January 15, 2016

Ruth Lunn, Dr.P.H.
Director, Office of the Report on Carcinogens
National Toxicology Program Division
National Institute of Environmental Health Sciences
111 T.W. Alexander Drive
P.O. Box 12233
Research Triangle Park, NC  27709

Re: Request for Information on Nominated Topics—Fluoride

Dear Dr. Lunn:

On behalf of our 158,000 members, the American Dental Association is pleased to offer comments on fluoride being one of several substances the National Toxicology Program (NTP) has been asked to evaluate as a potential cancer risk. We offer these comments in response to your Federal Register notice of October 7, 2015 (80 FR 60692).

Fluoride is a naturally-occurring mineral that helps prevent cavities in children and adults by making the outer surface of the teeth (enamel) more resistant to the acid attacks that cause tooth decay. It can be applied topically (e.g., fluoride toothpaste, fluoride varnishes, etc.) and/or ingested systemically (e.g., community water fluoridation, dietary fluoride supplements, etc.). The Centers for Disease Control and Prevention (CDC) has hailed community water fluoridation as one of ten great public health achievements of the 20th century.¹-²

For the last 70 years, people have raised well-meaning questions about the safety and effectiveness of fluoride, including whether fluoride is somehow associated with cancer. According to the American Cancer Society, most of the cancer-related concern about fluoride seems to be around osteosarcoma (bone cancer).³

Last April, the U.S. Public Health Service (USPHS) noted there was no compelling evidence that fluoride is associated with osteosarcoma.⁴⁻⁵ The announcement was based on a four-year evidence review of the effects of fluoride on human health, including any potential links with cancer. After careful review, the USPHS concluded that the available literature does not support classifying fluoride as a carcinogen.⁶

More recently, Dr. Vivek H. Murthy, United States Surgeon General, issued a statement⁷ reiterating what his predecessors have said⁸⁻¹² for years—that optimizing the level of fluoride in community water supplies is still one of the safest and most beneficial public health measures communities can take to prevent tooth decay, even with the widespread availability of fluoride from other sources, such as fluoride toothpaste.¹³⁻¹⁴

In the nine months since the USPHS announced there was no compelling evidence that fluoride is associated with osteosarcoma, we are not convinced a sufficient number of new
high-quality studies have been published to generate a high-quality systematic review at this time. However, we do believe the public would benefit from knowing more about the therapeutic range of water fluoridation up to limits set by the Environmental Protection Agency (EPA).

Enclosed you will find our comments on key studies examining potential associations between fluoride and cancer in humans. Later this year, we will be publishing an updated version of *Fluoridation Facts*—the ADA’s premier informational resource on community water fluoridation. We will be happy to share a copy when it is released.

*Fluoridation Facts* provides answers to frequently asked questions about fluoride and community water fluoridation. Our goal is to provide clear answers—supported by thousands of credible scientific articles—to help policy makers and the public navigate through the many myths and misperceptions about fluoride.

Again, we welcome calls for new studies about the safety and public health benefits of fluoride. However, we are not convinced a sufficient number of new high-quality studies are currently available to produce a high-quality systematic review at this time.

We appreciate the opportunity to comment on this important public health issue. If you have any questions, please contact Mr. Robert J. Burns at 202-789-5176 or burnsr@ada.org. Information is also available at ADA.org/fluoride.

Sincerely,

Carol Gomez Summerhayes, D.D.S.
President

Kathleen T. O'Loughlin, D.M.D., M.P.H.
Executive Director

Enclosures (2)
References

5 Katherine Weno, Centers for Disease Control and Prevention, Statement on the Evidence Supporting the Safety and Effectiveness of Community Water Fluoridation, April 2, 2015.
The American Dental Association (ADA) is pleased to offer our technical comments regarding the nomination of fluoride as a substance for possible review in future editions of the National Toxicology Program (NTP) Report on Carcinogens (RoC). We offer these comments in response to your Federal Register notice of October 7, 2015 (80 FR 60692).

The ADA recommends that the Office of the Report on Carcinogens (ORoC) should:

Refer to the *Fluoride: Exposure and Relative Source Contribution Analysis* documents developed by the Environmental Protection Agency’s (EPA’s) Office of Water (OW) published in Federal Register notices of January 19, 2011 (76 FR 3422) and April 6, 2011 (76 FR 19001) as well as the comments on these documents submitted by the ADA on June 30, 2011 as part of its review.

The ADA comments of June 30, 2011 are attached as Appendix 1.

**Consider the following studies in different populations and at different times that have failed to demonstrate an association between fluoridation and the risk of cancer.**

Since community water fluoridation was introduced in 1945, numerous epidemiologic studies in different populations and at different times have failed to demonstrate an association between fluoridation and the risk of cancer. Studies have been conducted in the United States, Japan, the United Kingdom, Canada and Australia. In addition, several independent bodies have conducted extensive reviews of the scientific literature and concluded that there is no relationship between fluoridation and cancer.

A study from England published in January 2014 was the largest study that has ever been conducted examining the possible association between fluoride in drinking water and risk of osteosarcoma or Ewing sarcoma. It analyzed 2,566 osteosarcoma and 1,650 Ewing’s sarcoma cases in the period between 1980 and 2005. The findings from this study provide no evidence that higher levels of fluoride (whether natural or added) in drinking water in Great Britain lead to greater risk of either osteosarcoma or Ewing sarcoma.

In 2011, fluoride was evaluated by three different entities as a potential carcinogen and each time was found not to cause cancer. The results mentioned below add to the growing body of information that demonstrates no association between fluoridation and the risk of cancer.

**Harvard Study on Osteosarcoma**

A Harvard study released in July 2011 in the *Journal of Dental Research* found that bone fluoride levels are not associated with osteosarcoma. A team of researchers from Harvard University, the Medical College of Georgia and the National Cancer Institute analyzed hundreds of bone samples from 137 patients with osteosarcoma and a control group (N=51) to measure fluoride levels in the bone. The study from nine U.S. hospitals over an eight-year period was considered the most extensive to date that examined a potential association between fluoride
levels in bone and osteosarcoma. The results indicated no correlation. The study was the
second of a two-part study implemented through a 15-year collaboration between the Harvard
School of Dental Medicine and the three branches of the National Institutes of Health. The
National Cancer Institute (NCI) approved the design of the study, and the NCI, the National
Institute of Environmental Health Sciences (NIEHS), and the National Institute of Dental and
Craniofacial Research (NIDCR) provided funding for the research. The cases for this study were
recruited from existing cases between 1993 and 2000.

Cases for the first part of the study, published in 2006, were recruited from existing cases of
osteosarcoma between 1989 and 1992. It is important to note that the authors referred to the
study as an exploratory analysis which found an association between estimated fluoride
exposure in drinking water during childhood and the incidence of osteosarcoma among males
but not in females. However, the authors concluded that further research was necessary to
confirm or refute the observation. In addition, two Harvard University researchers who were
principal investigators of the larger Harvard study, issued a commentary in the same issue of
that journal urging readers to consider the results of the study with caution, stating that the
authors themselves were aware of additional findings from other study cases that did not appear
to replicate the findings.

Additionally, an earlier paper authored by the same team discussed problems with assessing
exposure of fluoride in drinking water for any specific individual over time. The article stated that
researchers need to be aware of the limitations that exist when using secondary data to
determine fluoride exposure estimates. Assessing exposure of fluoride in drinking water by
using secondary data can provide misleading or incomplete information. Yet is was this type of
data that was used to complete the 2006 study which reported to find an association between
fluoride and osteosarcoma.

Ruling by the Cancer Identification Committee of the California Office of Environmental
Health Hazard Assessment

In October 2011, the California Office of Environmental Health Hazard Assessment (OEHHA)
through its Cancer Identification Committee (CIC) determined that fluoride does not cause
cancer. The review was part of California’s Proposition 65 listing process.

Proposition 65 was enacted in 1986 with the intent to protect California citizens and the State’s
drinking water sources from chemicals known to cause cancer, birth defects or other
reproductive harm and to inform citizens about exposure to such chemicals. It requires the
Governor to publish, at least annually, a list of chemicals known to the state to cause cancer or
reproductive toxicity. The California Office of Environmental Health Hazard Assessment
(OEHHA) administers meetings of the Carcinogen Identification Committee (CIC) and the list of
items to be reviewed through the Proposition 65 process.

On May 29, 2009, fluoride was selected by OEHHA for review by the CIC. Due to widespread
exposure to fluoride, it was identified as one of five high priority chemicals to be evaluated. A
public comment period followed. On July 8, 2011, as the next step of the Proposition 65
process, the CIC released a hazard identification document, "Evidence on the Carcinogenicity of
Fluoride and its Salts". It was used by the CIC in its deliberations on whether fluoridogenicity should be
listed as a carcinogen under Proposition 65. A second public comment period followed.
At a public meeting on October 12, 2011, the CIC heard additional testimony and then voted on the question, "Do you believe that it has been clearly shown, through scientifically valid testing according to generally accepted principles, that fluoride causes cancer?" The CIC's vote was unanimous (6-0) that fluoride had not been clearly shown to cause cancer.31

European Union – Evaluation by the Scientific Committee on Health and Environmental Risks

A scientific evaluation of fluoridating agents of drinking water was done by the Scientific Committee on Health and Environmental Risks (SCHER) upon request by the European Commission (EC). The EC is the European Union's (EU) executive body with responsibility to manage EU policy. The Committee was asked to critically evaluate any new evidence on the hazard profile, health effects and human exposure to fluoride. The final report, Critical review of any new evidence on the hazard profile, health effects, and human exposure to fluoride and the fluoridating agents of drinking water was released in 2011.21 The report stated, "SCHER agrees that epidemiological studies do not indicate a clear link between fluoride in drinking water, and osteosarcoma and cancer in general. There is no evidence from animal studies to support the link, thus fluoride cannot be classified as carcinogenic."

Additional Studies

Despite the abundance of recent scientific evidence to the contrary, claims of a link between fluoridation and increased cancer rates continue. This assertion is largely based on a 1983 study comparing cancer death rates in ten large fluoridated cities versus ten large non-fluoridated cities in the United States. The results of this study have been refuted by a number of organizations and researchers.32

Scientists at the National Cancer Institute analyzed the same data and found that the original investigators failed to adjust their findings for variables, such as age and gender differences, that affect cancer rates. A review by other researchers pointed to further shortcomings in the study. The level of industrialization in the fluoridated cities was much higher than the non-fluoridated cities. Researchers noted that a higher level of industrialization is usually accompanied by a higher incidence of cancer. While the researchers noted that the fluoridated cities did have higher cancer rates over the twenty-year study, the rate of increase in the non-fluoridated cities was exactly the same (15%) as the fluoridated cities. Following further reviews of the study, the consensus of the scientific community continues to support the conclusion that the incidence of cancer is unrelated to the introduction and duration of water fluoridation.24

In a 1990 study, scientists at the National Cancer Institute evaluated the relationship between fluoridation of drinking water and cancer deaths in the United States during a 36-year period and the relationship between fluoridation and the cancer rate during a 15-year period. After examining more than 2.3 million cancer death records and 125,000 cancer case records in counties using fluoridated water, the researchers saw no indication of a cancer risk associated with fluoridated drinking water.24

In 2001, researchers from Japan analyzed data on cancers taken from the International Agency for Research on Cancer World Health Organization in 1987, 1992 and 1997 and concluded that fluoridation may increase the risk for numerous types of cancers.33 However, the methodology used in this analysis was inherently flawed as there are major and obvious differences in a
number of factors relevant to the risk for cancer in the fluoridated and non-fluoridated communities. For example, this analysis did not control for differences in urbanization, socioeconomic status, geographic region, occupations, industries, diet, medical practices or tobacco use between the fluoridated and non-fluoridated communities. Thus, any attempt to interpret cancer risk between these communities with this number of uncontrolled variables is scientifically inappropriate.

Finally, a ten-year (1943-1953) comparison study of long-time residents of Bartlett and Cameron, Texas, where the water supplies contained 8.0 and 0.4mg/L fluoride, respectively, included examinations of organs, bones and tissues. Other than a higher prevalence of dental fluorosis in the Bartlett residents, the study indicated that long term consumption of dietary fluoride (resident average length of fluoride exposure was 36.7 years), even at levels considerably higher than recommended for tooth decay prevention, resulted in no clinically significant physiological or functional effects. 34

References


Consider the following individuals with expertise related to fluoride.

Dr. Gary M. Whitford  
Professor  
Department of Oral Biology  
School of Dentistry  
Medical College of Georgia  
Augusta, GA  

Dr. Chester W. Douglass  
Professor of Oral Health Policy and Epidemiology, Emeritus  
Harvard Medical School  
Oral Health Policy & Epidemiology  
188 Longwood Ave  
Boston MA 02115  

Dr. Steven M. Levy  
Wright-Bush-Shreves Endowed Professor of Research  
Department of Preventive & Community Dentistry  
University of Iowa  
College of Dentistry  
Iowa City, IA  

Dr. Jayanth V. Kumar  
State Dental Director  
California Department of Public Health  
1616 Capitol Ave, Ste 74.459  
PO Box 997377  
Sacramento, CA 95899  

Dr. Howard Pollick  
Health Sciences Clinical Professor  
Department of Preventive and  
Restorative Dental Sciences  
School of Dentistry, UCSF  
707 Parnassus Avenue, Box 0758  
San Francisco, CA 94143-0758  

Dr. Robert N. Hoover  
Director  
Epidemiology and Biostatistics Program  
DHHS/NIH/NCI/DCEG/EBP  
Rockville MD 20850  

Dr. Marilia Afonso Rabelo Buzalaf  
Head, Department of Biological Sciences  
Bauru Dental School, University of Sao Paula  
Brazil  

Dr. Angeles Martinez Meir
Professor and Chair, Department of Cariology, Operative Dentistry and Dental Public Health
Director, Binational/Cross-Culture Health Enhancement Center
Indiana University School of Dentistry
Indianapolis, IN
June 30, 2011

Office of Pesticide Programs  
Docket No. EPA-HQ-OPP-2005-0174  
Regulatory Public Docket (7502P)  
Environmental Protection Agency  
1200 Pennsylvania Ave NW  
Washington, DC 20460-0001

To Whom It May Concern:

On behalf of our 157,000 members, we are pleased to comment on the *Fluoride: Dose-Response Analysis For Non-cancer Effects* and *Fluoride: Exposure and Relative Source Contribution Analysis* documents developed by the Environmental Protection Agency’s (EPA’s) Office of Water (OW). We offer these comments in response to your Federal Register notices of January 19, 2011 (76 FR 3422) and April 6, 2011 (76 FR 19001).

In sum, we hope you will give weight to the Butler et al. study in any future decisions regarding MCLG and MCL levels. The Butler study demonstrates fluorosis prevalence at a time when fluoride was available from a number of sources, unlike the Dean study when fluoride intake was limited to drinking water and diet. Additionally, the OW benchmark and Reference Dose should be re-evaluated as the calculations used required numerous assumptions and uncertainties associated with the contributions from sources other than drinking water.

The ADA strongly supports the U.S. Department of Health and Human Services recommendation to set the level for optimally fluoridated water at 0.7 parts per million. The decision is consistent with our longstanding position that dental fluorosis can be minimized – and tooth decay can be reduced – by adjusting the fluoride content in drinking water to an optimal level.

Enclosed you will find more detailed comments regarding these issues. We commend the EPA for the work that has been begun and look forward to the EPA’s continued effort to maintain the public’s safety while working with the HHS to extend the benefits of fluoride in drinking water. If you have any questions, please contact Mr. Robert J. Burns at 202-789-5176 or burnsr@ada.org.

Sincerely,

/s/ Raymond F. Gist, D.D.S.  
President

/s/ Kathleen T. O'Loughlin, D.M.D., M.P.H.  
Executive Director

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Comments on the
Environmental Protection Agency’s
Fluoride Risk Assessment and Relative Risk Contribution Documents
Docket Number: EPA-HQ-OPP-2005-0174

June 30, 2011

The American Dental Association (ADA) is pleased to comment on the Fluoride: Dose-Response Analysis For Non-cancer Effects and Fluoride: Exposure and Relative Source Contribution Analysis documents developed by the Environmental Protection Agency’s (EPA’s) Office of Water (OW). We offer these comments in response to docket number EPA-HQ-OPP-2005-0174, as published your Federal Register notices of January 19, 2011 (76 FR 3422) and April 6, 2011 (76 FR 19001).

The American Dental Association recommends the EPA should:

- Clearly state that using severe dental fluorosis as the clinical end point will protect against skeletal fluorosis and any other toxic effects.

As noted in the March 2006 National Research Council’s report, Fluoride in Drinking Water: A Scientific Review of EPA’s Standards, severe dental fluorosis occurs at a lower fluoride dose and over a shorter period of exposure time than does stage II skeletal fluorosis and/or bone fractures.

The ADA has long supported the secondary maximum contaminant level (SMCL) to protect against not only severe, but also moderate dental fluorosis.

The ADA notes with interest the increase in the prevalence of fluorosis as reported in the comparison of the CDC/NCHS National Health and Nutrition Examination Survey 1999-2004 and the National Institute of Dental Research, National Survey of Oral Health in U.S. Children, 1986-1987 outlined in the 2010 NCHS data brief, no 53. While both surveys are national in scope, there are important differences to be noted. There are other factors beside fluoride exposure that may aid in explaining the increase in fluorosis prevalence. The CDC report states that “Differences in study design between NIDR 1986-1987 and NHANES 1999-2002 should be considered when drawing inferences about changes in prevalence and severity of enamel fluorosis.”

As noted in the NCHS data brief, “In the analyses of changes in prevalence between both national surveys, moderate and severe dental fluorosis were aggregated into one category because all estimates of severe fluorosis were statistically unreliable after stratification (standard error of the percentage was greater than 30% the value of the percentage).” The apparently unreliable estimate of low prevalence of severe dental fluorosis and its combination with moderate dental fluorosis makes it virtually impossible to determine with scientific confidence whether the increase seen is in moderate or severe fluorosis.

There are also major differences in the sample designs. The 1986–1987 NIDR sample included a selection of school districts, schools, and classrooms. Because the 1986–1987 NIDR survey
was school-based, children not attending school were not part of the sampling frame. The 1999-2004 NHANES survey was a household survey that included oversampling of non-Hispanic black and Mexican-American subgroups. Since fluorosis reportedly is more severe in non-Hispanic blacks, this can be an important difference.

While examiner reliability was considered generally acceptable in these surveys, it appears there were a significantly larger number of examiners in the 1986-87 survey (14) than in either the 1999-2002 (4) or 2003-2004 NHANES survey (2). While examiners in the first two surveys were calibrated against a standard examiner, an interrater evaluation process was used in 2003-2004. These types of process changes, along with perhaps a secular change in societal awareness and, therefore, a possible “examiner shift,” regarding the desirability of the “perfect smile” may account for what appears to be a notable shift in prevalence away from “unaffected” and “questionable” to “very mild,” “mild” and “moderate and severe.” In fact, the reduction in “questionable” is almost completely reflected in the increase in “very mild.” Of additional interest is dramatic change in the prevalence of “normal” and “questionable fluorosis” between the 1999-2002 and 1999-2004 survey estimates.

See Figure 3 from the NCHS Data Brief, No. 53 that follows, noting the changes in dental fluorosis prevalence between the 1986-1987 and 1999-2004 national surveys.

Lastly, it is impossible to link fluorosis prevalence to types of fluoride exposures because neither survey reports any information related to fluoride histories of those examined. There is no information regarding years of residence in a fluoridated or non-fluoridated community or in a
community with naturally occurring fluoride over the MCL or MCLG. Nor is there information on use of topical fluoride products including toothpastes, dietary fluoride supplements or fluoride varnish. When the ADA first granted the seal to a fluoride toothpaste in 1964, the total market share of fluoride toothpaste was approximately 20%. By 1980, the market share had grown to 90% and today fluoride toothpaste is ubiquitous. By 1980, proportionately more young children were using fluoride toothpaste than in earlier times. Health care professionals did not widely stress the importance of limiting the amount of fluoride toothpaste used or the importance of monitoring children’s toothbrushing until the early mid 1990s. The results of those changes on the prevalence of fluorosis will not be fully seen in the adolescent cohort until after 2004. 

- **Use the Butler et al. study** from Texas for determining the point of departure (POD) for severe dental fluorosis and consider a fluoride level where severe dental fluorosis is observed consistently.

The ADA recognizes the EPA’s desire to locate and use a study from a time period when water was the major, if not only, significant source of fluoride. And we recognize the value of the Dean study from a number of perspectives. However, the choice of Dean’s study may be problematic due to uncertainties associated with the study (analytical testing method/lack of fluoride exposure histories/lack of cultural diversity of participants).

The ADA recommends that the EPA focus on a study already included in the list of the EPA document references. This research conducted in 1980-81 and supported by a grant from the EPA was conducted by Butler et al. in sixteen communities in Texas using 2,592 school-aged children from “Black, Spanish and White” families who were lifetime residents of their respective communities. This study shows a level of 3.3 mg/L as the point of departure (POD) for severe dental fluorosis.

In the Butler et al. study, the participants were exposed to multiple sources of fluoride. In the early 1980’s fluoride toothpaste was ubiquitous and there were no recommendations regarding limiting the amount of fluoride toothpaste to be used. Additionally in the early 1980s, infant formula manufactures had not yet voluntarily lowered the fluoride levels of infant formulas. In this study, few individuals used dietary fluoride supplements but many subjects had received professionally applied topical fluoride treatments. It would appear that the fluoride exposures and intakes for individuals may have been greater in these communities in Texas at the time of the study than it is today. The Butler et al. study provides a population threshold to establish the point of departure for severe fluorosis when other sources of fluoride are available. This eliminates the uncertainties associated with the estimates of fluoride ingested from sources other than drinking water.

Additionally, when compared to Dean’s study, the Butler et al. study was conducted when there were improved methods to analyze the levels of fluoride and other contaminants in drinking water. The Butler et al. study has well-documented fluoride histories and a cultural diverse group of study participants.

- **Use the 95th percentile exposure column in Table 5-4 (rather than the mean) to determine the Reference Dose (RfD) and revise the Office of Water benchmark.**

According to the EPA Dose Response Analysis document, the BMD is very close to the LOAEL of 2.2 mg/L for 0.7% severe dental fluorosis identified in the Dean (1942) study (see Table 4-1.) and the BMDL is only slightly below the NOAEL of 1.9 mg/L identified for the community of
Galesburg, IL. This suggests that children in Clovis, NM whose drinking water intake estimates were in the 95th percentile exposure column in Table 5-4., that follows, did not develop severe dental fluorosis.

*Table 5-4. Estimates of Fluoride Doses at Specific Tap Water Intakes for Age Groupings During the Sensitive Window for Development of Severe Enamel Fluorosis (at 1.87 mg F/L)

<table>
<thead>
<tr>
<th>Age Range (Years)</th>
<th>Fluoride Exposure (mg/kg/day)</th>
<th>Mean</th>
<th>75th Percentile</th>
<th>90th Percentile</th>
<th>95th Percentile</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.5 – 0.9,</td>
<td></td>
<td>0.07</td>
<td>0.10</td>
<td>0.14</td>
<td>0.16</td>
</tr>
<tr>
<td>1–3</td>
<td></td>
<td>0.09</td>
<td>0.10</td>
<td>0.15</td>
<td>0.19</td>
</tr>
<tr>
<td>4–6</td>
<td></td>
<td>0.07</td>
<td>0.09</td>
<td>0.12</td>
<td>0.14</td>
</tr>
<tr>
<td>7–10</td>
<td></td>
<td>0.05</td>
<td>0.06</td>
<td>0.08</td>
<td>0.10</td>
</tr>
<tr>
<td>11–14</td>
<td></td>
<td>0.04</td>
<td>0.05</td>
<td>0.06</td>
<td>0.08</td>
</tr>
</tbody>
</table>

*Dose estimates for infants may underestimate the actual doses because of the lack of reliable information on the type of formula used for bottle-fed infants.


- **Use the Benchmark Dose lower 95% bound (BMDL) approach based on the Butler et al. to developing the Maximum Contaminant Level Goal (MCLG) due to uncertainties with the RFD estimate.**

When conducting risk assessments involving exposures through drinking water, the BMDL of 1.87 mg/L should be used in place of the RfD as the appropriate point of departure for determination of the MCLG, as it does not include the uncertainties associated with assumptions used to calculate the RfD. The determination of fluoride intake computed from recall estimates of diet and toothpaste use weakens the RfD. We note that the EPA notes the confidence in the RfD derivation as “medium.”

- **Due to the uncertainties outlined in these documents, the EPA should conduct/support studies to obtain more accurate estimates of the prevalence of severe fluorosis at varying levels of fluoride in drinking water and fluoride intake (particularly from fluoride toothpaste). Currently, the population data are not consistent with the EPA model.**

Fluorosis studies indicated that fluorosis has increased in both fluoridated and non-fluoridated communities. However, moderate and severe fluorosis remain at low levels and appear to be somewhat isolated except in cases where children are subject to drinking water above 2 mg/L. Figure 4-1 below, shows the plot of more than 94 prevalence estimates conducted over a period spanning half a century. Despite a wide range of methodologies, fluorosis indices, fluoride measurement methods and population characteristics, a clear trend is evident. In communities with water fluoride concentrations below 2 mg/L, the prevalence of severe fluorosis is close to zero. Lowering the fluoride level in adjusted water systems alone may not appreciably affect the prevalence of severe dental fluorosis.
The EPA should also work to assist water systems with fluoride levels above the MCLG to come into compliance. While most are small systems, many systems across the country still provide water to consumers with levels above the MCLG and MCL.
References


