cards, letters, and flats; USPS Marketing Mail automation letters and flats; USPS Marketing Mail Carrier Route, High Density, and Saturation letters; Periodicals Outside County barcoded or Carrier Route letters and flats; Periodicals In-County automation or Carrier Route letters and flats; and Bound Printed Matter Presorted, non-DDU barcoded flats. Mailers who present at least 95 percent of their eligible First-Class Mail and USPS Marketing Mail volume as Full-Service in a calendar month would receive electronic address correction notices for their qualifying Basic automation and non-automation First-Class Mail and USPS Marketing Mail pieces, at the address correction fee for pieces eligible for the Full-Service Intelligent Mail option as described in DMM 705.23.0 for future billing cycles. The Basic First-Class Mail and USPS Marketing Mail mailpieces must:

1. Bear a unique IMb printed on the mailpiece;
2. Include a Full-Service or OneCode ACS STID in the IMb;
3. Include the unique IMb in eDoc;
4. Be sent by an eDoc submitter providing accurate Mail Owner identification in eDoc, and;
5. Be sent by an eDoc submitter maintaining 95 percent Full-Service compliance to remain eligible for this service and undergo periodic Postal Service re-evaluation.

* * * * *

4.2.8 Address Correction Service Fee

[Revise 507.4.2.8 by deleting the old language and replacing with new language as follows:]

ACS fees would be assessed as follows:

a. The applicable fee for address correction is charged for each separate notification of address correction or the reason for nondelivery provided, unless an exception applies.

b. Once the ACS fee charges have been invoiced, any unpaid fees for the prior invoice cycle (month) would be assessed an annual administrative fee of 10 percent for the overdue amount.

c. Mailers who present at least 95 percent of their eligible First-Class Mail and USPS Marketing Mail volume as Full-Service in a calendar month would receive electronic address correction notices for their qualifying Basic automation and non-automation First-Class Mail and USPS Marketing Mail mailpieces, as specified in 4.2.2. The electronic address correction notices are charged at the applicable Full-Service address correction fee for all future billing cycles.

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600 Basic Mailing Standards for All Mailing Services

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602 Addressing

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5.0 Move Update Standards

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[Revise 602.5.3 by deleting former contents and replacing with new title and contents as follows:]

5.3 Move Update Verification

Mailers who submit any Full-Service volume in a calendar month will be verified pursuant to the Address Quality Census Measurement and Assessment Process beginning in the next calendar month. First-Class Mail and USPS Marketing Mail letter and flat-size mailpieces with addresses that have not been updated in accordance with the Move Update Standard will be subject to the Move Update assessment charge, if submitted via eDoc with unique Basic or Full-Service IMbs. Supporting details are described in Publication 6850, *Publication for Streamlined Mail Acceptance for Letters and Flats,* available at *www.postalpro.usps.com.*

[Revise 602.5.4 as follows:]

5.4 Mailer Certification

The mailer's signature on the postage statement or electronic confirmation during eDoc submission certifies that the Move Update standard has been met for the address records including each address in the corresponding mailing presented to the USPS.

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700 Special Standards

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705 Advanced Preparation and Special Postage Payment Systems

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23.0 Full-Service Automation Option

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23.5 Additional Standards

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23.5.2 Address Correction Notices

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[Revise 705.23.5.2a as follows:]

a. Address correction notices would be provided at the applicable Full-Service address correction fee for letters and flats eligible for the Full-Service option, except for USPS Marketing Mail ECR flats, BPM flats dropshipped to DDUs, or BPM carrier route flats. Mailers who present at least 95 percent of their eligible First-Class Mail and USPS Marketing Mail volume as Full-Service in a calendar month would receive electronic address correction notices for their qualifying Basic automation and non-automation First-Class Mail and USPS Marketing mailpieces charged at the applicable Full-Service address correction fee for future billing cycles. The Basic automation and non-automation First-Class Mail and USPS Marketing Mail mailpieces must:

1. Bear a unique IMb printed on the mailpiece.
2. Include a Full-Service or OneCode ACS STID in the IMb.
3. Include the unique IMb in eDoc.
4. Be sent by an eDoc submitter providing accurate Mail Owner identification in eDoc.
5. Be sent by an eDoc submitter maintaining 95 percent Full-Service compliance to remain eligible for this service and undergo periodic USPS re-evaluation.

* * * * *

We will publish an appropriate amendment to 39 CFR part 111 to reflect these changes, if our proposal is adopted.

Stanley F. Mires,

Attorney, Federal Compliance.

[FR Doc. 2017–03723 Filed 2–24–17; 8:45 am]

BILLING CODE 7710–12–P

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Chapter I

[EPA–HQ–OPPT–2016–0763; FRL–9959–74]

Fluoride Chemicals in Drinking Water; TSCA Section 21 Petition; Reasons for Agency Response

AGENCY: Environmental Protection Agency (EPA).

ACTION: Petition; reasons for Agency response.

SUMMARY: This document announces the availability of EPA's response to a petition it received on November 23, 2016, under section 21 of the Toxic Substances Control Act (TSCA). The TSCA section 21 petition was received from the Fluoride Action Network, Food & Water Watch, Organic Consumers Association, the American Academy of Environmental Medicine, the International Academy of Oral Medicine and Toxicology, and other individual petitioners. The TSCA section 21 petition requested that EPA exercise its authority under TSCA section 6 to ''prohibit the purposeful addition of fluoridation chemicals to U.S. water supplies.'' After careful consideration,

EPA has denied the TSCA section 21 petition for the reasons discussed in this document.

DATES: EPA's response to this TSCA section 21 petition was signed February 17, 2017.

FOR FURTHER INFORMATION CONTACT:

For technical information contact: Darlene Leonard, National Program Chemicals Division (7404T), Office of Pollution Prevention and Toxics, Environmental Protection Agency, 1200 Pennsylvania Ave. NW., Washington, DC 20460–0001; telephone number: (202) 566–0516; fax number: (202) 566–0470; email address: *leonard.darlene@epa.gov.*

For general information contact: The TSCA-Hotline, ABVI-Goodwill, 422 South Clinton Ave., Rochester, NY 14620; telephone number: (202) 554–1404; email address: *TSCA-Hotline@epa.gov.*

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this action apply to me?

This action is directed to the public in general. This action may, however, be of interest to individuals or organizations interested in drinking water and drinking water additives, including fluoride. Since other entities may also be interested, the Agency has not attempted to describe all the specific entities that may be affected by this action.

B. How can I access information about this petition?

The docket for this TSCA section 21 petition, identified by docket identification (ID) number EPA–HQ–OPPT–2016–0763, is available online at *http://www.regulations.gov* or in person at the Office of Pollution Prevention and Toxics Docket (OPPT Docket), Environmental Protection Agency Docket Center (EPA/DC), EPA West Bldg., Rm. 3334, 1301 Constitution Ave. NW., Washington, DC. Six binders containing copies of references were submitted along with the petition (Ref. 1). Those binders are not available electronically in the docket but may be reviewed in the Public Reading Room. The Public Reading Room is open from 8:30 a.m. to 4:30 p.m., Monday through Friday, excluding legal holidays. The telephone number for the Public Reading Room is (202) 566–1744, and the telephone number for the OPPT Docket is (202) 566–0280. Please review the visitor instructions and additional information about the docket available at *http://www.epa.gov/dockets.*

II. TSCA Section 21

A. What is a TSCA section 21 petition?

Under TSCA section 21 (15 U.S.C. 2620), any person can petition EPA to initiate a rulemaking proceeding for the issuance, amendment, or repeal of a rule under TSCA sections 4, 6, or 8 or an order under TSCA sections 4, 5(e), or 5(f). A TSCA section 21 petition must set forth the facts that are claimed to establish the necessity for the action requested. EPA is required to grant or deny the petition within 90 days of its filing. If EPA grants the petition, the Agency must promptly commence an appropriate proceeding that is ''in accordance'' with the underlying TSCA authority. If EPA denies the petition, the Agency must publish its reasons for the denial in the **Federal Register**. 15 U.S.C. 2620(b)(3). A petitioner may commence a civil action in a U.S. district court to compel initiation of the requested rulemaking proceeding within 60 days of either a denial or the expiration of the 90-day period. 15 U.S.C. 2620(b)(4).

B. What criteria apply to a decision on a TSCA section 21 petition?

TSCA section 21(b)(1) requires that the petition ''set forth the facts which it is claimed establish that it is necessary'' to issue the rule or order requested. 15 U.S.C. 2620(b)(1). Thus, TSCA section 21 implicitly incorporates the statutory standards that apply to the requested action. In addition, TSCA section 21 establishes standards a court must use to decide whether to order EPA to initiate rulemaking in the event of a lawsuit filed by the petitioner after denial of a TSCA section 21 petition. 15 U.S.C. 2620(b)(4)(B). Accordingly, EPA has relied on the standards in TSCA section 21 (and those in the provisions under which action has been requested) to evaluate this TSCA section 21 petition.

III. TSCA Section 6

Of particular relevance to this TSCA section 21 petition are the legal standards regarding TSCA section 6(a) rules. These standards were significantly altered in 2016 by the ''Frank R. Lautenberg Chemical Safety for the 21st Century Act,'' Public Law 114–182 (2016), which amended TSCA. One of the key features of the new law is the requirement that EPA now systematically prioritize and assess existing chemicals, and manage identified risks. Through a combination of new authorities, a risk-based safety standard, mandatory deadlines for action, and minimum throughput requirements, TSCA effectively creates a ''pipeline'' by which EPA will conduct review and management of existing chemicals. This new pipeline—from prioritization to risk evaluation to risk management (when warranted)—is intended to drive forward steady progress on the backlog of existing chemical substances left largely unaddressed by the original law. (Ref. 2).

In the initial phase of the review pipeline, EPA is to screen a chemical substance for its priority status, propose a designation as either high or low priority, and then issue a final priority designation within one year of starting the screening process. 15 U.S.C. 2605(b)(1)(C). If the substance is high priority, EPA must initiate a risk evaluation for that substance. 15 U.S.C. 2605(b)(4)(C). EPA must define the scope of the risk evaluation within six months of starting, 15 U.S.C. 2605(b)(4)(D), and complete the risk evaluation within 3 to 3.5 years. 15 U.S.C. 2605(b)(4)(G). If EPA concludes that a chemical substance presents an unreasonable risk, EPA must propose a risk management rule under TSCA section 6(a) within one year and finalize that rule after another year, with limited provision for extension. 15 U.S.C. 2605(c). As EPA completes risk evaluations, EPA is to designate replacement high-priority substances, on a continuing basis. 15 U.S.C. 2605(b)(2)(C) and (b)(3)(C).

In general, to promulgate a rule under TSCA section 6(a), EPA must first determine ''in accordance with section 6(b)(4)(A) that the manufacture, processing, distribution in commerce, use, or disposal of a chemical substance or mixture . . . presents an unreasonable risk.'' 15 U.S.C. 2605(a). TSCA section (b)(4)(A) is part of the risk evaluation process whereby EPA must determine ''whether a chemical substance presents an unreasonable risk of injury to health or the environment,'' and thus, whether a rule under TSCA section 6(a) is necessary. 15 U.S.C. 2605(b)(4)(A). In particular, EPA must conduct this evaluation ''without consideration of costs or other non-risk factors, including an unreasonable risk to a potentially exposed or susceptible subpopulation identified as relevant to the risk evaluation by the Administrator, under the conditions of use.'' Id. Unless EPA establishes an exemption under TSCA section 6(g) (whereby certain unreasonable risks may be allowed to persist for a limited period) or EPA is addressing a persistent, bioaccumulative, and toxic substance as set forth in TSCA section 6(h), the standard for an adequate rule under TSCA section 6(a) is that it regulates ''so that the chemical

substance or mixture no longer presents'' unreasonable risks under the conditions of use. 15 U.S.C. 2605(a).

Prior to the 2016 amendment of TSCA, EPA completed risk assessments that were limited to selected uses of chemical substances. The amended TSCA authorizes EPA to issue TSCA section 6 rules that are not comprehensive of the conditions of use, so long as they are consistent with the scope of such pre-amendment risk assessments. 15 U.S.C. 2625(l)(4). But EPA has interpreted the amended TSCA as requiring that forthcoming risk evaluations encompass all manufacture, processing, distribution in commerce, use, and disposal activities that the Administrator determines are intended, known or reasonably foreseen. (Ref. 2, p. 7565). EPA interprets the scope of post-risk-evaluation rulemaking under TSCA section 6(a) in a parallel fashion: While risk management rules for a certain subset of the conditions of use may be promulgated ahead of rulemaking for the remaining conditions of use, rules covering the complete set of conditions of use must be promulgated by the deadlines specified in TSCA section 6(c). 15 U.S.C. 2605(c). While EPA has authority under TSCA section 6(a) to establish requirements that apply only to ''a particular use,'' the restriction of just one particular use would not constitute an adequate risk management rule unless that particular use were the only reason that the chemical substance presented an unreasonable risk.

TSCA section 21(b)(4)(B) provides the standard for judicial review should EPA deny a request for rulemaking under TSCA section 6(a): ''If the petitioner demonstrates to the satisfaction of the court by a preponderance of the evidence that . . . the chemical substance or mixture to be subject to such rule . . . presents an unreasonable risk of injury to health or the environment, without consideration of costs or other non-risk factors, including an unreasonable risk to a potentially exposed or susceptible subpopulation, under the conditions of use,'' the court shall order the EPA Administrator to initiate the requested action. 15 U.S.C. 2620(b)(4)(B). EPA notes that bills preceding the final amendment to TSCA retained language in section 21 that resembled the pre-amendment criteria for rulemaking under section 6. Compare 15 U.S.C. 2620(b)(4)(B)(ii) (2015) (amended 2016), 15 U.S.C. 2605(a) (2015) (amended 2016), S. Rep. 114–67 at 135 (Ref. 3), and H.R. Rep. No. 114–176 at 81 (Ref. 4). But the effect of the revision in the final bill is to align the standard for judicial review of a TSCA section 21 petition with the standard for EPA's preparation of risk evaluation under TSCA section 6(b)(4)(A). Consistent with these revisions, EPA concludes that Congress intended for a petition to set forth facts that would enable EPA to complete a risk evaluation under TSCA section 6(b).

In light of this, EPA interprets TSCA section 21 as requiring the petition to present a scientific basis for action that is reasonably comparable, in its quality and scope, to a risk evaluation under TSCA section 6(b). This requirement includes addressing the full set of conditions of use for a chemical substance and thereby describing an adequate rule under TSCA section 6(a)—one that would reduce the risks of the chemical substance ''so that the chemical substance or mixture no longer presents'' unreasonable risks under all conditions of use. 15 U.S.C. 2605(a). Specifically, EPA interprets section 21(a)—which authorizes petitions ''to initiate a proceeding for the issuance . . . of a rule under . . . section 6''—as authorizing petitions for rules that *would comply with the requirements of* sections 6(a) and 6(c).

EPA recognizes that information on a single condition of use could, in certain instances, suffice to demonstrate that a chemical substance, as a whole, presents an unreasonable risk. Nonetheless, EPA concludes that such information does not fulfill a petitioner's burden to justify ''a rule under [TSCA section 6],'' under TSCA section 21, since the information would merely justify a subset of an adequate rule. To issue an adequate rule under section 6, EPA would need to conduct a catch-up risk evaluation addressing all the conditions of use not addressed by the petition, and either determine that those conditions do not contribute to the unreasonable risk or enlarge the scope of the rule to address those further conditions of use. See 15 U.S.C. 2605(a). To issue this rule within the time required by section 6(c), EPA would have to proceed without the benefit of the combined 4 to 4.5-year period that TSCA section 6(b) would ordinarily afford EPA (*i.e.,* time to prioritize a chemical substance, conduct a careful review of all of its conditions of use, and receive the benefit of concurrent public comment). Additionally, before even initiating the prioritization process for a chemical substance, EPA would generally screen the chemical substance to determine whether the available hazard and exposure-related information are sufficient to allow EPA to complete both the prioritization and the risk evaluation processes. (Ref. 5).

EPA's interpretation is most consonant with the review pipeline established in TSCA section 6. In particular, the prioritization process established in section 6(b) recognizes that a number of chemical substances may present an unreasonable risk of injury to health or the environment and charges EPA with prioritizing those that should be addressed first. EPA is required to have 10 chemical substances undergoing risk evaluation as of December 19, 2016, and must have a steady state of at least 20 high-priority substances undergoing risk evaluation by December 2019 (and as many as 10 substances nominated for risk evaluation by manufacturers). 15 U.S.C. 2605(b)(2)(A), (B), 2605(b)(4)(E)(i). EPA is obligated to complete rulemakings to address any unreasonable risks identified in these risk evaluations within prescribed timeframes. 15 U.S.C. 2605(c)(1). These required activities will place considerable demands on EPA resources. Indeed, Congress carefully tailored the mandatory throughput requirements of TSCA section 6, based on its recognition of the limitations of EPA's capacity and resources, notwithstanding the sizeable number of chemical substances that will ultimately require review. Under this scheme, EPA does not believe that Congress intended to empower petitioners to promote chemicals of particular concern to them above other chemicals that may well present greater overall risk, and force completion of expedited risk evaluations and rulemakings on those chemicals, based on risks arising from individual uses.

EPA recognizes that some members of the public may have safety concerns that are limited to a single condition of use for a chemical substance. But EPA's interpretation of TSCA section 21 does not deprive such persons of a meaningful opportunity to request that the Administrator proceed on their concerns. For example, such persons may submit a petition under the Administrative Procedure Act, requesting EPA to commence a ''risk-based screening'' of the chemical substance under TSCA section 6(b)(1)(A), motivated by their concern about a single condition of use.

IV. Summary of the TSCA Section 21 Petition

A. What action was requested?

On November 23, 2016, a TSCA section 21 petition was submitted by the Fluoride Action Network, Food & Water Watch, Organic Consumers Association, the American Academy of Environmental Medicine, the

International Academy of Oral Medicine and Toxicology, Moms Against Fluoridation, and the following individuals signing on behalf of themselves and their children: Audrey Adams of Renton, Washington, Jacqueline Denton of Asheville, North Carolina, Valerie Green of Silver Spring, Maryland, Kristin Lavelle of Berkeley, California, and Brenda Staudenmaier of Green Bay, Wisconsin (Ref. 1). The general object of the petition is to urge EPA ''to protect the public and susceptible subpopulations from the neurotoxic risks of fluoride by banning the addition of fluoridation chemicals to water'' (Ref. 1). The specific action sought is a rule, under TSCA section 6(a)(2), to ''prohibit the purposeful addition of fluoridation chemicals to U.S. water supplies.'' However, such a restriction on the allowable use of fluoridation chemicals would actually be based on a rule under TSCA section 6(a)(5), not a rule under TSCA section 6(a)(2). In light of the discrepancy between the description of the rule sought and the cited authority, EPA interprets the petition as requesting *both* a TSCA section 6(a)(5) rule whereby the purposeful addition of any fluoridation chemical to a drinking water supply would be prohibited *and* a TSCA section 6(a)(2) rule whereby the manufacture, processing, or distribution in commerce of any fluoridation chemical for such use would be prohibited.

B. What support does the petition offer?

The petition is focused on the potential for fluoride to have neurotoxic effects on humans; it cites numerous studies bearing on this issue. The petition contends that the purposeful fluoridation of drinking water presents an unreasonable risk to human health from neurotoxicity, and that a ban on this use of fluoridation chemicals is necessary to curtail this unreasonable risk. The following is a summary of the primary support given in the petition for this view:

1. *Fluoride neurotoxicity at levels relevant to U.S. population.* The petition claims that fluoride poses neurotoxic risks to the U.S. population. The petition claims that the cited studies of fluoride-exposed human populations have consistently found neurotoxic effects (lower-than-average IQs) at water fluoride levels below the current Maximum Contaminant Level Goal of 4 mg/L set by EPA's Office of Water. The petition argues that the difference between the fluoride levels in the United States and the greater levels in rural China (where most of the cited IQ studies were conducted) is ''lessen[ed]'' by the abundance of fluoridated toothpaste in the U.S.

2. *Recent epidemiological studies corroborate neurotoxic risk in Western populations.* The petition cites two studies from Western populations to attempt to corroborate the assertion that exposure to fluoride in drinking water presents unreasonable risks for neurotoxicity (Refs. 6 and 7).

3. *Neurotoxic risks supported by animal and cell studies.* The petition argues that studies on both experimental animals and cell cultures are consistent with cited human research linking fluoride exposure with neurotoxic effects in humans.

4. *Susceptible subpopulations are at heightened risk.* The petition argues that certain subpopulations (*e.g.,* infants, the elderly, and persons with nutritional deficiencies, kidney disease or certain genetic predispositions) are more susceptible to fluoride neurotoxicity.

5. *RfD/RfC derivation and uncertainty factor application.* The petition argues that EPA's 1998 *Guidelines for Neurotoxicity Risk Assessment* support the need to apply a 10-fold uncertainty factor in deriving an oral Reference Dose (RfD) or inhalation Reference Concentration (RfC).

6. *Benefits to public health.* The petition bases, in part, its claim of unreasonable risk on the assertion that the fluoridation of drinking water confers little benefit to public health, relative to the alleged neurotoxic risks. The petition argues that since fluoride's primary benefit comes from topical contact with the teeth, there is little benefit from swallowing fluoride, in water or any other product. The petition argues that there is therefore ''little justification'' in exposing the public to ''*any* risk'' of fluoride neurotoxicity.

7. *Extent and magnitude of risk from fluoridation chemicals.* The petition bases, in part, its claim of unreasonable risk on estimates of the extent and magnitude of risk posed to portions of the U.S. population living in areas where artificial fluoridation occurs.

8. *Consequences of eliminating use of fluoridation chemicals.* The petition argues that the risks of fluoride exposure from fluoridated drinking water are unreasonable, in part, because they could be easily and cheaply eliminated, and because alternative products containing topical fluoride are widely available.

9. *Link to elevated blood lead levels.* The petition argues that artificial fluoridation chemicals are linked with pipe corrosion and elevated blood lead levels. The petition interprets data in several studies as demonstrating an association between fluoridation chemicals and elevated blood lead levels.

In addition to supplying the petition, on January 30, 2017, the petitioners also delivered an in-person oral presentation of their views (Ref. 8). At their oral presentation, petitioners reiterated the information already supplied in writing, and requested that EPA also consider an additional study that was not part of the petition (Ref. 9). EPA has discretion (but not an obligation) to consider extra-petition materials when evaluating a petition submitted under TSCA section 21. In cases where the petitioners themselves attempt to enlarge the scope of materials under review while EPA's petition review is pending, EPA exercises its discretion to consider or not consider the additional material based on whether the material was submitted early enough in EPA's petition review process to allow adequate evaluation of the study prior to the petition deadline, the relation of the late materials to materials already submitted. Given the particularly late submittal of the additional study, EPA conducted an abbreviated review of the study and found that the health concerns covered were substantially the same as those covered in other studies submitted with the petition. Based on this abbreviated review, EPA does not believe that the new study provided any new scientific grounds for granting the petition.

V. Disposition of TSCA Section 21 Petition

A. What was EPA's response?

After careful consideration, EPA denied the TSCA section 21 petition, primarily because EPA concluded that the petition has not set forth a scientifically defensible basis to conclude that any persons have suffered neurotoxic harm as a result of exposure to fluoride in the U.S. through the purposeful addition of fluoridation chemicals to drinking water or otherwise from fluoride exposure in the U.S. In judging the sufficiency of the petition, EPA considered whether the petition set forth facts that would enable EPA to complete a risk evaluation under TSCA section 6(b).

EPA also denied the petition on the independent grounds that the petition neither justified the regulation of fluoridation chemicals as a category, nor identified an adequate section 6 rule as the action sought. Rather than comprehensively addressing the conditions of use that apply to a particular chemical substance, the petition requests EPA to take action on a single condition of use (water

fluoridation) that cuts across a category of chemical substances (fluoridation chemicals). A copy of the Agency's response, which consists of a letter to the petitioners, is available in the docket for this TSCA section 21 petition.

B. What were EPA's reasons for this response?

To take the actions under TSCA section 6 requested by the petitioners, EPA would need to make a determination of whether a chemical substance or substances present an unreasonable risk to human health or the environment. This section describes why the petitioners have not provided adequate and sufficient scientific information to make such a determination.

1. *Fluoride neurotoxicity at levels relevant to U.S. population.* The petition ignores a number of basic data quality issues associated with the human studies it relies upon. Many of the human studies cited in the petition are cross-sectional in design, and are affected by antecedent-consequent bias. The antecedent-consequent bias means it cannot be determined whether the exposure came before or after the health effects, since both are evaluated at the same time. Cross-sectional studies are most useful for developing hypotheses about possible causal relationships between an exposure and a health effect, but are rarely suitable for the development of a dose-response relationship for risk assessment. These studies are most useful in supporting more robust epidemiological studies in which defined exposures can be linked quantitatively to an adverse outcome.

The petition also does not properly account for the relatively poor quality of the exposure and effects data in the cited human studies (*e.g.,* it appears to give all studies equivalent weight, regardless of their quality). When an association is suggested between an exposure and a disease outcome, the studies need to be assessed to determine whether the effect is truly because of exposure or if alternate explanations are possible. The way to do that is to adjust for potential confounders, such as diet, behavior, and socioeconomic status, in order to appropriately assess the real relationship between the exposures to a specific substance and health effects. In other words, when these confounding factors are potentially present, but not recognized or controlled for, it is not possible to attribute effects to the contaminant of concern (fluoride) as opposed to other factors or exposures. The evidence presented did not enable EPA to determine whether various confounding factors (*e.g.,* nutritional deficiencies) were indeed placing particular subpopulations at a ''heightened risk of fluoride neurotoxicity,'' as alleged, because the evidence did not adequately account for the possibility that the *confounding factors themselves,* rather than concurrent fluoride exposure, were partly or wholly responsible for the health effects observed. Specific confounding factors or variables were noted by the National Research Council (NRC) (Ref. 10). They may include climate, drinking water intake, excessive dietary fluoride, low calcium intake, drinking water sources with fluctuating fluoride levels, and industrial pollution such as use of coal for domestic heating. These factors have the potential to confound efforts to identify a causal relationship between drinking water fluoride exposure and particular health effects, either by introducing additional, unaccounted for sources of fluoride exposure, by being associated with the pertinent health endpoint through some mechanism other than fluoride toxicity, or by directly affecting the health endpoint.

The petition relies heavily on two meta-analyses which include human cross-sectional (Ref. 11) and case control (Ref. 19) studies. All of the studies listed in Table 1 of the petition were examined in detail by the 2012 Choi et al. study (Ref. 11) as part of their systematic review and meta-analysis to investigate the possibility that fluoride exposure delays neurodevelopment in children. The Choi *et al.* analysis analyzes studies in which IQ was measured using various IQ tests, compares children of various fluoride exposure ranges without accounting for differences in susceptibility to fluoride by age, and used different exposure measures which only delineated between high and low exposure groups. A variety of measures of fluoride exposure were present across studies included in the Choi et al. study, including levels of fluoride in drinking water, observed dental fluorosis, coal burning in houses (*i.e.,* air fluoride levels), and urine fluoride. Despite this disparate collection of types of measurements, all exposure measures were treated equally in the analysis (Ref. 11, Table 1). The authors of the analysis identified a variety of data quality issues associated with this collection of studies. For example, they recognized that several of the populations studied had fluoride exposures from sources other than drinking water (*e.g.,* coal burning; Refs. 13–15); they therefore controlled for this confounding factor by excluding such studies from their analysis. Co-exposures to other potentially neurotoxic chemicals (*e.g.,* iodine) (Refs. 16–18) and arsenic (Refs. 19–22) were also recognized and accounted for in the Choi et al. analysis to understand confounding by these factors. Yet the petitioners include such studies in making their assertion that fluoride is neurotoxic, but have not indicated any attempts to control for the confounding factors. Choi et al. also noted that basic information such as the study subjects' sex and parental education was missing in 80 percent of the studies and household income was missing in 93 percent of studies; they stated that they could not therefore control for these co-variables in their analysis. Consideration of these confounding factors and their impact on the applicability of these studies in a risk assessment context is evident in the authors' discussion. The authors caution readers that ''our review cannot be used to derive an exposure limit, because the actual exposures of the individual children are not known'' and they are measured in their conclusions (*i.e.,* ''our results support the possibility of adverse effects of fluoride exposures on children's neurodevelopment'') (Ref. 11). The authors indicate that ''further research should formally evaluate dose-response relationships based on individual-level measures of exposure over time, including more precise prenatal exposure assessment and more extensive standardized measures of neurobehavioral performance, in addition to improving assessment and control of potential confounders'' (Ref. 11). EPA agrees with the conclusions by Choi et al. (Ref. 11) that the studies included in Table 1 of the petition are unsuitable for evaluating levels of fluoride associated with neurotoxic effects and for deriving dose-response relationships necessary for risk assessment.

The petition also cites an article by Grandjean and Landrigan (Ref. 23), for the proposition that fluoride is ''known'' to cause developmental neurotoxicity in humans. Grandjean and Landrigan refer only to the study of Choi et al. (2012), of which Grandjean is a co-author, in discussing fluoride. EPA's observations about the limitations of Choi et al. (2012) thus apply with equal force to the cited statement from Grandjean and Landrigan. Grandjean and Landrigan summarize that Choi et al. (2012) ''*suggests* an average IQ decrement of about seven points in children exposed to raised fluoride concentrations.'' (Ref. 23). But Grandjean and Landrigan do not opine on whether fluoride exposures, arising from the purposeful addition of fluoridation chemicals to

U.S. water supplies, are in fact causing developmental neurotoxic effects to persons in the U.S. The petition itself concedes that the actual existence of such effects is unestablished, in urging EPA to conduct ''a diligent risk assessment, per EPA's *Guidelines,* to ensure that the general public, and sensitive subpopulations, are not ingesting neurotoxic levels'' (Ref 1, p. 3).

The other meta-analysis cited in the petition (Ref. 12) showed that, based on 16 case-control studies in China, children living in an area with endemic fluorosis are more likely to have low IQ compared to children living in an area with slight fluorosis or no fluorosis. While this analysis may suggest an association between fluorosis and lowered IQ (both of which are possible effects of fluoride exposure at certain levels) any fluoride concentration-to-IQ effect relationship (*i.e.,* dose-response relationship) is only inferred because actual fluoride exposures were not measured. Further, the two effects (fluorosis and lower IQ) both occur at fluoride exposures well above those found in fluoridated U.S. drinking water, such that any inference would only apply at fluoride concentrations not relevant to exposures in the U.S. The studies in the Tang et al. review (Ref. 12) correlate one effect (fluorosis) to another effect (neurotoxicity), but do not establish a dose-response relationship between fluoride exposure and neurotoxicity. This lack of a dose-dependent increase in effect with increasing exposure is a critical limitation of these data. Establishing a dose-response relationship between exposure to a toxicant and an effect ''is the most fundamental and pervasive concept in toxicology. Indeed, an understanding of this relationship is essential for the study of toxic materials'' (Ref. 12). Likewise, the IQ changes noted in Table 1 (Ref. 1) do not increase with increasing water fluoride concentration (*e.g.,* dose) (Ref. 1).

The petition suggested that a dose-response relationship between urinary fluoride and IQ is seen in several studies (Refs. 24–26) shown in Figures 1–5 of the petition (Ref. 1). Assuming, as the petitioners claim, that all children were malnourished in the Das and Mondal (Ref. 26) study, it is not possible to determine whether effects on IQ were due to fluoride or to malnutrition (*i.e.,* nutritional status may be an uncontrolled confounding factor). The study authors caution that ''it is difficult to determine with any degree of accuracy whether the difference of children's IQ scores solely depends on the exposure dose because many social and natural factors like economic condition, culture and geological environments are also responsible'' (Ref. 26). Hence, extrapolating relationships from this study population to other populations is not scientifically defensible.

Choi et al. (2015) (Ref. 27) report that moderate and severe dental fluorosis was significantly associated with lower cognitive functions. However, associations between drinking water and urine fluoride and the same cognitive functions were not found to be significantly associated. They reached this conclusion from a study of 51 children in China and a comparison group of eight with dental fluorosis (Table 4 in Choi et al., 2015). The authors discuss potential problems associated with using these biomarkers of exposure to fluoride. For example, water samples may be imprecise because internal dose of fluoride depends on total water intake, and urine samples may be affected by the amount of water the subject drank prior to sampling. With regard to fluorosis, the degree of dental fluorosis is dependent not only on the total fluoride dose but also on the timing and duration of fluoride exposure. A person's individual response to fluoride exposure depends on factors such as body weight, activity level, nutritional factors, and the rate of skeletal growth and remodeling. These variables, along with inter-individual variability in response to similar doses of fluoride, indicate that enamel fluorosis cannot be used as a biological marker of the level of fluoride exposure for an individual (Ref. 28). Hence, the petitioner's use of fluorosis levels as a surrogate for evidence of neurotoxic harm to the U.S. population is inappropriate evidence to support an assertion of unreasonable risk to humans from fluoridation of drinking water.

The petition also cites four studies (Refs. 24, 29–31) that rely on human urine or serum fluoride concentrations as biomarkers of exposure but does not discuss the limitations associated with the biomarkers used in the studies. In their report, *Human Biomonitoring for Environmental Chemicals,* NRC defines properties of biomarkers and created a framework for grouping biomarkers of exposure (Ref. 32). Figure 3–1 in the NRC report illustrates the relationship between external dose (*e.g.,* water), internal dose (*e.g.,* fluoride concentration) and biological effects, and indicates that internal dose is measured through biomonitoring (*e.g.,* fluoride concentrations measured in urine or serum). NRC grouped the quality of biomarkers based on the robustness of these relationships. NRC designated biomarkers for substances that have been observed in bodily fluids, but that lack established relationships between external dose (*e.g.,* water), internal dose (*e.g.,* urine or serum) and biological effects (*e.g.,* neurotoxicity) as ''Group I'' biomarkers. Although many human studies have been collated and reviewed in the petition, for the reasons outlined previously—particularly study design and confounding factors—relationships between urine and serum fluoride (internal doses), water fluoride concentration (external dose), and neurotoxic effects in humans have not been established. Further, serum and urine biomarkers for fluoride reflect only recent exposures, not long-term exposures, and may be different from the exposures during the specific time when developmental effects can occur. A lack of established sampling protocols and analytical methods are also hallmarks of ''Group I'' biomarkers. The main studies cited in the petition which attempt to relate urine or serum levels to possible neurotoxic effects suffer from either lack of good sampling protocols or absence of documenting the sampling protocols. Important issues such as the timing and methods of sample collection were also often not reported in the studies. Using the NRC Framework, urine and serum fluoride levels would be at best ''Group I'' biomarkers for fluoride-related neurotoxicity. The NRC Framework states ''[b]iomarkers in this category may be considered useless'' for risk assessment purposes (Ref. 32, p. 78).

2. *Recent epidemiological studies corroborate neurotoxic risk in Western populations.* The petition cites two studies from Western populations to attempt to corroborate the assertion that exposure to fluoridated water presents unreasonable risks for neurotoxicity. Two population-level studies were cited which link fluoridated water to attention-deficit/hyperactivity disorder (ADHD) prevalence in the U.S. (Ref. 6) and drinking water exposures and hypothyroidism prevalence in England (Ref. 7). These studies use cross-sectional population-level data to examine the association between ADHD and hypothyroidism and fluoridated water levels. The studies make reasonable use the population-level data available, but causal inference cannot be made from these studies (Ref. 3).

As stated in the conclusion of Malin and Till, an association has been reported, but ''[p]opulation studies designed to examine possible mechanisms, patterns and levels of exposure, covariates and moderators of

this relationship are warranted'' (Ref. 6, p. 8). In epidemiology, studies using cross-sectional data are most often used to generate hypotheses that need to be further studied to determine whether a ''true'' association is present. Ideally, the study designs and methods are improved by each study that is undertaken, such as, among other things, identifying additional potential confounders, considering timing issues or resolving ambiguity in collection of samples and disease outcome, improving upon the exposure analysis, and evaluating the magnitude and consistency of the results, so that the evaluation can adequately assess the association (Ref. 34). For example, the authors assert that there are design issues with their study, especially related to the exposure categories, and they suggest how to address these issues in future studies. Although it is possible that there may be biological plausibility for the hypothesis that water fluoridation may be associated with ADHD, this single epidemiological study is not sufficient to ''corroborate'' neurotoxic health effects, as stated in the petition. More study would be needed to develop a body of information adequate to make a scientifically defensible unreasonable risk determination under TSCA.

The Peckham et al. study (Ref. 7) suffers from similar issues noted in Malin and Till (Ref. 6). Adjustment for some confounders was considered, including sex and age, but other potential confounders (such as iodine intake) were not assessed. Fluoride from other sources and other factors associated with hypothyroidism were not assessed in this study. Exposure misclassification, in which populations are placed in the wrong exposure categories based on the water fluoridation status, is very possible in either of the studies presented and is a limitation of the study designs.

3. *Neurotoxic risks supported by animal and cell studies.* The National Toxicology Program (NTP) conducted a systematic review of animal and cell studies on the effects of fluoride on learning and memory available up to January 2016 (Ref. 35). Almost all (159 out of 171) of the animal and cell culture studies cited in the petition in Appendix D–E were included in the NTP systematic review. From among 4,656 studies identified in the NTP database search, 4,552 were excluded during title and abstract screening, 104 were reviewed at the full-text level and 68 studies were considered relevant and were included in the analysis. NTP assessed each study for bias, meaning a systematic error in the study that can over or underestimate the true effect and further excluded any studies with a high risk of bias. Of the 68 studies, including studies provided by the Fluoride Action Network, 19 were considered to pose a very serious overall risk of bias, primarily based on concern for at least three of the following factors: Lack of randomization, lack of blinding at outcome assessment in conjunction with not using automated tools to collect information, lack of reporting on what was administered to animals (source, purity, chemical form of fluoride), lack of control for litter effects, lack of expected response in control animals, and lack of reporting of key study information such as the number or sex of animals treated. Of the studies cited in Table 4 in the petition, two were excluded from the NTP analysis because of serious concerns for study bias (Refs. 36 and 37). Based on its review of animal and cell studies, NTP concluded that ''[t]he evidence is strongest (moderate level-of-evidence) in animals exposed as adults tested in the Morris water maze and weaker (low level-of-evidence) in animals exposed during development'' and ''[v]ery few studies assessed learning and memory effects at exposure levels near 0.7 parts per million, the recommended level for community water fluoridation in the United States.'' The animal studies cited in the petition (Ref. 1, p. 14, Table 4) reflect these high drinking water exposures ranging from 2.3 mg/L to 13.6 mg/L, equivalent to 3–20 times the levels to which drinking water is fluoridated in the U.S. Overall, NTP concluded that, ''[r]esults show low-to-moderate level-of-evidence in developmental and adult exposure studies for a pattern of findings suggestive of an effect on learning and memory'' (Ref. 35, p. 52). Based on this review of available evidence, and the identified limitations in the database, NTP is currently pursuing experimental studies in rats to address key data gaps, starting with pilot studies that address limitations of the current literature with respect to study design (*e.g.,* randomization, blinding, control for litter effects), and assessment of motor and sensory function to assess the degree to which impairment of movement may impact performance in learning and memory tests. If justified, follow-up studies would address potential developmental effects using lower dose levels more applicable to human intakes.

Two studies included in Table 4 (Ref. 1) were not included in the NTP review, but do not show neurotoxicity effects at doses relevant to U.S. populations. One study aimed to establish vitamin A as a marker for fluoride neurotoxicity (Ref. 38), but changes in vitamin A were measured only at an excessive fluoride dose of 20 mg/L. The other study dosed rats with fluoride in drinking water (Ref. 39) and showed effects on behavior and brain neurotransmitters at a dose of 5 mg/L, a level well above the 0.7 parts per million level recommended for community water fluoridation in the United States. Other studies in Table 4, which, according to the title of the table, are indicative of ''Water Fluoride Levels Associated with Neurotoxic Effects in Rodents,'' erroneously report effect levels not supported by the studies themselves. In Wu et al. (Ref. 36), which NTP excluded based on high bias, no adverse effects were seen at a dose of 1 mg/kg-day as claimed in the petition. In fact, the behavioral effects occurred only at doses of 5 and 25 mg/L. In Chouhan et al. (Ref. 40), which NTP excluded in the initial screen for relevancy, no significant neurotoxicity was seen at 1 mg/L fluoride, in contrast to what the petition claims. In addition, the petition's statement that ''rats require 5 times more fluoride in their water to achieve the same level of fluoride in their blood as humans'' (Ref. 1) as a rationale for why higher exposure levels in animals are relevant to lower levels in humans is not supported by the NTP review in the petition. The NTP review indicates that ''assuming approximate equivalence [of drinking water concentrations in rodents and humans] is not unreasonable'' (Ref. 35, p. 58). These several erroneously reported studies do not change EPA's agreement with the conclusions of the NTP report that their ''[r]esults show low-to-moderate level-of-evidence in developmental and adult exposure studies for a pattern of findings suggestive of an effect on learning and memory'' (Ref. 35, p. 52).

In cell studies cited in the petition, two studies demonstrated effects following exposure of artificial brain cells to fluoride at concentrations in the range purported to be in the bloodstream of humans. However, relevance of cell assays to humans is limited because the concentrations of fluoride experienced by cells by themselves in culture are not directly comparable to an animal or human exposure due to lack of metabolism, interactions between cells, and the ability to measure chronic (long-term) effects (Ref. 41). Extrapolation from concentrations in cell cultures to human exposures is not straightforward. Pharmacokinetic modeling is necessary to convert the concentrations to a

human equivalent dose relevant to risk assessment (Ref. 42), but the petition did not address whether data are available or lacking to complete such an analysis.

4. *Susceptible subpopulations are at heightened risk.* The data and information provided in the petition do not support the claims that ''nutritional status, age, genetics and disease are known to influence an individual's susceptibility to chronic fluoride toxicity.'' The only reference the petition presents that specifically addresses the claim that nutrient deficiencies (*i.e.,* deficiencies in iodine and calcium) can ''amplify fluoride's neurotoxicity'' is the study by Das and Mondal (Ref. 26). However, the study did not measure any nutrients in their test subjects. Rather, they measured Body Mass Index (BMI), acknowledging that ''BMI is the most commonly used measure for monitoring the prevalence of overweight and obesity at population level'' and ''it is only a proxy measure of the underlying problem of excess body fat or underweight cases.'' Not only is the BMI an indirect proxy for the iodine and calcium deficiencies supposed in the petition, the BMI results presented in this study are themselves equivocal, as they show that BMIs ranged from underweight to overweight to obesity depending on the sex and age of the study subjects. Furthermore, the petition concedes that the Das and Mondal study data are only ''suggestive'' of an area with chronic malnutrition. A few human studies cited provide only suggestive evidence that low levels of iodine may increase the effects of high levels of fluoride in children, but these studies suffer from study design and confounding issues already described previously. Other cited studies describe the effects of iodine or calcium on rats or rat brain cells in addition to irrelevantly high fluoride levels. The petition also claims that a certain ''COMT gene polymorphism greatly influences the extent of IQ loss resulting from fluoride exposure,'' citing a study by Zhang et al. (Ref. 29) as support. The COMT gene encodes for the enzyme, catechol-O-methyltransferase, which is responsible for control of dopamine levels in the brain. Zhang et al. concludes that, ''[t]he present study has several limitations. First, the cross-sectional observational design does not allow us to determine temporal or causal associations between fluoride and cognition. Second, the study has a relatively small sample size, which limits the power to assess effects of gene-environmental interactions on children's IQ'' (Ref. 29). Zhang et al. continues ''[d]espite the study limitations, this is the first gene-environment study investigating the potential impact of COMT single-nucleotide polymorphism (SNP) on the relationship between children's cognitive performance and exposure to elemental fluoride'' (Ref. 29). Several studies are cited in the petition to support the assertion that infants, the elderly and individuals with deficient nutritional intake and kidney disease are more susceptible to fluoride neurotoxicity. However, the level of supporting evidence from these studies (*i.e.,* to specify the potentially greater susceptibility of any particular subpopulation) is insufficient to overcome the petition's broader failure to set forth sufficient facts to establish that fluoridation chemicals present an unreasonable risk to the general population, to allow EPA to reach a risk evaluation.

5. *RfD/RfC derivation and uncertainty factor application.* An oral Reference Dose or inhalation Reference Concentration is a daily exposure to the human population, including sensitive subgroups, that is likely to be without an appreciable risk of deleterious effects during a lifetime (Ref. 43). The petition cites EPA's 1998 guidance document, *Guidelines for Neurotoxicity Risk Assessment* (Ref. 44), purporting that it demonstrates the necessity of applying an uncertainty factor of at least 10. It appears that the petition has selected the eight studies presented in Table 5 (Ref. 1, p. 19) as candidates for deriving a Reference Dose (RfD) or Reference Concentration (RfC). The petition asserts that these dose or concentration values are relevant oral reference values for neurotoxic effects. However, the petition fails to recognize that the question of applying an uncertainty factor does not even arise until one has first appropriately performed a hazard characterization for all health endpoints of concern (Ref. 30, Section 3.1). As outlined in EPA's document, *A Review of the Reference Dose and Reference Concentration Processes* (Ref. 43), the first step in deriving an RfD or RfC is to evaluate the available database. The petition does not set forth the strengths and limitations of each of the studies in the overall database of available studies nor any criteria or rationale for selecting the eight particular studies from which to derive an RfD or RfC. Without setting forth the strengths and limitations associated with each study and the weight of evidence provided by the available database, a necessary step in any assessment, it is not possible to determine whether uncertainty factors are necessary.

Following hazard characterization and identification of suitable studies for an RfD or RfC, uncertainty factors are generally applied to a lower limit dose or concentration on the continuum of observed effects (dose-response curve) in an individual study (*e.g.,* NOAEL, LOAEL, Benchmark Dose, etc.). The selection of uncertainty factors and their magnitude should be based on the quality of the data, extent of the database and sound scientific judgment and consider the impact of having adverse effects from an inadequate exposure as well as an excess exposure. Uncertainty factor values may be considered appropriate to account for uncertainties associated with extrapolating from (1) a dose producing effects in animals to a dose producing no effects, (2) subchronic to chronic exposure in animals, (3) animal toxicological data to humans (interspecies), (4) sensitivities among the members of the human population (intraspecies), and (5) deficiencies in the database for duration or key effects (Ref. 43). Conflicting statements in the petition indicate that there is both a robust and certain dose-response relationship between fluoride exposure and IQ including for sensitive subpopulations. However, the petition does not clearly identify which sources/types of uncertainty in the data exist, nor which of the aforementioned uncertainty factors should be applied based on the review of the selected studies.

6. *Benefits to public health.* The petition asserts that the fluoridation of drinking water confers little benefit to public health, claiming that the primary benefit of fluoride comes from topical fluoride contact with the teeth and that there is thus little benefit from ingesting fluoride in water or any other product. The petition claims there are no randomized controlled trials on the effectiveness of fluoridation, and that few studies adequately account for potential confounding factors. In addition, the petition states that modern studies of fluoridation and tooth decay have found small, inconsistent and often non-existent differences in cavity rates between fluoridated and non-fluoridated areas. Further, the petition questions the cost-effectiveness of fluoridation relative to costs associated with what have been asserted to be fluoridation-related drops in IQ. The petition argues, then, that there is ''little justification'' in exposing the public to ''*any* risk'' of fluoride neurotoxicity (Ref. 1).

EPA does not believe that the petition has presented a well-founded basis to doubt the health benefits of fluoridating drinking water. The petition's argument about fluoridation benefits (*i.e.,* that the risks of neurotoxic health effects from fluoridation are unreasonable in part because they outweigh the expected health benefits arising from exposure to fluoride) depends on first setting forth sufficient facts to establish the purported neurotoxic risks, to which the countervailing health benefits from fluoridation could be compared. But as noted earlier, EPA and other authoritative bodies have previously reviewed many of the studies cited as evidence of neurotoxic effects of fluoride in humans and found significant limitations in using them to draw conclusions on whether neurotoxicity is associated with fluoridation of drinking water. Irrespective of the conclusions one draws about the health benefits of drinking water fluoridation, the petition did not set forth sufficient facts to justify its primary claims about purported neurotoxic effect from drinking fluoridated water.

The petition cites several studies as evidence that water fluoridation does not have any demonstrable benefit to the prevention of tooth decay (Refs. 45–49). However, EPA has found substantial concerns with the designs of each of these studies including small sample size and uncontrolled confounders, such as recall bias and socioeconomic status. Additionally, in Bratthall et al. (Ref. 45), for example, the appropriate interpretation of the responses of the 55 dental care professionals surveyed, based on the data provided in the paper, is that in places where water is fluoridated, the fluoridation is the primary reason for the reduction in dental caries. Diesendorf (Ref. 49) cites only anecdotal evidence and Cheng et al. (Ref. 46) is commentary only, with no supporting data.

EPA is mindful of the public health significance of reducing the incidence of dental caries in the U.S. population. Dental caries is one of the most common childhood diseases and continues to be problematic in all age groups. Historically, the addition of fluoride to drinking water has been credited with significant reductions of dental caries in the U.S. population. In 2000, the then-Surgeon General noted that ''community water fluoridation remains one of the great achievements of public health in the twentieth century—an inexpensive means of improving oral health that benefits all residents of a community, young and old, rich and poor alike.'' The U.S. Surgeon General went on to note, ''it [is] abundantly clear that there are profound and consequential disparities in the oral health of our citizens. Indeed, what amounts to a silent epidemic of dental and oral diseases is affecting some population groups.'' (Ref. 50).

At that time, among 5- to 17-year-olds, dental caries was more than five times as common as a reported history of asthma and seven times as common as hay fever. Prevalence increases with age. The majority (51.6 percent) of children aged 5 to 9 years had at least one carious lesion or filling in the coronal portion of either a primary or a permanent tooth. This proportion increased to 77.9 percent for 17-year-olds and 84.7 percent for adults 18 or older. Additionally, 49.7 percent of people 75 years or older had root caries affecting at least one tooth (Ref. 50).

More recently, from the National Health and Nutrition Examination Survey (NHANES) for 2011–2012, approximately 23% of children aged 2–5 years had dental caries in primary teeth. Untreated tooth decay in primary teeth among children aged 2–8 was twice as high for Hispanic and non-Hispanic black children compared with non-Hispanic white children. Among those aged 6–11, 27% of Hispanic children had any dental caries in permanent teeth compared with nearly 18% of non-Hispanic white and Asian children. About three in five adolescents aged 12–19 years had experienced dental caries in permanent teeth, and 15% had untreated tooth decay (Refs. 51).

Further, in 2011–2012, 17.5 percent of Americans ages 5–19 years were reported to have untreated dental caries, while 27.4 percent of those aged 20–44 years had untreated caries (Ref. 52). For those living below the poverty line, 24.6 percent of those aged 5–19 years and 40.2 percent of those aged 20–44 years had untreated dental caries (Ref. 52). Untreated tooth decay can lead to abscess (a severe infection) under the gums which can spread to other parts of the body and have serious, and in rare cases fatal, results (Ref. 53). Untreated decay can cause pain, school absences, difficulty concentrating, and poor appearance, all contributing to decreased quality of life and ability to succeed (Ref. 54).

These data continue to suggest dental caries remains a public health problem affecting many people. Fluoride has been proven to protect teeth from decay by helping to rebuild and strengthen the tooth's surface or enamel. According to the Centers for Disease Control and Prevention and the American Dental Association, water fluoridation prevents tooth decay by providing frequent and consistent contact with low levels of fluoride (Refs. 55 and 56). Thus, the health benefits of fluoride include having fewer cavities, less severe cavities, less need for fillings and removing teeth, and less pain and suffering due to tooth decay (Ref. 55).

Fluoride protects teeth in two ways—systemically and topically (Ref. 57). Topical fluorides include toothpastes, some mouth rinse products and professionally applied products to treat tooth surfaces. Topical fluorides strengthen teeth already in the mouth by becoming incorporated into the enamel tooth surfaces, making them more resistant to decay. Systemic fluorides are those ingested into the body. Fluoridated water and fluoride present in the diet are sources of systemic fluoride. As teeth are developing (pre-eruptive), regular ingestion of fluoride protects the tooth surface by depositing fluorides throughout the entire tooth surface (Ref. 56). Systemic fluorides also provide topical protection as ingested fluoride is present in saliva which continually bathes the teeth (Ref. 56). Water fluoridation provides both systemic and topical exposure which together provide for maximum reduction in dental decay (Ref. 56).

The Surgeon General, the Public Health Service and the Centers for Disease Control and Prevention reaffirmed in 2015 the importance of community water fluoridation for the prevention of dental caries and its demonstrated effectiveness (Refs. 54 and 58). In the Public Health Service's 2015 *Recommendation for Fluoride Concentration in Drinking Water,* they note ''there are no randomized, double-blind, controlled trials of water fluoridation because its community-wide nature does not permit randomization of individuals to study and control groups or blinding of participants. However, community trials have been conducted, and these studies were included in systematic reviews of the effectiveness of community water fluoridation. As noted, these reviews of the scientific evidence related to fluoride have concluded that community water fluoridation is effective in decreasing dental caries prevalence and severity'' (Ref. 59).

7. *Extent and magnitude of risk from fluoridation chemicals.* The petition argues that the purported risks of drinking water fluoridation are unreasonable in part because they are borne by a large population. The petition (in its discussion of the extent and magnitude of risk posed) cites the total U.S. population and estimates the

number of U.S. children under the age of 18 years who live in areas where artificial fluoridation occurs. That estimate is then multiplied by an estimate of the average decrease in lifetime earnings associated with IQ point loss to calculate the overall potential IQ point loss and associated decrease in lifetime earnings for the segment of the U.S. population under the age of 18 years potentially exposed to artificially fluoridated water. The petition concludes, based on the potential extent and magnitude of exposure to fluoridation chemicals, that fluoridation would have caused ''a loss of between 62.5 to 125 million IQ points'' (Ref. 1, p. 24).

The petition has not set forth a scientifically defensible basis to conclude that any persons have suffered neurotoxic harm as a result of exposure to fluoride in the U.S. through the purposeful addition of fluoridation chemicals to drinking water or otherwise from fluoride exposure in the U.S. Still less has the petition set forth a scientifically defensible basis to estimate an aggregate loss of IQ points in the U.S., attributable to this use of fluoridation chemicals. As noted previously, EPA has determined the petition did not establish that fluoridation chemicals present an unreasonable risk of injury to health or the environment, arising from these chemical substances' use to fluoridate drinking water. The fact that a purported risk relates to a large population is not a basis to relax otherwise applicable scientific standards in evaluating the evidence of that purported risk. EPA and other authoritative bodies have previously reviewed many of the studies cited as evidence of neurotoxic effects of fluoride in humans and found significant limitations in using them to draw conclusions on whether neurotoxicity is associated with fluoridation of drinking water. In contrast, the benefits of community water fluoridation have been demonstrated to reduce dental caries, which is one of the most common childhood diseases and continues to be problematic in all age groups. Left untreated, decay can cause pain, school absences, difficulty concentrating, and poor appearance, all contributing to decreased quality of life and ability to succeed (Ref. 54).

8. *Consequences of eliminating use of fluoridation chemicals.* Apparently citing to a repealed provision of TSCA (15 U.S.C. 2605(c)[1](A) (2015)) and guidance issued with respect to that statutory provision, the petition argues that the following factors are germane to determining whether the alleged neurotoxic risks presented by fluoridation chemicals are unreasonable: ''the societal consequences of removing or restricting use of products; availability and potential hazards of substitutes, and impacts on industry, employment, and international trade.'' Along these lines, the petition includes claims such as the following: That any risks of fluoridation chemicals could be easily reduced by discontinuing purposeful fluoridation practices; that alternative topical fluoride products have widespread availability; and that the impacts on the requested rule on industry, employment, and international trade would be little, if any. In short, the petition urges EPA to conclude that the risks of fluoridation chemicals are unreasonable, in part because if EPA found that the risks were unreasonable, the cost and non-risk factors that EPA would need to address in ensuing risk management rulemaking could be readily addressed. But this sort of ends-driven reasoning is forbidden by the texts of section 6(b)(4)(A) and 21(b)(4)(B)(ii) of the amended TSCA, which exclude ''costs or other non-risk factors'' from the unreasonable risk determination. It is also plainly inconsistent with Congress' intent, in amending TSCA, to ''de-couple'' the unreasonable risk decision from the broader set of issues (*e.g.,* chemical alternatives and regulatory cost-effectiveness) that may factor into how best to manage unreasonable risks, once particular risks have been determined to be unreasonable. See S. Rep. 114–67 at 17 (Ref. 3); H.R. Rep. 114–176 at 23 (Ref. 4); and 162 Cong. Rec. S3516 (Ref. 60).

9. *Link to elevated blood lead levels.* To support the contention that TSCA (and not the Safe Drinking Water Act [SDWA]) is the appropriate regulatory authority, the petition asserts an association between fluoridation chemicals and elevated blood lead levels and claims that there is laboratory and epidemiological research linking artificial fluoridation chemicals with pipe corrosion. The petition then argues that issuing a rule under TSCA section 6 rather than SDWA would allow EPA to specifically target and prohibit the addition of fluoridation chemicals to drinking water. The petition argues that SDWA would not allow EPA to distinguish between intentionally-added, artificial and naturally-occurring fluoride. It is in the public interest, says the petition, to opt for the regulatory option that is less expensive and can be more narrowly tailored.

Regarding the claims about the relative extent of legal authorities under TSCA and SDWA, EPA notes that the petition has not set forth any specific legal basis for its views on the purported limitations of SDWA. For this reason, and because the petition has not set forth facts sufficient to show that the fluoridation of drinking water presents an unreasonable risk under TSCA, the Agency need not resolve such legal questions in order to adjudicate this petition.

EPA has further observations about the petition's claims that drinking water fluoridation is linked to lead hazards. The Centers for Disease Control and Prevention (CDC) studied the relationship between fluoridation additives and blood lead levels in children in the United States (Ref. 61). More than 9,000 children between the ages of 1–16 years were included in the study's nationally representative sample. The petition argues that the study, and Table 4 in particular, shows that fluorosilicic acid was associated with increased risk of high blood lead levels. In fact, Macek et al. concluded that their detailed analyses did not support concerns that silicofluorides in community water systems cause high lead concentrations in children. The petition also points to another study (Ref. 62) which re-analyzed CDC's data and concluded that children exposed to ''silicofluoridated'' water had an elevated risk of having high blood lead levels. Coplan et al. (Ref. 62) criticized the Macek et al. approach as flawed and reevaluated the NHANES data comparing systems that used silicofluorides to all systems (*e.g.,* a combination of fluoridated, nonfluoridated and naturally fluoridated) and found a small difference between the number of children in each group with blood lead levels >5 μg/dL; the results were not evaluated to see if the difference was statistically significant. A number of other chemical characteristics are known to increase lead release into water sources such as pH, natural organic matter, water hardness, oxidant levels, and type of piping, age of housing; the Coplan et al. study did not evaluate these factors.

In any event, the Agency is not persuaded that the examination of the relationship between fluoridation chemicals, pipe corrosion, and elevated blood lead levels nor their bearing on the comparative efficacy of TSCA or SDWA is germane to the disposition of the petition. Under TSCA, where the EPA Administrator determines ''that the manufacture, processing, distribution in commerce, use, or disposal of a chemical substance or mixture . . . presents an unreasonable risk of injury

to health or the environment, the Administrator shall by rule [regulate a] . . . substance or mixture to the extent necessary so that the chemical substance or mixture no longer presents such risk'' 15 U.S.C. 2605(a). As previously discussed, the petition does not demonstrate that purposeful addition of fluoridation chemicals to U.S. water supplies presents such unreasonable risk.

10. *Regulation of fluoridation chemicals as a category.* EPA has broad discretion to determine whether to regulate by category under TSCA section 26(c) rather than by individual chemical substances. In a prior evaluation of a section 21 petition seeking the regulation of a category of chemical substances, EPA explained that it does so in light of Congress' purpose in establishing the category authority: To ''facilitate the efficient and effective administration'' of TSCA. See 72 FR 72886 (Ref. 63) (citing Senate Report No. 94–698 at 31). It is of course self-evident that various chemical substances constituting ''fluoridation chemicals'' would have in common their use to fluoridate drinking water. But as discussed in Unit III., the inquiry does not end there. If EPA were to grant the petitioner's request, the Agency would become obligated to address all conditions of use of the category. If certain chemical substances comprising the category present conditions of use that other members do not, and any of those conditions of use would be significant to whether the category as a whole presents an unreasonable risk to human health or the environment, then the overall approach of regulating by category is less suited to the efficient and effective administration of TSCA. But the petition does not set forth facts that would enable the Agency to reasonably evaluate whether a category approach on fluoridation chemicals would be consistent with the efficient and effective administration of TSCA. Nor does the petition set forth the specific chemical substances that should comprise the category of fluoridation chemicals.

11. *Specification of an adequate rule under TSCA section 6(a).* As discussed earlier, the petition does not set forth facts that satisfactorily demonstrate to the Agency that fluoridation chemicals present an unreasonable risk to human health, specifically arising from these chemical substances' use to fluoridate drinking water. But even if the petition had done so, it would still be inadequate as a basis to compel the commencement of section 6(a) rulemaking proceeding under TSCA section 21. This is because the petition does not address whether fluoridation chemicals would still present an unreasonable risk, even after implementing the requested relief, arising from other conditions of use. As discussed earlier in Unit III., EPA interprets TSCA section 21 as requiring a petition to address the full set of conditions of use for a chemical substance and thereby describe an adequate rule under TSCA section 6(a), as opposed to a rule that would merely address a particular subset of uses of special interest. The petition at issue pays little or no attention to the other conditions of use of the various fluoridation chemicals (*i.e.,* uses other than the eponymous use to treat drinking water) and makes no claim for any of these chemical substances that the risks to be addressed by curtailing drinking water fluoridation would be the only unreasonable risks or even the most significant unreasonable risks. This problem is compounded by the petition's lack of specificity as to which chemical substances are being construed as ''fluoridation chemicals.''

EPA acknowledges that its interpretation of the requirements of TSCA section 21, for petitions seeking action under TSCA section 6, was not available to petitioners at the time they prepared this petition. EPA has issued general guidance for preparing citizen's petitions, 50 FR 56825 (1985), but that guidance does not account for the 2016 amendments to TSCA. Particularly relevant under these circumstances, the Agency wishes to emphasize that its denial does not preclude petitioners from obtaining further substantive administrative consideration, under TSCA section 21, of a substantively revised petition under TSCA section 21 that clearly identifies the chemical substances at issue, discusses the full conditions of use for those substances, and sets forth facts that would enable EPA to complete a risk evaluation under TSCA section 6(b) for those substances.

VI. References

As indicated under **ADDRESSES**, a docket has been established for this document under docket ID number EPA–HQ–OPPT–2016–0763. The following is a listing of documents that are specifically referenced in this notice. The docket itself includes both these referenced documents and further documents considered by EPA. The docket also includes supporting documents provided by the petitioner and cited in the petition, which are not available in the electronic version of the docket. For assistance in locating these printed documents, please consult the technical person listed under **FOR FURTHER INFORMATION CONTACT**.

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5. EPA. Procedures for Prioritization of Chemicals for Risk Evaluation Under the Toxic Substance Control Act; Notice. **Federal Register** (82 FR 4831, January 17, 2017).
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List of Subjects

Environmental protection, Fluoridation chemicals, Drinking water, Toxic Substances Control Act (TSCA).

Dated: February 17, 2017.

Wendy Cleland-Hamnett,

Acting Assistant Administrator, Office of Chemical Safety and Pollution Prevention.

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