C. ANDREW WATERS, ESQ., CA Bar No. 147259
MICHAEL CONNETT, ESQ., CA Bar No. 300314
WATERS, KRAUS & PAUL
222 N. Pacific Coast Hwy, Suite 1900
El Segundo, CA 90245
310-414-8146 Telephone
310-414-8156 Facsimile

Attorneys for Plaintiffs

UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF CALIFORNIA
AT SAN FRANCISCO

FOOD & WATER WATCH, et al., Plaintiffs, vs. U.S. ENVIRONMENTAL PROTECTION AGENCY, et al. Defendants.	Civ. No. 17-CV-02162-EMC **PLAINTIFFS' NOTICE OF MOTION & MOTION FOR SUMMARY JUDGMENT AND PARTIAL SUMMARY JUDGMENT** Date: November 7, 2019 Time: 1:30 a.m. Judge: Hon. Edward Chen Courtroom: 5, 17th Floor

NOTICE OF MOTION

PLEASE TAKE NOTICE THAT on November 7, 2019, at 1:30 p.m., before the Honorable Edward Chen, Courtroom 5, 17th Floor, 450 Golden Gate Avenue, San Francisco, California, 94102, Plaintiffs Food & Water Watch, et al. will and hereby do move the Court for an order granting summary judgment and partial summary judgment in their favor.

Plaintiffs are entitled to full and partial summary judgment under Federal Rule of Civil Procedure 56 for three reasons. First, there is no genuine dispute of fact that, under EPA's longstanding risk assessment policies, fluoridation chemicals pose an "unacceptable risk" to human health when added to drinking water. EPA's experts have not materially disputed this; instead they argue that there is not yet factual certainty that fluoride in water at the concentration of 0.7 mg/L causes neurotoxicity. But factual certainty is not, nor ever has been, the standard that EPA uses to assess and regulate *risk*, nor is it

the standard that Congress established for TSCA, or the standard that courts have used when assessing risk determinations under health-protective statutes. Second, by the admission of EPAs own expert on TSCA, any benefits that fluoride has to dental health are a "non-risk factor" and thereby should be excluded from this case in light of Congress's prohibition against considering "costs or *non-risk factors*" in TSCA risk evaluations. 15 U.S.C. § 2605(b)(4)(A) (emphasis added). Third, EPA has indicated that it intends to request a lengthy delay in the implementation of rulemaking proceedings should the Court make an unreasonable risk determination. EPA has failed, however, to establish the predicate findings under 15 U.S.C. § 2620(b)(4)(B)(ii) to warrant this relief because it has failed to produce any evidence to establish that the extent of the risk posed by fluoridation chemicals is less than the risk of chemicals that EPA is taking action on under TSCA.

This motion is based on the pleadings and other papers filed in this case; the attached memorandum, declarations, and exhibits filed with this motion; and such other matters as may be presented to the Court.

Dated: October 9, 2019

Respectfully submitted,

/s/ Michael Connett
MICHAEL CONNETT
Attorney for Plaintiffs

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I. STATEMENT OF ISSUES TO BE DECIDED

1. Is there a genuine dispute of fact as to the unreasonable risk posed by the condition of use of a chemical where Plaintiffs produced evidence showing an unacceptable risk using EPA's own risk assessment procedures, and EPA responded—not by countering the assessment of risk—but by presenting evidence that there is not conclusive proof of harm at the specific exposure level caused by the use?

2. Does the Toxic Substances Control Act, as amended in 2016, permit consideration of the health benefits of a chemical during the risk evaluation phase when the statute (15 U.S.C. § 2605(b)(4)(A)) expressly forbids risk evaluations from considering "costs or non-risk factors"?

3. Is relief under 15 U.S.C. § 2620(b)(4)(B)(ii) precluded when EPA has no evidence to demonstrate that the extent of the risk alleged by the petitioner is less than the extent of the risk that the EPA is taking action under TSCA?

II. INTRODUCTION

This is a case about the risks that arise when a neurotoxicant is widely dispersed into the environment. In 2016, Plaintiff citizens filed a Citizen Petition with the EPA asking the Agency to use its authority under the Toxic Substances Control Act to prohibit the addition of fluoridation chemicals to water—a practice that affects over 200 million Americans—on the grounds that this use of fluoride presents an unreasonable risk of neurologic harm. As noted by Dr. Philippe Grandjean from Harvard School of Public Health, the "unfortunate conclusion we have to accept today . . . is that fluoride exposures, as they occur in America today, primarily from fluoridated drinking water, are toxic to brain development in humans." **Ex. 14** at 76:3-8. After over two years of litigation, Plaintiffs now move the Court for summary judgment on the following three grounds:

First, there is no genuine dispute of fact that, under EPA's longstanding risk assessment policies, fluoridation chemicals pose an "unacceptable risk" to human health when added to drinking water. EPA's experts have not disputed this; they argue, instead, that there is not yet conclusive proof that fluoride in drinking water at 0.7 mg/L causes neurotoxicity in humans. Conclusive proof of harm, however, is not, nor ever has been, the standard that EPA uses to assess and regulate risk. *See, e.g.*, **Ex. 32** at 303:4-14. Were this the standard, EPA would not have been able to order the phase-out of lead from gasoline in the 1970s. *Ethyl Corp. v. U.S. E.P.A.*, 541 F.2d 1, 8 &12 (D.C. 1976). In fact, Congress specifically rejected the need

for "factual certainty" when it enacted the Toxic Substances Control Act ("TSCA") stating that its "overriding purpose" was "to *prevent* harm" *before* it occurred. **Ex. 37** at 7 & 32.

Second, Congress recently amended TSCA to prohibit any consideration of "costs and non-risk factors" in TSCA risk evaluations. 15 U.S.C. § 2605(b)(4)(A). Dr. Tala Henry, the Deputy Director of EPA's Office of Pollution Prevention and Toxics, testified as a non-retained expert for EPA that any dental health benefits of fluoride would be a "non-risk factor" that can only be considered during the rule-making (i.e., risk-management) phase. **Ex. 32** at 383:11-387:3, 389:8-13, 390:8-391:5. This admission, and the plain meaning of the Act, make clear that caries prevention should be stricken from this case.

Third, EPA has indicated that it will ask the Court to exercise its authority under 15 U.S.C. § 2620(b)(4)(B)(ii) to postpone any rulemaking proceeding should the Court make an unreasonable risk finding. However, relief under § 2620(b)(4)(B)(ii) is only available if the EPA can show that the "extent of risks to health" from fluoridation chemicals are less than those posed by the chemicals for which EPA is currently taking action on under TSCA. EPA has proffered one witness in this case to establish the requisite finding for a delay. **Ex. 50** at 17-23. The witness testified, however, that she has no opinion, nor even a basis for an opinion, on this subject. **Ex. 32** at 482:5-15, 491:17-492:2, 499:12-19, 500:10-14, 505:3-19, 506:20-507:18; *see also id.* at 94:3-95:18. Accordingly, EPA has no evidentiary basis to meet its burden and partial summary judgment should be granted.

III. STATEMENT OF FACTS

A. Plaintiffs' Section 21 Citizen Petition

On November 22, 2016, Plaintiffs filed a Citizen Petition requesting that the EPA exercise its authority under the Toxic Substances Control Act (TSCA), 15 U.S.C. § 2605(a), to prohibit the addition of fluoridation chemicals to drinking water. Plaintiffs filed the petition on the grounds that "application of the Agency's *Guidelines for Neurotoxicity Risk Assessment* to the existing database on fluoride shows that (1) neurotoxicity is a hazard of fluoride exposure, and (2) the reference dose that would reasonably protect against this hazard is incompatible with the doses now ingested by millions of Americans in fluoridated

areas." **Ex. 1**[1] at 1. EPA denied the petition on February 27, 2017, and Plaintiffs thereupon filed the instant *de novo* action.

B. EPA's Current Regulation of Fluoride in Drinking Water

EPA currently regulates fluoride under the Safe Drinking Water Act (SDWA) because the Agency recognizes fluoride in drinking water can produce adverse health effects. 402 U.S.C. § 300f(1)(B); 40 CFR § 141.51 & 141.62. EPA's current drinking water standard (i.e., Maximum Contaminant Level Goal, or MCLG[2]) for fluoride was established in 1985 to protect against an advanced form of fluoride poisoning known as crippling skeletal fluorosis. **Ex. 2** at 80:20-81:5. Neurotoxicity was not considered by EPA when setting this standard. **Ex. 2** at 150:9-19; 152:9-14; **Ex. 3A**, at 5.

The lead scientist on fluoride issues at EPA's Office of Water, Dr. Joyce Donohue, admitted that the MCLG is an "out of date" standard. **Ex. 4** at 88:7-10. By only protecting against the *crippling* phase of skeletal fluorosis, the MCLG does not protect against earlier stages of the disease, despite admissions by the EPA and Department of Health & Human Services that the earlier stages of the disease cause "chronic joint pain" and "calcification of the ligaments." **Ex. 2** at 81:6-82:20, 83:9-16; 84:5-20; **Ex. 4** at 79:23-80:6; **Ex. 5** at 46; **Ex. 6** at 69:24-71:17, 72:9-25, 73:2-10. The MCLG also permits up to 22% of children to develop a condition known as "severe dental fluorosis," a disfiguring disorder marked by extensive staining and pitting of the teeth that can cause "severe embarrassment" and "profound" effects on a child's self-esteem. **Ex. 2** at 34:4-7, 62:2-10, 63:4-10, 75:8-16, 77:15-78:8, 80:3-19; **Ex. 4** at 73:14-20, 77:7-25, 79:13-22; **Ex. 7** at 2-3. Dr. Donohue noted that children with this condition do not smile because they're too embarrassed to show their teeth. **Ex. 4** at 76:9-22.

From the outset, the MCLG for fluoride was vigorously opposed by some of EPA's own scientists,

[1] All exhibits cited herein are attached to the accompanying declaration by Michael Connett.

[2] The MCLG is a non-enforceable health-based goal that must protect against known and anticipated adverse health effects with an adequate margin of safety and without consideration of cost or technical feasibility. 42 U.S.C. 300g-1(b)(4). The enforceable standard (the MCL) is permitted to consider costs and technical feasibility. (*Id*.) In the case of fluoride, the MCLG and MCL are both set at 4 mg/L. 40 CFR § 141.51 & 141.62.

who took the unprecedented step of filing an amicus brief in support of an environmental organization's lawsuit against the Agency over the standard. **Ex. 2** at 91:10-13, 128:7-13; **Ex. 6** at 79:20-81:12, 81:25-82:8. In 2006, the National Research Council (NRC) unanimously concluded that the MCLG is unsafe and called on EPA to lower the level.[3] **Ex. 2** at 164:3-9, 166:3-14; **Ex. 4** at 90:15-21. Thirteen years have passed since the NRC issued its conclusions, but the EPA has *still* not yet lowered the MCLG. **Ex. 4** at 88:2-10.

C. Fluoride Neurotoxicity

Research from 1990s to 2006: In the 30+ years since EPA enacted the MCLG, a large body of peer-reviewed science has emerged linking fluoride exposure to adverse effects on the brain, including neurochemical changes, IQ decrements, and ADHD. **Ex. 3A** at 5-6, 10-11, 15-18. One of the first studies to raise red flags about fluoride's impact on the brain was co-authored by an EPA neurotoxicologist named Dr. Karl Jensen. **Ex. 4** at 38:2-13, 95:9-97:6; **Ex. 6** at 100:22-101:17, 102:17-104:12. Dr. Jensen's study found that rats drinking 1 mg/L of fluoride in their water (the same concentration then used to fluoridate drinking water) developed neuronal deformations and other cellular damage in the brain. **Ex. 6** at 100:22-101:17. Dr. Jensen's findings caused concern among EPA's professional staff; in 1999, the union representing EPA professionals at EPA headquarters called for an end fluoridation, based in part on Dr. Jensen's findings. **Ex. 8** at 180 & 182-83. The EPA took no action at that time, but in 2003, the Agency asked the NRC to review the new toxicologic data on fluoride, including the neurotoxicity literature. **Ex. 4** at 89:23-90:14. The NRC did so and released its landmark report in 2006. *Id.* at 90:15-17.

The NRC's 2006 report is widely recognized as an authoritative and extensive review of the toxicologic literature on fluoride; both the EPA and CDC have accepted NRC's findings as an "accurate summary of [fluoride's] hazard." **Ex. 10** at i; **Ex. 11** at 19:16-21, 114:17-24, 115:5-23. In its "Findings" section on neurotoxicity, the NRC concluded that fluoride causes neurochemical and anatomical changes

[3] In response to NRC's recommendation, EPA established a new "reference dose" for fluoride in 2010 to protect against severe dental fluorosis. This reference dose (0.08 mg/kg/day) is exceeded by some children living in communities with artificially fluoridated water (0.7 mg/l), particularly infants receiving formula reconstituted with tap water. **Ex. 3A** at 42-43; Ex. 9 at 233:11-15.

in animal brain, including: inhibition of acetylcholinesterase, increased oxidative stress, reductions in lipid/phospholipid content, alterations in neurotransmitter concentration and function, and diminishment in the energy available to the brain. **Ex. 11** at 120:14-121:9, 121:23-125:3, **Ex. 12** at 221-22; **Ex. 13** at 297:5-299:1, 301:8-12, 301:17-25, 302:18-22, 304:4-12, 317:17-318:17. Based on these findings, the NRC had enough information to conclude that fluoride "interferes with the brain" in animals. **Ex. 4** at 100:14-23; **Ex. 13** at 299:4-18; **Ex. 14** at 319:5-16). It was unclear to the NRC, however, if the neurochemical changes and cellular damage seen in the brain would manifest into outwardly demonstrable deficits in cognition/behavior (i.e., "functional" effects). **Ex. 12** at 223. The NRC called, therefore, for more animal research to examine fluoride's impact on cognitive skills. *Id.* at 223.

The NRC also reviewed the then-existing human data on neurotoxicity, which included four epidemiological studies from China associating fluoride exposure with reduced IQ in children. **Ex. 3A** at 6. Unlike the animal data, the NRC found that the human data—while suggestive of a problem—was not yet sufficient to draw conclusions. **Ex. 12** at 220-221. The NRC thus recommended that additional research be conducted in human populations to investigate fluoride's effects on the human brain, including studies to examine fluoride's relationship to IQ and dementia. **Ex. 2** at 172:20-173:20; **Ex. 12** at 223. Based on NRC's findings and recommendations, it was clear that "there were some big potential problems from fluoride exposure that needed to be investigated." **Ex. 4** at 107:18-24.

Human Studies from "High" Fluoride Areas: Subsequent to the NRC's review, a large number of human studies on fluoride and IQ have been published, the vast bulk of which have reported significant associations between fluoride and IQ loss.[4] Most of these studies have been conducted in China, and have examined the effect of waterborne fluoride levels (>1.5 mg/L) that generally exceed the levels added to water in the U.S. (0.7 mg/L). According to the National Toxicology Program (NTP), these studies provide "a strong suggestion" that fluoride reduces IQ at water fluoride levels exceeding 1.5 mg/L. **Ex. 15** at 3.

[4] Sixty-seven human studies have associated fluoride with cognitive deficits. **Ex. 3A** at 15, n. 102.

While these studies have a number of methodological limitations, they have been remarkably consistent in finding associations between fluoride and reduced IQ. In 2012, Dr. Philippe Grandjean, an environmental epidemiologist at the Harvard School of Public Health and renowned expert in the field of developmental neurotoxicity, published a meta-analysis which found that fluoride was associated with reduced IQ in 26 of the 27 studies that met the inclusion criteria of the analysis. **Ex. 13** at 315:18-316:6; **Ex. 16** ¶ 4. According to Dr. Grandjean, it is "very unlikely" that the consistent association between fluoride and IQ can be explained away by chance or some other factor. **Ex. 13** at 315:18-316:6; **Ex. 16B** at 6. Thus, in 2014, Dr. Grandjean and his colleague Dr. Phil Landrigan included fluoride as 1 of just 11 industrial chemicals[5] known to be a developmental neurotoxicant in humans. **Ex. 16A** at 9; **Ex. 17** at 1.

NIH-Funded Prospective Birth Cohort Studies: Since 1997, EPA has partnered with the National Institutes of Health (NIH) to "investigate new frontiers in the field of children's environmental health research." **Ex. 18** at 8. As part of this effort, EPA and NIH have funded prospective birth cohort studies to study the effect of environmental chemicals, including fluoride, on children's health. **Ex.** 18 at 70-71. Short of intentionally dosing humans in controlled experiments (which are prohibited for ethical reasons), prospective cohort studies are the "ideal study design" for understanding the impact of environmental chemicals on human health. **Ex. 19** at 162:25-163:7; **Ex. 20** at 182:23-183:11.

Two of the prospective cohort studies—the "ELEMENT" cohort in Mexico City and the "MIREC" cohort in Canada—have investigated the relationship between prenatal fluoride exposure and neurodevelopmental outcomes. **Ex. 20** at 15:5-14. In 2017, the first NIH-funded study ("Bashash 2017") on the relationship between prenatal fluoride and IQ was published. **Ex. 20** at 97:21-98:22. The study, which examined 299 mother-child pairs in the ELEMENT cohort, found a linear dose-response relationship between prenatal fluoride exposure (as measured in the urine of the mother) and reduced IQ in the children at ages 4 and 6-12. **Ex. 20** at 33:2-34:15; 108:20-24, 203:18-204:19. Notably, the concentrations of fluoride

[5] In addition to its use as a water treatment chemical, fluoride is the active ingredient in several pesticides and many dental products. *See, e.g.*, **Ex. 45** at 1.

found in the urine of the mothers were roughly the same as the maternal urinary fluoride concentrations measured in pregnant women living in areas with artificially fluoridated drinking water. **Ex. 20** at 31:4-32:11, 165:2-15, 181:5-12; **Ex. 21** at 209:3-12. Each 0.5 mg/L increase of fluoride in the mother's urine was associated with 3.15 less IQ points among the 4-year olds—an effect size that rivals the effects of lead. **Ex. 20** at 205:2-206:8.

In 2018, the second NIH-funded prospective study on fluoride was published ("Bashash 2018"), again from the ELEMENT cohort, but this time focusing on ADHD symptoms instead of IQ. *Id.* at 125:15-126:13. This second study found that prenatal fluoride exposure (as measured in the urine of the mother) was significantly associated with increased symptoms (i.e., inattention) of ADHD in the offspring. *Id.* at 34:16-35:6. According to Dr. Howard Hu, the results from the ELEMENT studies "are consistent with and support the conclusion that fluoride is a developmental neurotoxicant at levels of exposure seen in the general population in artificially fluoridated communities." *Id.* at 180:18-181:4. Dr. Donohue, from EPA's Office of Water, testified that the ELEMENT studies are "well-conducted," and further justify a reassessment of fluoride policies to ensure that children are not being overexposed. **Ex. 4** at 243:12-22, 254:18-255:4; 257:24-258:16, 271:2-272:19.

In August of 2019, the results of the NIH-funded prospective study of the MIREC cohort ("Green 2019") were published in *JAMA Pediatrics*. **Ex. 21** at 120:20-121:7. The study examined 512 mother-child pairs, and found that prenatal fluoride exposure was significantly associated with reduced IQ in boys at age 3 to 4. *Id.* at 124:19-23, 209:15-210:13. Each 1 mg/L increase of fluoride in the mothers' urine was associated with 4 to 5 less IQ points among the boys, an effect size on par with lead. *Id.* at 211:10-213:8. The study also found significant associations between IQ (in both boys and girls) and maternal fluoride intake from beverages *Id.* at 157:14-19. According to Dr. Bruce Lanphear, the senior investigator on the study, the findings from the MIREC study are "convergent" with the findings from the ELEMENT cohort and suggest that the *in utero* period is a "susceptible period of life vis-à-vis fluoride toxicity." *Id.* at 62:11-

64:9, 215:20-216:5. Based on these results, Dr. Lanphear recommends that pregnant women "begin taking steps to reduce their fluoride intake, including but not limited to reducing their consumption of fluoridated water." *Id.* The editor of *JAMA Pediatrics* made a similar recommendation in a *JAMA* podcast accompanying the release of the study. Connett Decl. ¶ 52; **Ex. 21** at 216:6-218:25.

In addition to the *JAMA Pediatrics* study, Dr. Lanphear's team has just finished a second study of the MIREC cohort ("Till, in press") examining the association between IQ and fluoride intake during *infancy*. **Ex. 21** at 165:18-166:1, 166:16-18. While not yet published, the new study of 398 children found a significant association between fluoride ingestion during infancy and reduced non-verbal IQ. *Id.* at 193:6-194:12. According to Dr. Lanphear, "this association remained significant after controlling for fetal fluoride exposure and other relevant covariates, suggesting that the susceptibility to fluoride's adverse neurological effects may extend into infancy." *Id.* at 223:20-224:7.

In this litigation, EPA has retained experts from an industrial consulting firm (Exponent) who have critiqued the precision of the prenatal exposure measurements used in the ELEMENT and MIREC cohorts.[6] While the studies use of spot urine sampling does introduce some imprecision into the exposure assessment, this limitation makes it *harder*, not easier, to detect an association between prenatal fluoride and IQ, and is thus an implausible explanation for the significant associations that have been found. **Ex. 16A** at 23 & 31; **Ex. 20** at 189:5-192:10; **Ex. 21** at 231:1-20.

Animal Studies: Subsequent to the NRC's 2006 review, over 100 animal studies investigating fluoride's neurotoxicity have been indexed in the National Library of Medicine's online database ("PubMed"). **Ex. 3A** at 10. Most of these animal studies have continued to focus on fluoride's neurochemical and cellular effects, with the overwhelming majority confirming and expanding upon NRC's conclusion that fluoride damages animal brain on the neurochemical and cellular level. *Id.* at 11.

[6] As noted above, EPA funded the ELEMENT study, and an EPA document states that archived biological samples from prospective birth cohort studies represent a "critical" means for assessing the impact of prenatal toxicant exposures. **Ex. 18** at 9; *see also* **Ex. 19** at 199:25-200:4, **Ex. 21** at 231:21-232:24.

Consistent with NRC's recommendations, a subset of these studies have also examined fluoride's "functional" effects on learning and memory. *Id.* Most of these "functional" studies have detected impaired performance in the fluoride-treated animals, confirming the *a priori* suspicion that neurochemical damage will ultimately manifest in outwardly demonstrable deficits. **Ex. 13** at 380:21-381:12.

In 2016, the National Toxicology Program (NTP) published a systematic review of the subset of animal studies that investigated fluoride's effects on learning and memory. **Ex. 12**. While the NTP identified a number of methodological limitations with the studies, the NTP concluded that that the overall evidence "suggests adverse effects on learning and memory in animal [sic] exposed to fluoride." *Id.* at vii. The evidence was strongest ("moderate level-of-confidence") in adult animals due primarily to more low-dose studies being available for adult than young animals. **Ex. 19** at 87:7-89:2, 92:3-18. According to the lead author of NTP's review, Dr. Kristina Thayer, a "moderate" level of confidence is the second highest level of confidence that NTP can have, and means "we think there's something happening here." *Id.* at 11:15-12:3, 75:9-14, 76:7-13, 248:21-249:5. One of the limitations that the NTP identified is that the studies did not rule out the possibility that fluoride-induced "motor impairments" could be the cause of the impaired test performance. *Id.* at 96:22-97:16. But motor impairments are themselves a form of neurotoxicity. *Id.* at 99:5-9. Thus, Dr. Thayer, who is now a scientist at EPA, agrees that the animal data on fluoride "supports the biological plausibility" of fluoride causing neurotoxic effects in humans. *Id.* at 164:17-165:4, 167:11-18.

For this litigation, EPA retained a toxicologist, Dr. Joyce Tsuji, to systematically review the animal data on learning and memory. At her deposition, Dr. Tsuji agreed that rodent studies consistently show adverse effects on learning/memory when the fluoride concentration in water exceeds 20 mg/L, which Dr. Tsuji admitted is the approximate human equivalent of 1.2 mg/L. **Ex. 9** at 240:20-241:8, 362:1-363:2. Dr. Tsuji also agreed that the animal studies are "virtually unanimous" in showing adverse effects at 45 mg/L, which is the human equivalent concentration of 4 mg/L (i.e., the same level as EPA's MCLG). *Id.* at 241:10-

19, 291:6-13, 367:16-368:11. Although Dr. Tsuji placed greatest weight on the findings of a recent NTP study ("McPherson 2018") which found no significant decrements in learning/memory at the human equivalent concentration of 1.2 mg/L, the NTP study found that fluoride-treated rats had significantly increased sensitivity to pain, which Dr. Tsuji admitted is an adverse neurologic effect. *Id.* at 256:19-257:21, 312:6-315:15. In addition, the NTP study found suggestive evidence that the fluoride-treated rats swam faster, which is an effect consistent with (albeit not proof of) hyperactivity. *Id.* at 317:19-319:15.

D. Absence of Safety Data

Throughout this litigation, Plaintiffs have comprehensively sought to obtain any data that demonstrates or supports the neurological *safety* of fluoride, including data that may not yet be in the public domain. Plaintiffs served discovery requests on the Centers for Disease Control; the Food & Drug Administration; the manufacturers of fluoridation chemicals in the US (Mosaic, Simplot, and Solvay);[7] and the private organization that certifies the safety of water treatment chemicals (NSF International). Connett Decl. ¶ 53. Each of these entities responded that they were not in possession of any data that would support the neurological safety of fluoride exposure. **Ex. 11** at 19:16-21, 127:14-128:16, 129:8-134:23; **Ex. 22 ¶¶** 15 & 17; **Ex. 23**; **Ex. 24** at 10:20-22, 16:17:17:1, 17:21-19:11, 59:15-61:2, 61:24-63:6, 64:17-66:8.

Plaintiffs served similar discovery requests on the EPA. As with the third-party entities, EPA admitted to being unaware of any data that supports the neurological safety of fluoride, with a few limited exceptions. **Ex. 24** at 5-17. In response to Plaintiffs' request for data supporting the neurological safety of *prenatal* fluoride exposure, EPA did identify one study—an animal study published by Mullenix, et al., in 1995. **Ex. 25** at 7. Contrary to EPA's assertion, however, the Mullenix study reported neurotoxic effects from prenatal exposure, as its own expert in this case admittted. **Ex. 6** at 107:6-108:21; **Ex. 9** at 414:3-18. In contrast to EPA's written discovery response, EPA scientist Dr. Donohue testified at her deposition that she was not aware of any data that supports the neurological safety of prenatal fluoride exposure. **Ex. 4** at

[7] Mosaic, Simplot, and Solvay are the primary manufacturers of fluoridation chemicals in the U.S. **Ex. 6** at 140:15-141:2.

275:1-9, 276:10-22, 278:13-280:12. Similarly, EPA's retained epidemiologist, Dr. Ellen Chang, admitted that every human epidemiological study which has measured prenatal fluoride exposure has found significant associations with neurodevelopmental harm in the offspring. **Ex. 27** at 213:18-214:6.

E. TSCA Risk Evaluations

Under the Toxic Substances Control Act (TSCA), Congress gave EPA the authority to prohibit, limit, or otherwise regulate the "particular use" of a chemical substance if the use poses an "unreasonable risk" to human health or the environment. 15 U.S.C. § 2605(a). In 2016, Congress amended TSCA to make clear that (1) risk evaluations under TSCA cannot consider "costs or other nonrisk factors," and (2) that an unreasonable risk to human health includes a risk that only affects a "susceptible subset" of the population. *Id.* § 2605(b)(4)(A).

i. "Non-Risk Factors," Including *Benefits*, Cannot Be Considered

The parties have each identified two experts who are prepared to offer opinions about the purported benefits of fluoridation chemicals in water. Connett Decl. ¶¶ 55-56. It has become clear in this litigation, however, that Congress's recent amendments to the TSCA statute preclude any consideration of fluoridation's benefits, or lack thereof, during the *risk* evaluation phase.

Background on Community Water Fluoridation: Fluoride is the only chemical added to drinking water that does not treat the water; [8] it treats the people drinking it. **Ex. 24** at 40:18-25. Fluoride is added to water to prevent tooth decay ("caries"). **Ex. 28** at 274:7-17. In the U.S., approximately 200 million people drink artificially fluoridated water, which is more than the rest of the world combined. *Id.* at 156:9-157:1. Yet, the U.S. still has the highest per capita costs for oral disease. **Ex. 29** at 80:4-81:18. By contrast, most of Europe has rejected water fluoridation, including Belgium, Denmark, Finland, France, Germany, Italy, Netherlands, Norway, Sweden, Switzerland, and most of Spain and the United Kingdom. **Ex. 6** 131:17-134:4; **Ex. 30** ¶ at 8; **Ex. 30A** at 8. Despite rejecting fluoridation, data from the World Health

[8] Like arsenic and lead, fluoride is found in the Earth's crust and can be naturally present at high levels in some water supplies. **Ex. 14** at 307:22-308:21.

Organization shows that Europe generally has lower rates of tooth decay today than the U.S. **Ex. 6** at 134:5-135:14; **Ex. 14** at 64:22-65:9; **Ex. 30** ¶ at 8; **Ex. 30A** at 20-22; **Ex. 31** at 218:6-219:1.

When fluoridation first began in the 1940s, it was assumed that fluoride's benefits came from *ingestion*, but this paradigm has changed. Today, it is understood that fluoride's predominant benefit comes from *topical* contact with the surface of the teeth—not from swallowing it—and that fluoride is *not* an essential nutrient. **Ex. 2** at 330:1-4, 330:11-331:4, 332:10-17; **Ex. 11** at 198:21-199:3; **Ex. 19** at 169:3-7; **Ex. 9** at 398:10-18; **Ex. 28** at 258:15-259:9, 260:5-8, 261:14-262:8, 267:23-268:3; **Ex. 30** ¶ 3; **Ex. 31** at 142:3-21; **Ex. 46** ¶ 5. There is thus no physiological need to swallow fluoride for any purpose, including caries prevention. **Ex. 6** at 98:2-10. Consistent with this, the CDC conceded in this case, as did EPA's experts, that *prenatal* exposure to fluoridated water provides no known benefits to teeth. **Ex. 11** at 19:16-21, 213:19-23, 217:3-19; **Ex. 29** at 284:11-286:18. The CDC also conceded that fluoridated water provides no known benefits to *infants* during the first six months of life (i.e., prior to tooth eruption). **Ex. 11** at 19:16-21, 224:7-11; *see also* **Ex. 9** at 403:16-404:5.

Benefits Are a Non-Risk Factor*:* The amended TSCA statute defines "costs and non-risk factors" as including "the benefits of the chemical substance," "the reasonably ascertainable economic consequences of the rule," and the effect of the proposed regulation "on public health." 15 U.S.C. § 2605(c)(2). These factors can only be considered as part of the "risk management" (i.e., rulemaking) phase, not during the risk evaluation. **Ex. 32** at 383:11-387:3. Consistent with the plain language of the statute, EPA's expert on TSCA risk evaluations, Dr. Tala Henry, testified that fluoride's "benefits for caries prevention" and "the economic costs of banning fluoridation" are issues for the "*rulemaking proceeding*," not the risk evaluation. *Id.* at 389:8-13, 390:8-391:5. Dr. Henry further testified that EPA can consider granting an exception to fluoridation (i.e., not regulating it) if it determines during the *rulemaking proceeding* that fluoridation "provides a substantial *benefit* to health" as compared to "reasonably available alternatives." *Id.* at 387:4-12, 387:19-389:7 (discussing 15 U.S.C. § 2605(g)(1)(C)). In response to

questions from EPA's attorney later in the deposition, Dr. Henry stated she didn't know how it "would even be possible" to include benefits in a *risk* evaluation, noting that the "the law is quite explicit about nonrisk factors not being considered as part of the risk evaluation." **Ex. 32** at 476:6-14, 477:3-13.

ii. Susceptible Subpopulations: Fetus, Infants, and Elderly

Under the 2016 amendments, a TSCA risk evaluation must consider susceptible subpopulations, which the statute defines as "a group of individuals within the general population identified by the Administrator who, due to either greater susceptibility or greater exposure, *may* be at greater risk than the general population of adverse health effects from exposure to a chemical substance or mixture, such as *infants*, children, *pregnant women*, workers, or the *elderly*." 15 U.S.C. § 2602(12) (emphases added).

In the context of *neurotoxicity* risk assessments, the EPA has recognized that *life stage* is a critical criterion for identifying susceptible populations, noting that "a population subgroup is susceptible if exposure occurs during a period of sensitivity." **Ex. 33** at 64-65; **Ex. 34** at 42; **Ex. 19** at 109:4-15.

Fetus: According to EPA, "[t]he developing brain is distinguished by the absence of a blood-brain barrier. The development of this barrier is a gradual process, beginning *in utero* and complete at approximately 6 months of age. Because the blood-brain barrier limits the passage of substances from blood to brain, in its absence, toxic agents can freely enter the developing brain." **Ex. 35** at 58; *see also* **Ex.** 9 at 177:18-179:11; **Ex. 14** at 70:16-71:11, **Ex. 19** at 104:4-12; **Ex. 20** at 185:4-186:8. EPA's toxicologist, Dr. Joyce Tsuji, agreed that the absence of a blood brain barrier renders the brain more vulnerable to the harm posed by neurotoxicants. **Ex.** 9 at 180:3-16. Dr. Tsuji also agreed that fluoride passes through the placenta, and gets into the fetal brain. *Id.* at 180:17-181:5. Further, based on the recent prospective studies, Dr. Tsuji agreed that the *in utero* period is a critical period of concern for fluoride neurotoxicity. *Id.* at 182:12-183:5.

Infants: EPA's 30(b)(6) representative in this litigation agreed that the addition of fluoridated water to infant formula "dramatically" increases an infant's exposure to fluoride. **Ex. 2** at 286:18-287: 1. Based on data identified by EPA, infants receiving formula made with fluoridated water ingest roughly 100 times

more fluoride than an exclusively breastfed baby. *Id.* at 257:20-258:1, 258:21-259:6, 259:12-260:18. As a result, EPA's expert, Dr. Tsuji, admitted that infants drinking formula made with fluoridated water can exceed EPA's reference dose (0.08 mg/kg/day) for severe dental fluorosis, and that this is a cause for concern. **Ex. 9** at 233:7-15; 403:16-404:5. There is no dispute, therefore, that fluoridation exposes infants to fluoride. Nor is there a dispute that infancy is a "period of sensitivity" for neurotoxicity: indeed, EPA has termed the neonatal period "a critical window of development" for the brain, because it is "a period of rapid development of the nervous system" without the protection of a fully developed blood brain barrier. **Ex. 34** at 42; **Ex. 36** at 5-4.

Elderly: In addition to the developing brain, the EPA has recognized that the elderly brain is also "at particular risk because of the limited ability of the nervous system to regenerate or compensate to neurotoxic insult." **Ex. 33** at 65. Plaintiff's risk assessment expert, Dr. Kathleen Thiessen, has identified a number of chemical-specific factors that will render the elderly more vulnerable to the neurotoxic effects of fluoride. **Ex. 3A** at 44-45. EPA's experts did not counter Dr. Thiessen's assessment, and thus the record on this issue is not in dispute.

iii. Key Principles of Risk Assessment

As with all EPA regulatory decisions on chemicals, a TSCA risk evaluation requires that a *risk assessment* be conducted. **Ex. 32** at 433:3-16.

Factual Certainty Is Not Required to Make a Risk Determination: In its seminal treatise on risk assessment (i.e., the "Red Book"), the NRC stated that "conclusive direct evidence of a threat to human health is rare," and "there are few chemicals on which the human data are definitive." **Ex. 37** at 11 & 19. The EPA recognizes this, noting that "all risk estimates are uncertain to some degree," because "in real life, information is usually limited on one or more" key issues. **Ex. 38** at 2. EPA often has to extrapolate, therefore, from high dose studies in order to assess risks to humans in the general population. **Ex 39** at 2; **Ex. 40** at 2. In light of the uncertainties involved in assessing risk, EPA does not require "conclusive proof"

to make a risk determination. **Ex. 32** at 303:4-14.

EPA's Decision to Phase Lead Out of Gasoline: When EPA made its historic, and valiant, decision to order the phase-out of lead from gasoline,[9] the EPA did not have "factual proof of actual harm." *Ethyl Corp. v. U.S. E.P.A.*, 541 F.2d 1, 8 &12 (D.C. 1976). Instead, the Agency had a basis to *infer* the existence of a "significant risk" based on the "inconclusive but suggestive results of numerous studies." *Id.* at 37-38. An *en banc* panel of the D.C. Circuit Court of Appeals held this was enough, because the existence of a danger "must be decided by assessment of risks as well as by proof of facts." *Id.* at 24. The court reasoned that "[w]here a statute is *precautionary* in nature" it would defeat the purpose of the act to require "rigorous step-by-step proof of cause and effect."[10] *Id.* at 25.

The *Ethyl Corp* decision was published in 1976, the same year that Congress enacted TSCA. As TSCA's legislative history makes clear, Congress shared a similar vision of environmental regulation as the D.C. court. **Ex. 41** at 7 & 32. The House Report explained that TSCA was "protective legislation" whose "overriding purpose is to provide protection of health and the environment through authorities which are designed to *prevent* harm." *Id.* at 7. Towards this end, the House Report explained that "factual certainty respecting the existence of an unreasonable risk of a particular harm may not be possible and *the bill does not require it*."[11] *Id.* at 32.

Default Assumptions: Since uncertainty is an inherent feature of risk assessment, the NRC recommended that EPA establish "inference guidelines" (i.e., defaults) to ensure consistency in how EPA fills in data gaps from one chemical to the next. **Ex. 37** at 7. As the NRC has explained, "without uniform guidelines, risk assessments might be manipulated on an ad hoc basis according to whether regulating a substance is thought be politically feasible." **Ex. 42** at 87 & 91. The NRC has thus recommended that EPA

[9] On the parallels between fluoride and lead, see **Ex. 14** at 158:21-160:18; **Ex. 21** at 226:7-229:1.
[10] *Id.* at 25 ("Certainty in the complexities of environmental medicine may be achievable only after the fact, when scientists have the opportunity for leisurely and isolated scrutiny of an entire mechanism.").
[11] *See also* John S. Applegate, *The Perils of Unreasonable Risk: Information, Regulatory Policy, and Toxic Substances Control*, 91 COLUM. L. REV. 261, 271–73 (1991) (describing the unreasonable risk standard as "a regulation of risk instead of actual harm").

implement default options for how it will fill data gaps, and that these defaults should be used in each risk assessment "in the absence of evidence to the contrary." **Ex. 43** at 201. The defaults "assign the burden of persuasion" to those wishing to use an alternative to the default for a given chemical; this burden of persuasion is satisfied if the alternative inference "is clearly superior" to the default. **Ex. 42** at 29; **Ex. 43** at 201.

EPA's Guidelines for Neurotoxicity Risk Assessment: In response to the NRC's recommendation to establish default guidelines, EPA created *Guidelines for Neurotoxicity Risk Assessment* (hereafter *Guidelines*) that EPA has stated it "*will* use" to "evaluate data on potential neurotoxicity associated with exposure to environmental toxicants," including in risk evaluations under TSCA. **Ex. 33** v & 1; **Ex. 32** at 250:10-251:9, 251:21-252:15. The *Guidelines* describe four steps to the risk assessment: (1) Hazard Characterization, (2) Quantitative Dose Response, (3) Exposure Assessment, and (4) Risk Characterization. **Ex. 33** at 2-3; *see also* **Ex. 37** at 3; **Ex. 39** at 1-2.

In the Hazard Characterization analysis, EPA seeks to determine if there is "sufficient evidence" to conclude that a chemical can cause neurotoxicity at some level of exposure. "Sufficient evidence" is established if, *inter alia*, epidemiological studies show that "some neurotoxic effect is *associated* with exposure." **Ex. 33 at** 53. The *Guidelines* distinguish association from *causation*, and note that "causality is a more stringent criterion than association." *Id.* The *Guidelines* further note that sufficient evidence can be established by single "well-conducted study." *Id.* at 55.

In the second step of the risk assessment, the dose-response relationship between the chemical and the hazard is analyzed to determine the dose (i.e., "reference dose") of the chemical that "is likely to be without an appreciable risk of deleterious effects during a lifetime." *Id.* at 57. To do this, the collective data is reviewed to identify a "Point of Departure" (POD), which can be a "Benchmark Dose" (BMD), "No Observed Adverse Effect Level" (NOAEL), or "Lowest Observed Adverse Effect Level" (LOAEL). *Id.*

After the Point of Departure is identified, it is divided by "uncertainty factors" to "compensate for

human variability in sensitivity, the need to extrapolate from animals to humans," and potentially other sources of uncertainty. *Id.* at 59. The *Guidelines* state that uncertainty factors "are typically multiples of 10." *Id.* EPA has stated that an uncertainty factor of 10 for intraspecies (human-to-human) variability should generally be used "in the absence of convincing data to the contrary," and that a lower uncertainty factor can "only" be justified if certain prerequisite conditions are met.[12] **Ex. 36** at 5-17; **Ex. 44** at 4-42.

Risk Characterization: The last part of a risk assessment under the *Guidelines* is called "risk characterization." In this step of the analysis, EPA compares the dose that is believed to be without appreciable risk to the doses that humans receive. The *Guidelines* recommend the "Margin of Exposure" (MOE) method for performing this analysis as it "is one of the standard approaches" that EPA uses for noncancer risk assessment. **Ex. 32** at 287:2-4, 288:5-10; **Ex. 33** at 3 & 65-66. EPA routinely uses the MOE method for pesticides, including *sodium fluoride*. **Ex. 45** at 10-11 & 17. The MOE method is the standard method that EPA uses to characterize risk under TSCA. **Ex. 32** at 267:20-268:9, 288:5-10.

A description of the MOE method has been provided by the Ninth Circuit, and other federal courts.[13] In short, EPA calculates a "Benchmark MOE" (i.e., "Acceptable MOE"), which is the ratio between the Point of Departure (i.e., BMD, NOAEL, or LOAEL) and human exposure that is deemed to pose an "acceptable" risk. **Ex. 3A** at 64-65. This "benchmark" ratio is derived by applying the uncertainty factors that are used to address intraspecies and interspecies variability. *Id.* at 65. If the "Calculated MOE" (i.e., "Actual MOE") is *less* than the Benchmark MOE, an "unacceptable risk" is presumed to exist. **Ex. 32** at 288:13-289:11. Up until 2016, if a TSCA risk evaluation identified an unacceptable risk using the MOE method, an unreasonable risk determination was made. *Id.* at 290:3-16. Today, if a TSCA risk evaluation finds an unacceptable risk using the MOE method, EPA "*may* consider other risk related factors," including the number of people exposed to the chemical, the severity of the effect, and reversibility. *Id.* at 270:14-

[12] In each of the 9 neurotoxicity risk assessments that EPA has conducted pursuant to the Guidelines, EPA has applied an uncertainty factor of 10. **Ex. 3A** at 51-52.

[13] *See, e.g.*, *Nat. Res. Def. Council v. U.S. E.P.A.*, 735 F.3d 873, 881-84 (9th Cir. 2013); *Nat. Res. Def. Council v. U.S. E.P.A.*, 658 F.3d 200, 207-09 (2d Cir. 2011).

273:11.

F. Plaintiffs Experts' Undisputed Testimony on Risk

Plaintiffs' experts have assessed the neurologic risk of fluoridation chemicals in three separate and distinct ways. As summarized herein, EPA's experts have not refuted any of these three opinions.

Dr. Bruce Lanphear: Dr. Lanphear is a medical doctor and expert on the impact of environmental chemicals on children's health. **Ex. 21** at 31:9-32:14, 211:10-15. Dr. Donohue, from EPA's Office of Water, agrees that Dr. Lanphear has done "very reliable and important research" on lead toxicity, and EPA litigation expert, Dr. Tsuji, agrees that Dr. Lanphear has published "seminal research on low-level lead and IQ." **Ex. 4** at 257:1-16; **Ex. 9** at 296:4-10. In this case, Dr. Lanphear agreed to appear as a non-retained expert and has offered the opinion that the evidence is now sufficiently definitive to recommend that pregnant women not drink fluoridated water. **Ex. 21** at 215:20-218:6. EPA's experts have not commented on this recommendation. *See, e.g.*, **Ex. 9** at 407:14-408:19.

Dr. Philippe Grandjean: Dr. Grandjean is an NIH-funded epidemiologist and specialist in the field of developmental neurotoxicity at the Harvard School of Public Health. **Ex. 16** ¶ 4. As part of his expert reports in this case, Dr. Grandjean conducted Benchmark Dose (BMD) analyses of the prospective birth cohort studies to determine the level of maternal urinary fluoride that is associated with a 1-point drop in IQ. *Id.* ¶¶ 5-8. As Dr. Grandjean explained, BMD analysis is a standard method that EPA uses to assess risk and, when applied to fluoride, this method demonstrates a substantial neurologic risk from fluoridation that rivals the effects of lead (i.e., up to 23 million lost IQ points for children aged 0 to 5). **Ex. 13** at 75:13-78:9, 96:3-14; **Ex. 16** ¶¶ 6-8; **Ex. 16A** at 40-43).

EPA's own experts (Dr. Chang and Dr. Tsuji) previously performed a nearly identical BMD analysis for *arsenic* and IQ as Dr. Grandjean has done with fluoride. As with Dr. Grandjean, EPA's experts used a prospective cohort study (from Bangladesh) to calculate the maternal urinary level of arsenic associated with loss of 1 IQ point. **Ex. 9** at 370:8-13, 372:12-17, 373:9-374:14, 377:2-13, 377:14-378:2,

379:9-381:4, 416:15-19, 417:2-5; **Ex. 16B** at 16-17; **Ex. 27** at 100:24-103:14. At her deposition, Dr. Tsuji agreed that the MIREC cohort study was just as suitable to deriving an RfD as the Bangladeshi study she and Dr. Chang used to derive an RfD for arsenic. **Ex. 9** at 383:20-384:4.

None of EPA's experts addressed Dr. Grandjean's BMD analysis in their reports. The closest that EPA's experts came to offering a critique can be found in the deposition of EPA's non-retained risk assessment scientist, Dr. Tala Henry. In her report, Dr. Henry critiqued Dr. Grandjean for using methods that are "used to derive a Maximum Contaminant Level Goal under the Safe Drinking Water Act." **Ex. 47** at 23. Yet, at her deposition, Dr. Henry admitted that EPA uses BMD analyses for evaluating risk under TSCA, and that she had forgotten Dr. Grandjean conducted a BMD analysis. **Ex. 32** at 340:16-346:4. Upon realizing her error, Dr. Henry offered the meritless criticism that Dr. Grandjean "didn't even provide the data, let alone what reference it came from." *Id.* at 347:11-12. To the contrary, Dr. Grandjean very clearly identified the source of the data (i.e., the ELEMENT and MIREC cohorts). **Ex. 16A** at 38-40; **Ex. 16B** at 15-16. This data was not only published, but was provided to EPA during discovery, and thus readily available to EPA's experts. Connett Decl. ¶ 57. Dr. Henry's impromptu criticism was thus incorrect, and Dr. Grandjean's BMD analysis stands unrefuted.

Dr. Kathleen Thiessen: Dr. Thiessen is a risk assessment scientist who has co-authored both of the NRC's reviews on fluoride toxicity since 2000, including the landmark 2006 report; she has also written health assessment reports for EPA, including on fluoride. **Ex 3** ¶ 3; **Ex. 9** at 420:21-422:14. In this case, Dr. Thiessen conducted a risk assessment pursuant to EPA's *Guidelines*. **Ex. 3** ¶ 4; **Ex. 13** at 318:5-16. Based on her assessment, Dr. Thiessen concluded *inter alia* that (1) neurotoxicity is a hazard of fluoride exposure, (2) the NOAELs and LOAELs for neurotoxicity in the animal literature produce reference doses that are, *without exception*, below current "safe" levels, and (3) EPA's Margin of Exposure (MOE) method demonstrates an unacceptable risk of neurotoxicity in fluoridated areas. **Ex. 3** ¶¶ 4, 7, 9-10.

None of EPA's experts addressed, let alone refuted, Dr. Thiessen's application of the *Guidelines*.

First, the two experts (Drs. Chang and Tsuji) EPA retained to review the fluoride neurotoxicity literature *did not do risk assessments*. **Ex. 9** at 41:12-42:3, 94:17-95:5, 321:12-13;. Instead, they conducted causal analyses in which they concluded that there is "insufficient evidence" to prove that fluoridated water at 0.7 mg/L causes neurotoxicity in humans. **Ex. 48** at 87-89; **Ex. 49** at 49-50. By "insufficient evidence," EPA's epidemiology expert Dr. Chang testified that she means "there's not enough compelling evidence to reach a *definitive* conclusion." **Ex. 27** at 125:19-126:2. Dr. Chang explained her conclusion of "insufficient evidence" is not inconsistent with the chemical being a *presumed* cause. *Id.* at 123:17-124:17, 125:9-126:17.

Second, EPA's expert on TSCA *risk* evaluations, Dr. Henry, had no criticisms to offer with respect to how Dr. Thiessen applied the *Guidelines*. The written summary of Dr. Henry's opinions does not reference Dr. Thiessen's use of the *Guidelines*, and at her deposition, Dr. Henry testified that she did not believe Dr. Thiessen even discussed the *Guidelines*—despite the fact that Dr. Thiessen's report references the Guidelines *more than 50 times*. **Ex. 32** at 260:18-263:20; **Ex. 47** at 19-22.

Third, although Dr. Henry critiqued certain aspects of Dr. Thiessen's risk characterization analysis, she offered no criticisms of Dr. Thiessen's *margin of exposure* analysis, which she admits is used for risk determination under TSCA. **Ex. 32** at 267:20-268:9, 334:15-335:6; **Ex. 47** at 21-22. At her deposition, Dr. Henry could not recall if Dr. Thiessen conducted a margin of exposure analysis, and thus had no opinions to offer. **Ex. 32** at 335:3-6.

Finally, although Dr. Tsuji's written report provides some nebulous criticisms of Dr. Thiessen's quantitative dose-response assessment (i.e., for using default uncertainty factors), Dr. Tsuji's deposition testimony makes clear that Dr. Tsuji will offer no meaningfully contrary opinion. **Ex. 49** at 46-47. First, Dr. Tsuji agreed that one of the Points of Departure that Dr. Thiessen used for her Margin of Exposure analysis (i.e., a 20 mg/L NOAEL) is a reasonable POD to use. **Ex. 9** at 259:14-21. Second, Dr. Tsuji agreed that uncertainty factors will need to be applied to derive the reference dose for fluoride. *Id.* at 331:12-18,

351:12-19. Third, Dr. Tsuji testified that she had no opinions as to what the size of the uncertainty factors should be. *Id.* at 334:18-335:6, 339:13-340:11, 345:9-11. Finally, when Dr. Thiessen used Dr. Tsuji's suggested Point of Departure (the 20 mg/L NOAEL) and applied just two uncertainty factors, the Margin of Exposure analysis showed an unacceptable risk. **Ex. 3** ¶ 9. Dr. Tsuji offered no opinions on this result.

EPA's "Systematic Review" Critique: Dr. Henry has offered the opinion that Dr. Grandjean's and Dr. Thiessen's reviews are not "credible" because they were not formal "systematic reviews." **Ex. 47** at 1-2. However, Dr. Henry testified that a risk assessment conducted pursuant to the *Guidelines*—which is what Dr. Thiessen did—would be a de facto systematic review and provide credible results.[14] **Ex. 32** at 254:4-8, 257:1-6. Further, whereas EPA's experts were not experts on fluoride prior to this case, Dr. Grandjean and Thiessen are subject matter experts and thus did not need to do a formal systematic review to identify the relevant research. **Ex. 3** ¶ 3; **Ex. 16** ¶ 4; **Ex. 9** at 15:2-5, 420:21-422:14; **Ex. 27** at 27:9-28:11; **Ex. 32** Henry at 16:14-18:14). In fact, EPA's experts admitted that Dr. Grandjean's and Dr. Thiessen's reviews *did not omit any studies* that would materially challenge their opinions. **Ex. 16** ¶ 11, **Ex. 16B** at 3-5; **Ex. 9** at 150:4-151:4; **Ex. 27** at 242:14-243:10, 246:8-247:25, 249:16-23, 268:9-19, 269:5-270:19, 270:22-272:17.

G. TSCA's Delay Clause

If Section 21 petitioners prove the existence of an unreasonable risk, a court "may permit the [EPA] to defer initiating the action requested by the petitioner until such time as the court prescribes." 15 U.S.C. § 2620(b)(4)(B)(ii). In order to do so, however, a finding must be made "that the extent of the *risk* to health or the environment alleged by the petitioner is *less than the extent of risks to health* or the environment

[14] EPA's contention that only systematic reviews can provide credible conclusions is at odds with the fact that EPA's experts on fluoride's purported benefits did not conduct systematic reviews. **Ex. 28** at 23:25-29:24; **Ex. 29** at 45:9-47:16. EPA's experts on benefits testified that narrative reviews can present reliable conclusions. **Ex. 28** at 25:16-20; **Ex. 29** at 46:6-21. In fact, were that not the case, countless of EPA's own risk assessments would need to be discarded because the Agency has only recently begun to conduct systematic reviews. **Ex. 19** at 289:6-12; **Ex. 32** at 11:18-19, 12:8-14:1, 212:7-14; 213:11-17; 236:1-8, 240:18-241:1.

with respect to which the Administrator is taking action under this chapter *and* there are insufficient resources available to the Administrator to take the action requested by the petitioner." *Id*. (emphases added).

In the event that the Court finds that fluoridation chemicals present an unreasonable risk, EPA has indicated its intent to request that the Court give the Agency "at least 4 years" to complete the rulemaking process. Ex. 50 at 5:13-15. EPA has identified one witness to support its position on this matter: Dr. Tala Henry, the Deputy Director of the Office of Pollution Prevention and Toxic Substances. *Id.* at 2:17-23. At her deposition, however, Dr. Henry repeatedly made clear that she has no basis regarding, and *no opinion*, regarding the relative risks posed by fluoridation chemicals versus the risks posed by the chemicals that EPA has prioritized for evaluation under TSCA and/or is currently evaluating. **Ex. 32** at 482:5-15, 491:17-492:2, 499:12-19, 500:10-14, 505:3-19, 506:20-507:18; *see also id.* at 24:5-30:5, 94:3-95:18.

IV. ARGUMENT

A. Summary Judgment Standard

A court shall grant summary judgment—or *partial* summary judgment on a "part of a claim of defense"—if "there is no genuine issue as to any material fact and the movant is entitled to judgment as a matter of law." Fed. R. Civ. Pro. 56(a); *Celotex Corp. v. Catrett*, 477 U.S. 317, 322-23 (1986). The movant has the burden of identifying those "portions of the materials on file that it believes demonstrate the absence of any genuine issue of material fact." *T.W. Elec. Serv., Inc. v. Pac. Elec. Contractors Ass'n*, 809 F.2d 626, 630 (9th Cir. 1987) (citing *Celotex Corp*., 477 U.S. at 323). Once the moving party has carried this burden, the nonmoving party "must set forth specific facts showing that there is a genuine issue for trial" and may not rely on mere denials. *Porter v. Cal. Dep't of Corr.*, 419 F.3d 885, 891 (9th Cir. 2005) (quoting *Anderson v. Liberty Lobby, Inc*., 477 U.S. 242, 256 (1986)). The nonmoving party will not meet its burden if these "specific facts" are general references to evidence. *S. Cal. Gas Co. v. City of Santa Ana*, 336 F.3d 885, 889 (9th Cir. 2003). "[A]t least some 'significant probative evidence"' must be produced. *T.W. Elec. Serv*., 809 F.2d at 630 (quoting *First Nat'l Bank of Ariz. v. Cities Serv. Co*., 391 U.S. 253 (1968)).

B. EPA Has Failed to Refute Plaintiffs Evidence that Fluoridation Chemicals Present an Unreasonable Risk

Plaintiffs' experts have shown that fluoridation chemicals present an unacceptable risk under EPA's own risk assessment methods, including benchmark dose analyses of human epidemiological data, and margin of exposure analyses that compare toxicity levels in animal studies with exposures in humans. **Ex. 3** ¶¶ 4, 7, 9-10; **Ex. 16** ¶¶ 5-8. It is undisputed that EPA uses risk assessment to make risk determinations under TSCA and that EPA uses the same methods that Plaintiffs' experts used. **Ex. 32** at 267:20-268:9, 346:1-4, 433:3-16. It is also undisputed that EPA recognizes that (1) uncertainty is an inherent aspect of risk assessment, (2) conclusive proof of harm is not necessary to make a risk determination, and (3) EPA often has to extrapolate findings from high dose human and animal studies in order to determine the risk to the general population. **Ex. 32** at 303:4-14; **Ex. 38** at 2; **Ex 39** at 2; **Ex. 40 a**t 2. Despite this, EPA's experts made no attempt to assess risk, and opted instead to conduct narrow causal analyses to determine if there is conclusive proof that the specific concentration of fluoride added to water (0.7 mg/L) causes neurotoxicity in humans. **Ex. 9** at 41:12-42:3, 94:17-95:5, 321:12-13; Ex. 27 at 123:17-124:17, 125:9-126:17; **Ex. 48** at 87-89; **Ex. 49 a**t 49-50. In so doing, EPA's experts abandoned essentially every basic tenet of EPA risk assessment.

The standard that EPA's experts used to determine causation (i.e., "compelling evidence to reach a definitive conclusion") is not only at odds with EPA risk assessment, it is at odds with the law. In *Ethyl Corp*, the D.C. Court of Appeals held that "factual proof of actual harm" is not a necessary predicate to a determination of "significant risk" because certainty in the field of environmental medicine is often only achievable "after the fact; when scientists have the opportunity for leisurely and isolated scrutiny of an entire mechanism." 541 F.2d at 12-13, & 25. Congress had the same vision in mind when it enacted TSCA, with the House Report stating that "factual certainty respecting the existence of an unreasonable risk of a particular harm may not be possible and the bill does not require it." **Ex. 41** at 7; *see also* Applegate, *supra* note 10, at 271 (describing the unreasonable risk standard as a "regulation of risk instead of actual harm"). Thus, even *if* EPA's experts were correct, their conclusions are ultimately immaterial, because (1) their standard of proof is incompatible with the standard set forth by EPA, Congress, and the Courts, and (2) their conclusions do not refute the risk determinations by Plaintiffs' experts.

C. Dental Health *Benefits* Are a Non-Risk Factor and Thus Cannot Be Considered in a TSCA Risk Evaluation

As EPA's expert, Dr. Tala Henry, stated during her deposition, fluoride's "benefits for caries prevention" and "the economic costs of banning fluoridation" are issues for the "rulemaking proceeding," not the risk evaluation. **Ex. 32** at 389:8-13, 390:8-391:5. Dr. Henry's interpretation of the statute is fully consistent with TSCA's command that risk evaluations not consider "costs or other nonrisk factors." 15 U.S.C. § 2605(b)(4)(A). It is also consistent with the fact that the statute specifically defines "costs and nonrisk factors" to include "the benefits of a chemical substance" and the impact of the proposed rule on "public health." 15 U.S.C. § 2605(c)(2); **Ex. 32** at 383:11-387:3.

There are additional considerations that counsel in favor of excluding dental benefits from this case. If the Court finds that fluoridation chemicals pose an unreasonable risk, the EPA has stated that it may elect to disregard the Court's command to implement a rule if it finds, during the rulemaking proceeding, that fluoridation chemicals present a "substantial benefit to health." **Ex. 50**, at 5:3-6 (citing 15 U.S.C. § 2605(g)(1)(C)); *see also* **Ex. 32.** at 387:4-12, 387:19-389:7. To the extent the Court considers benefits in the instant proceeding, a future decision by EPA to forego issuing a rule because of fluoridation's benefits would effectively render the Court's ruling into an advisory opinion. Additionally, since EPA will be considering benefits during the rulemaking proceeding, the interests of judicial economy counsel against wasting the Court's time with issues that will simply be relitigated at the administrative level. The parties have designated a total of four experts to testify on fluoride's dental benefits, and the total time of their collective testimony could be substantial. Connett Decl. ¶¶ 55-56.

D. EPA Is Not Able to Delay the Rulemaking Proceeding Under 15 U.S.C. § 2620(b)(4)(B)(ii) Because It Has No Evidence on the Relative Extent of Risks

Congress authorized the postponement of rulemaking proceedings under Section 21(b) if, and only if, two distinct predicate facts are established: (1) the extent of the risk alleged by the petitioner is less than the extent of the risk from the chemicals the Agency is already taking action on; *and* (2) there are

"insufficient resources" available to EPA to carry out both activities at once. 15 U.S.C. § 2620(b)(4)(B)(ii). EPA identified one witness, Dr. Henry, to establish these predicate findings. However, as Dr. Henry's testimony makes abundantly clear, she has no opinion and no competent basis to establish the predicate finding on relative risk. **Ex. 32** at 482:5-15, 491:17-492:2, 499:12-19, 500:10-14, 505:3-19, 506:20-507:18; *see also id.* at 24:5-30:5, 94:3-95:18. When asked whether she would be offering any opinion on the extent of the risks posed by the 10 chemicals EPA is currently evaluating under TSCA, Dr. Henry replied with a one-word answer, "No." *Id.* at 482:5-15. When asked if she would be offering any opinions on the risks posed by the 90 chemicals that EPA has designated for consideration under TSCA, Dr. Henry again replied, "No." *Id.* at 491:17-492:2. Dr. Henry's admissions make clear that EPA will be incapable of establishing its entitlement to a delay under Section 21(b).

V. CONCLUSION

Congress enacted Section 21 to "ensure that bureaucratic lethargy does not prevent the appropriate administration of [TSCA's] vital authority." *Environmental Defense Fund v. Reilly*, 909 F.2d 1497, 1499 (D.C. Cir. 1990). This is precisely why Plaintiffs filed their Citizen Petition, because EPA has failed to protect the public from the dangers posed by fluoride in drinking water. EPA's current "safe" drinking water standard for fluoride is so high that its own scientists sought to file an amicus brief to support an environmental organization's challenge to it. **Ex. 6** at 79:20-81:12, 81:25-82:8. EPA's current lead scientist on fluoride admits that EPA's fluoride standard is "out of date," and the National Research Council told the EPA 13 years ago that this standard is unsafe and needs to be lowed. **Ex. 4** at 88:7-10, 90:15-21. And yet EPA has done nothing. *Id.* at 88:2-6. It was thus left to citizen groups to compel the Agency to take action by exercising the authority that Congress invested in the citizenry for this very purpose. Now, after two years of litigation, Plaintiffs respectfully request—for the reasons stated above—that the Court grant their Motion for Summary Judgment and/or Partial Motion for Summary Judgment.

Dated: October 9, 2019

Respectfully submitted,

/s/ Michael Connett .
MICHAEL CONNETT

CERTIFICATE OF SERVICE

I hereby certify that a true and correct copy of the foregoing was served by Notice of Electronic Filing this 9th day of October, 2019, upon all ECF registered counsel of record using the Court's CM/ECF system.

/s/ Michael Connett .
MICHAEL CONNETT