 years of exposure (not 15 years as the EPA claims), while the earlier stages of clinical fluorosis can be caused after just 2 years of exposure (not 15). The fact that Roholm found clinical fluorosis after just 2 years (in an *adult*) is particularly significant considering that EPA's new MCL will allow children during their first 5 years of life a greater daily dose of fluoride (per kg of bodyweight) than the dose considered safe for adults.

3.5. Other concerns with children besides bone damage

Meanwhile, if we consider other health concerns besides bone damage, EPA's decision to increase the MCL for children becomes even more incomprehensible.

Of particular concern are the recent reports from China and Mexico reporting an impairment of cognitive function in children exposed to excess fluoride (Lin Fa-Fu 1991; Li 1994; Li 1995; Zhao 1996; Calderon 2000; Lu 2000; Xiang 2003 a, b).

While EPA has critiqued the methods of some of these studies, surely the seriousness and repetition of the findings should warrant at least some precaution among the EPA?

Indeed, in light of these findings of neurotoxicity, we are baffled by EPA's decision to raise the MCL for children. In doing so, we believe the EPA has exploited the existence of scientific uncertainty on this important endpoint in an exceptionally reckless manner unbecoming of its mandate to protect the public health.

3.6. EPA's alteration is conspicuously convenient

Why did EPA change its methodology at this juncture?

We have no way of knowing for sure, but it looks suspiciously like an attempt to get a dosage which a) is not exceeded by infants living in fluoridated communities and b) not exceeded when the incremental dosage is computed from the residues that will be left by the use by DOW Agrosiences' sulfuryl fluoride as a fumigant on many foods.

We note that in Table 5.1 in EPA's risk assessment of January 20, 2004, that the computed aggregate exposure for infants from sulfuryl fluoride residues, cryolite residues, background food, water, toothpaste and air, is 0.198 mg/kg/day. Without this new manipulation by the EPA, 0.198 mg/kg/day would represent 174% of the normally used chronic risk assessment dosage of 0.114 mg/kg/day and, consequently, DOW's petition would have to have been rejected. With the new manipulation, however, by the EPA 0.198 mg/kg/day becomes 35% of the new 0.571 mg/kg/day standard (see Table 5.1, <http://www.fluorideaction.org/pesticides/sf.jan.20.2004.epa.docket.pdf>).

Clearly, the acceptability of this manipulation requires a very careful analysis and **defense** and that is one of the reasons why FAN is submitting Objections and a Request for Hearing to EPA's Final Ruling (Federal Register, Jan 23, 2004).

4. EPA HAS VIOLATED MANDATE TO PROTECT INFANTS AND CHILDREN

Because it is commonly accepted that infants and young children are more susceptible to toxic exposure than adults, the Food Quality Protection Act (FQPA), passed into law on August 3, 1996, has mandated that the EPA design its regulatory decisions on pesticides to be MORE protective for children. This mandate for extra protection for children, when considering pesticide exposure, is very clearly spelled out in the FQPA:

When setting new tolerances, or reassessing existing tolerances or tolerance exemptions, EPA must now focus explicitly on exposures and risks to children and infants. EPA must, 1) explicitly determine that the tolerance, or exemption from tolerance, is safe for children; 2) consider the need for an additional safety factor of up to ten-fold to account for uncertainty in the data base relative to children unless there is evidence that a different factor should be used; and 3) consider children's special sensitivities and often unique exposure patterns to pesticides.

Federal Register on the Food Quality Protection Act (FQPA)

http://www.epa.gov/opppmsd1/PR_Notices/pr97-1.html

The analysis in section 3 above clearly indicates that the EPA is in violation of FQPA by not ensuring that its regulatory standards and decisions for fluoride are especially protective for children. Indeed, the very opposite was true when EPA created the new so called "safe" chronic risk assessment dosage (0.571 mg/kg BW/day) for children that is five times less protective than the adult "safe" dosage (0.114 mg/kg BW/day). In other words, EPA lowered the safety factor by 5 instead of raising it by 10 as recommended in the FQPA!

To summarize: establishing a safe dosage for infants which is five times higher than a safe dosage for adults, without any experimental or epidemiological data to support the claim, is absurd, reckless and goes in the opposite direction of the mandate and intentions of the FQPA which recommends consideration of adding a safety factor of 10 to account for children's extra sensitivity to toxic chemicals.

5. PROBLEMS WITH EPA'S EXPOSURE ANALYSIS

Another critical problem with EPA's risk assessment concerns EPA's estimates of the current and projected fluoride exposures among children.

5.1. EPA's Estimates of Total Exposure are Contradicted by Recent Empirical Data

Based on recent empirical data from the Iowa Fluoride Project (Levy 2003) it is clear that EPA has underestimated the total fluoride exposure among US children.

For instance, in Table 5.1 of EPA's Jan 20, 2004 risk assessment, EPA estimated the projected fluoride doses that children will receive *after* sulfuryl fluoride is added to the food supply. In addition to accounting for the projected exposure from sulfuryl fluoride residues, EPA's estimates were also based on children drinking water with 2 ppm fluoride.

Having accounted for these 2 extra sources of fluoride (sulfuryl fluoride + 2 ppm fluoride in water), one might reasonably expect that EPA's dose estimates would be higher than what has been documented among children currently living in 1 ppm communities with no sulfuryl fluoride exposure. However, this is *not* the case.

EPA estimates that the total fluoride dose among 1 to 2 year olds will reach 0.0877 mg/kg/day. According, however, to the Iowa Fluoride Project (Levy 2003; Appendix 2) this dose is already being exceeded by about 10% of 1 to 2 year olds (drinking water with just 1 ppm and unexposed to sulfuryl fluoride residues).

The situation is similar with EPA's dose estimates for the 3 to 5 year olds.

The EPA estimates that the 3-5 year olds will receive a dose of 0.0668 mg/day. However, according to the Iowa Fluoride Project, this dose is already being exceeded by 10 to 25% of the children in this age group.

The fact that EPA's estimates are based on the conservative assumption that children will consume water with 2 ppm fluoride in water, and the fact that these estimates from the EPA are already being exceeded by about 10% of children in 1 ppm areas (without exposure to sulfuryl fluoride residues), suggests the existence of serious problems with EPA's dose analysis.

5.2. EPA severely underestimated fluoride exposures from toothpaste

One readily identifiable incorrect aspect of EPA's analysis of fluoride exposure among children, is EPA's estimation of fluoride exposure from toothpaste.

According to EPA:

"Despite the variability in the estimates of ingested toothpaste, maximum exposures to fluoride observed in those studies appear to converge to approximately 0.3 mg/day (assuming 2 brushings per day)... The exposure estimates range from 0.004 to 0.04 mg/kg/day and should be considered conservative in nature..." (p. 34EP; Jan 20th, 2004).

EPA's assertion that 0.3 mg/day fluoride represents the "maximum" exposure from toothpaste is not correct. As will be seen, 0.3 mg/day of fluoride does not come close to representing the maximum reported exposure from toothpaste. Indeed, not only is 0.3 mg/day far lower than the reported maximum exposures from toothpaste, it is also lower than many of the reported *average* exposures.

In 1999, Levy presented data from 9 studies which measured the quantity of toothpaste ingested by children during brushing (see Appendix J). Of these 9 studies, 5 reported the maximum exposures. Of these 5 studies, all maximum exposures (range = 0.38 - 3.5 mg/day) exceeded EPA's so-called conservative estimate.

Perhaps more noteworthy, however, is the fact that the *average* fluoride exposures reported by all 9 of these studies (0.22 - 1.17 mg/day) almost always exceed EPA's purported *maximum* exposure (0.3 mg/day).

Moreover, according to the recent work of Bentley (1999), the average dose of fluoride ingested by 2 daily brushings of 1450 ppm toothpaste is 0.84 mg. Again, this *average* dose is actually higher (2.8 times higher) than the EPA's purported *maximum* dose of 0.3 mg/day. The maximum fluoride dose reported by Bentley (2.04 mg/day) is 6.8 times higher than EPA's purported maximum dose.

When expressing the dose in terms of bodyweight, Bentley (1999) found an average dosage of 0.06 mg/kg, and a maximum dosage of 0.14 mg/kg. This compares to EPA's purported maximum dosage from toothpaste of 0.023 mg/kg for the age of the children (2.5 year olds) being studied in this paper (Bentley 1999).

Finally, a more recent study by Levy (2003) has further undermined EPA's claim. According to this study, the 90th percentile fluoride dose from toothpaste was 0.75 mg/day in 3 year old children, and between 0.5 and 0.7 mg/day for 4 to 6 year olds. This 90th percentile dose is 1.7 to 2.5 times higher than EPA's purported "maximum" dose.

Based on this data, it is abundantly clear that EPA has made a serious error with respect to its analysis of fluoride exposure from toothpaste.

5.3. EPA has underestimated fluoride exposure from water consumption among infants

In its risk assessment, the EPA states that in determining the current exposures to fluoride, the EPA focused on people consuming 2 ppm fluoride in the water. To quote:

"In the current risk assessment, HED has assumed a residue level of 2 ppm for tap water... The use of 2 ppm fluoride in tap water... likely results in an overestimation of exposure for the general population, especially those on public water systems" (EPA, Jan 20, 2004, p. 29).

However, a close look at the data reveals either that EPA did not consistently use 2 ppm in its analysis, or it utilized water consumption rates well below EPA's 90th and 95th percentile rates.

This is particularly evident in EPA's exposure estimate for infants (< 1 year olds).

According to EPA, infants consuming water with 2 ppm will receive 0.1424 mg/kg/day. This dose estimate, however, understates the fluoride dose that would be expected based on EPA's own water consumption data published in April 2000. ("Estimated Per Capita Water Ingestion in the United States" - EPA-822-R-00-008).

According to EPA's water consumption data, infants drink more water per kilogram of bodyweight than any other age group. According to the data, 10% of infants drink at least 139 milliliters of water per kilogram of bodyweight. At an average weight of 7 kilograms, these infants would consume at least 0.278 mg/kg/day of fluoride. Thus, according to EPA's data, 10% of infants drinking 2 ppm will receive a fluoride dose roughly twice as high as estimated by the EPA (0.1424 mg/kg/day) in its January 20, 2004 risk assessment.

Moreover, according to EPA's water consumption data, at least 5% of infants drink 170+ milliliters of water per kilogram of bodyweight. Infants drinking this quantity of water would in turn consume 0.34+ mg/kg/day of fluoride if drinking water with 2 ppm fluoride. This dosage is 2.4+ times higher than the dose (0.1424 mg/kg/day) estimated by the EPA in its risk assessment.

Thus, it is apparent that EPA's estimates for fluoride consumption by infants underestimates the exposures that will be experienced by a sizeable percentage of infants drinking water at 2 ppm.

5.4. Some children are already receiving more fluoride than EPA's (modified) MCL

Based also on the recent empirical findings from the Iowa Fluoride Project (Levy 2003; Appendix 2), it can be seen that some children are already receiving more fluoride than EPA's newly modified MCLs for children.

For instance, EPA's modified MCL for 3 to 5 year olds is 0.182 mg/kg/day. EPA states that 3 to 5 year olds will not exceed this dose, even when living in a 2 ppm area. However, according to Levy (2003), some 3 to 5 year olds (*living in 1 ppm areas*) are receiving doses of up to 0.202, 0.223, 0.246, 0.254, and 0.283 mg/kg/day. All of these doses exceed EPA's modified MCL.

Regarding the 1 to 2 year olds, EPA's modified MCL is 0.307 mg/kg/day. EPA states that children living in 2 ppm areas will not exceed 28% of this dose. However, according to Levy (2003), some children in this age range (*living in 1 ppm areas*) are receiving up to 0.272 to 0.298 mg/kg/day. These 2 doses comprise 88% and 97% of the MCL respectively.

One can only wonder how much fluoride Iowa children would be receiving if they were living in a 2 ppm community. Certainly, many more of the children being studied would have exceeded the EPA's new MCLs.

Therefore, while we object to EPA increasing - without scientific justification (see section 3) - the MCLs for children, it is evident that some children are already receiving more fluoride than even the newly modified MCLs allow. Hence, even with the increased MCLs in place, it is apparent that there is no room for additional fluoride exposures in the US. DOW's request to use sulfuryl fluoride should be rejected accordingly.

5.5. EPA has ignored critical data on fluoride exposure

An additional problem with EPA's exposure analysis is the EPA's failure to address other measurements of fluoride exposure besides daily dose. These other measurements include the concentrations of fluoride in bone, the

concentrations of fluoride in serum, and the concentrations of fluoride in urine. EPA's risk assessment provides no data at all on any of these parameters.

The importance of these measurements is that they provide a critical indication of the type of cumulative fluoride burden being experienced by various segments of the population. EPA's failure to confront and discuss the data on any of these critical parameters is an important omission in its risk assessment which weakens its analysis.

In a recent analysis of published data by FAN (see appendix A & appendix B), it was found that at, or below, the current MCL of 4 ppm, some members of the population are attaining levels of fluoride in their blood, bone, and urine which have been associated with damage to bone.

We believe that if some members of the population are exhibiting levels of fluoride in the blood, bone or urine which equal or exceed the levels at which damage is known to occur, then any further additions of fluoride to the food supply should be prohibited. While the data on these parameters is still limited, we believe that there is enough current data to tentatively conclude that some people are being exposed to fluoride levels that cannot be considered safe. If EPA disagrees with us on this assessment, it needs to provide a convincing explanation to refute the data FAN has compiled (see appendix A & appendix B). It cannot simply ignore the problem, as it seems to have done in its January 20th, 2004 risk assessment.

6. EPA'S ATTEMPT TO DISMISS PAPERS IN THE SCIENTIFIC LITERATURE, SUBMITTED BY FAN, IS INAPPROPRIATE AND CAVALIER WITH RESPECT TO PROTECTING THE PUBLIC HEALTH.

In the preamble by Baetcke et al (November 18, 2003) of their review of articles submitted in FAN's appeal of DOW's petition, they state their intent was "to consider the studies submitted by FAN and identify those that may raise concern for the adequacy of the current MCL for fluoride." While we appreciate the amount of time and effort that has gone into reviewing the literature we submitted, it is unfortunate that it has taken the attempt by DOW to register a fluoride pesticide to motivate EPA to take this matter seriously.

Moreover, if indeed the concern at the EPA was for the "adequacy of the current MCL" some of Baetcke et al. time would have been more wisely spent reviewing the "scientific literature" that underpins the 1985 standard, with at least the same attention to detail which they used in analyzing the FAN submission. Had they done so, they would have found it absurdly deficient (see Section 2 above).

There is clearly a double standard operating here: on the one hand, it blindly accepts an MCL based upon the flimsiest of evidence for safety, especially with

respect to children, while nitpicking or ignoring numerous articles documenting harm which have been peer-reviewed and published in mainstream journals. The use of a double standard is often very revealing. It generally indicates a body "defending" a position, rather than truly investigating an issue objectively.

The EPA's dismissal of practically all the studies that FAN discussed and referenced is inappropriate, unscientific and often cavalier. At a minimum, fluoride ingestion is currently causing between 30-50% of American children to be impacted with dental fluorosis and some of this is of a truly disfiguring nature. While EPA appears content to dismiss this as a "cosmetic effect," the condition is a biomarker of over-exposure to a physiologically active and cumulative toxin. One of the key starting points for any risk assessment is hazard identification, and while some of the studies we cited may not allow definition of a LOAEL, they certainly identify a potential hazard.

The US, unlike European countries, gives little credence to the Precautionary Principle, yet one would have expected a little more caution in the face of so many uncertainties. Published research in peer-reviewed journals from around the world has found, among other effects, that fluoride accumulates in the human pineal gland, increases the uptake of aluminum into rat brain, lowers children's IQ, adversely affects the brain (appendix H), adversely affects male reproductive organs (appendix G) causes symptoms very similar to arthritis at levels less than the MCL, and triples hip fractures rates at levels in drinking water close to the MCL. Waiting for more and more epidemiological studies to quantify these dangers, while condoning exposure to yet another new source of fluoride in food residues, may well subject our population to unnecessary risks and possibly irreversible harm.

In appendix D we have examined EPA's responses to several key studies including the following areas: fluoride's effects on bone, the pineal gland, the thyroid gland, G-proteins, human fertility and reproduction, the central nervous system, chromosomal damage, osteosarcoma incidence, and individuals hypersensitive to fluoride.

7. EPA HAS FAILED TO CONSIDER REASONABLE ALTERNATIVES TO DOW'S USE OF SULFURYL FLUORIDE

Under the guidance of the United Nations Environment Programme, the Montreal Protocol on Substances that Deplete the Ozone Layer was drawn up in 1987. In 1992 Methyl Bromide was added to the list. In 1997 a global phase-out schedule for Methyl Bromide was put in place. The US EPA and Dow, the producer of Methyl Bromide, were well aware of the impending fate of this ozone depleting fumigant.

While other countries (for example, Denmark and the Netherlands) have ended the use of Methyl Bromide earlier than the phase-out date of January 1, 2005 for

developed countries, and have employed satisfactory and much safer alternative fumigants, such as steam and carbon dioxide, Dow and the US Department of Agriculture have been steadfastly promoting Sulfuryl Fluoride as if it were the only alternative fumigant for many food commodities. While Dow may have an economic interest in its single preoccupation with Sulfuryl Fluoride, the consumers of the food that will be fumigated with it, as well as the fumigators, would be better served if safer alternatives were investigated. We believe that both citizens and fumigant workers will be exposed to unnecessary risk if EPA's Final Rule is implemented.

FAN applauds the decision to phase out all known ozone depleters. We have no wish to prolong the use of Methyl Bromide. In fact, we wish that the US had been as swift in doing this as some European countries. It is unfortunate that it was not phased out years ago when its potential as an ozone depleter was first recognized. But it would double this misfortune if we were to be rushed, by our own tardiness, into using a dangerous substitute. Fortunately, there are alternatives to the use of Sulfuryl Fluoride as a fumigant. Not only are they available, they are in current use in many areas in the US and abroad.

The fumigant alternatives for the food categories for which Sulfuryl Fluoride has been approved are clearly listed on EPA's website "The Phaseout of Methyl Bromide" under "U.S. Matrix of Alternatives" available at <http://www.epa.gov/spdpublic/mbr/>.

7.1 Risk to Workers

Sulfuryl fluoride is well known for its extreme acute toxicity. The risks to the brains of workers if exposure should occur and the potential for dire health risks if high levels of fluoride accumulates in their bodies should be unacceptable in a civilized society. The tragedy is that there is a potential for worker exposure incidents at the same time there are safer alternatives available to avoid these risks.

While EPA's mission is "to protect people," it has no regulatory function to assess the risks to workers. Our Objection aside, we urge EPA to work with other agencies to construct a regulatory mechanism that would insure that health risk assessments for worker exposure to chemicals are performed.

8. EPA RISK ASSESSMENT LACKS CLARITY ON FLUORIDE EXPOSURE FROM "INERTS"

Sodium fluoride is approved for use in pesticides as a "List 4-B inert". FAN has asked EPA, on several occasions since March 1, 2004, if Sodium fluoride as a "List 4-B inert" was in pesticides that are used on food, and if EPA had considered this route of exposure. EPA has failed to respond to this request as of

this submission. Without this information, FAN cannot estimate fluoride exposure from the consumption of food.

There are a minimum of 198 pesticides (Appendix 1) that have residue tolerances for food commodities that will be fumigated with Sulfuryl fluoride. There is no information available to the public to know

- if Sodium fluoride as a "List 4-B inert" is approved for use in any or all of the 198 pesticides
- if EPA has calculated the levels of Sodium fluoride as a "List 4-B inert" in all pesticides used on food commodities in its exposure assessment: including the number of applications, and multiple products ,for one pesticide.
- if EPA has ever tested food commodities for fluoride from crops treated with pesticides that contain Sodium fluoride as a "List 4-B inert"
- if EPA has ever tested food commodities for fluoride levels from crops treated with Cryolite. If such testing was done, has EPA ever released the results to the public?
- of the 198 pesticides cited above, 31 are fluorinated pesticides (Appendix 1). Does EPA know if any of these 31 fluorinated pesticides release fluoride as a metabolite? Have they included this in their assessment?

EPA has not stated in its documents, included in the Final Rule docket, that Sodium fluoride as a "List 4-B inert" was considered in its exposure assessment. The potential for a very high level of exposure to, and risk from, fluoride exists for the consumer. **Because fluoride accumulates in the human body the public has a right to know which foods contain it.** It is incumbent upon the EPA to respond to this question.

Categories that Sulfuryl fluoride has been approved for use and the pesticides used in these categories:

Appendix 1 - Summary of the residue tolerances for Sulfuryl fluoride and the food categories with Residue Tolerances in these categories
<http://www.fluorideaction.org/pesticides/sf.inerts.all.categories.htm>

Appendix 1-A. 64 pesticides used on Barley
<http://www.fluoridealert.org/pesticides/sf.inerts.barley.htm>

Appendix 1-B. 83 pesticides on Corn

<http://www.fluorideaction.org/pesticides/sf.inerts.corn.htm>

Appendix 1-C. 18 pesticides used on Dried Fruit

<http://www.fluorideaction.org/pesticides/sf.inerts.fruit-dried.htm>

Appendix 1-D. 104 pesticides used on Grape

<http://www.fluorideaction.org/pesticides/sf.inerts.grape.htm>

Appendix 1-E. 6 pesticides on Millet

<http://www.fluorideaction.org/pesticides/sf.inerts.millet.htm>

Appendix 1-F. 277 pesticides on Nut

<http://www.fluorideaction.org/pesticides/sf.inerts.nuts.htm>

Appendix 1-G. 33 pesticides on Oat

<http://www.fluorideaction.org/pesticides/sf.inerts.oat.htm>

Appendix 1-H. 47 pesticides on Rice

<http://www.fluorideaction.org/pesticides/sf.inerts.rice.htm>

Appendix 1-J. 56 pesticides on Sorghum

<http://www.fluorideaction.org/pesticides/sf.inerts.sorghum.htm>

Appendix 1-K. 96 pesticides on Wheat

<http://www.fluorideaction.org/pesticides/sf.inerts.wheat.htm>

Questions related to specific residue tolerances for Sulfuryl fluoride

Corn, field, grits, postharvest.

Residue tolerance of 15.0 ppm for "Sulfuryl fluoride."

FAN questions this extraordinary high tolerance level. Is this a typographical error? EPA needs to clarify if this commodity is fed to farm animals or is consumed by humans.

Pistachio, postharvest.

It is confusing to have Pistachio in a separate category as it is included in the "Nut, Tree, Group 14" category. Will "Pistachio" be treated two times with Sulfuryl fluoride, eg: "Nut, Tree, Group 14" and "Pistachio, postharvest". EPA needs to clarify.

Sorghum, grain, postharvest

Will Sulfuryl fluoride be used on "Sorghum, Grain" only, or will be used also on the category: "Sorghum, Grain, Grain"? EPA needs to clarify.

9. EPA HAS NOT INFORMED PUBLIC OF TOTAL INORGANIC FLUORIDE LEVELS ESTIMATED FOR RAISIN & DRIED FRUIT

Raisin, Grape.

FAN has requested EPA's calculated estimates for total fluoride levels on Raisin on several occasions. FAN submitted the first request by email to Dennis McNeilly on March 1, 2004. EPA has failed to respond to this request as of this submission. Without this information, FAN cannot estimate fluoride exposure for children and high-end consumers. EPA needs to release its estimates for total fluoride levels on Raisin.

There is no information available to the public to know

- how many times various Cryolite products can be applied to grape.
- if each application of Cryolite to grape has an individual residue tolerance of 7 ppm, or if 7 ppm is the cumulative residue tolerance level for all applications of Cryolite to Grape.
- how many times Sulfuryl fluoride can be applied to Raisin.

If fluoride residues from cryolite on grape is 7 ppm, then one would expect the residues on raisin to be several times more because of the water lost in drying. Dried fruit weighs at least 2X less and maybe as much as 6X or more times less than fresh fruit. Since fluoride is not lost or destroyed in the drying process, the fluoride concentration would be 2 to 6X greater than 7 ppm! EPA must reveal the total fluoride residues they have calculated for Raisin including in this total the effect of drying on the final fluoride residue.

Note: Federal agencies and advocate groups are urging parents to provide healthier snacks to children to prevent obesity. Raisin is the number one dried fruit that children consume. The consumption of Raisin by children is likely to increase. The public does not know if EPA has factored this into its fluoride exposure assessment.

Dried Fruit (except Raisin)

Clarification is needed from EPA on the fruits that come under this category.

The public do not know if the following fruits are included in this category:

apricot, blackberry, blueberry, boysenberry, cherry, sweet and tart, cranberry, dewberry, kiwifruit, loganberry, melon, nectarine, peach, plum, prune, dried, raspberry, strawberry, youngberry.

These fruits have fluoride residue tolerances of 7 ppm, except Kiwifruit which is 15 ppm, from the use of Cryolite.

If these fruits come under this category, has EPA calculated the total fluoride residues from both cryolite and Sulfuryl fluoride.

Also, are figs and dates included in this category? EPA needs to inform the public of all the fruits that come under this category.

We repeat these comments we made on Raisin, above, for Dried Fruit. If cryolite fluoride residues on fresh fruits are 7 ppm (excepting Kiwifruit which is 15 ppm) then one would expect the residues on dried fruits to be several times more because of the water lost in drying. Dried fruit weighs at least 2X less and maybe as much as 6X or more times less than the fresh fruit. Since fluoride is not lost or destroyed in the drying process, the fluoride concentration would be 2 to 6X greater than 7 ppm!

EPA must reveal the total fluoride residues they have calculated for each fruit including in this total the effect of drying on the final fluoride residue. The public has a right to know what they are. FAN cannot estimate fluoride exposure for children and high end consumers without this information.

Note: The public does not know if EPA has factored into its fluoride exposure assessment high end users of dried fruit such as outdoor-enthusiasts and school children.

10. EPA HAS ISSUED RESIDUE TOLERANCES FOR 6 COMMODITIES THAT WERE NOT PETITIONED FOR.

We question the validity of the issuance of Residue Tolerances for commodities that were not petitioned for. The commodities are:

- Barley, bran, postharvest
- Barley, flour, postharvest
- Barley, pearled, postharvest
- Corn, aspirated grain fractions, postharvest
- Oat, flour, postharvest
- Oat, rolled, postharvest

These commodities are not in Dow's petition of February 15, 2002. Because they were not petitioned for, the Tolerances must be withdrawn. EPA must clarify.

11. EPA HAS APPROVED EXCESSIVE RESIDUE TOLERANCES COMPARED TO WHAT DOW PETITIONED FOR.

There are three tolerances that have intolerable excessive increases in the Final Rule compared to Dow's petition in the February 15, 2002, Federal Register. The increases are intolerable and should be withdrawn.

Corn, field, grits, postharvest.

The tolerances for "Sulfuryl fluoride" in the Final Rule vs Petition:

15.0 ppm vs 0.04 ppm. respectively.

We can only assume that this is a typographical error.

Wheat, flour, postharvest:

The tolerances for "Fluoride" in the Final Rule vs Petition:

125.0 ppm vs 10 ppm respectively

Wheat, milled byproducts, postharvest:

The tolerances for "Fluoride" in the Final Rule vs Petition:

130.0 ppm vs 35 ppm respectively.

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NOTE: References for the accompanying appendices are listed with the appendix

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