

# Memorandum



CITY OF DALLAS

DATE December 1, 2023  
TO Honorable Committee Chair Bazaldua and Members of the Quality of Life, Arts and Culture Committee  
SUBJECT **October 16 and November 7, 2023 - Quality of Life, Arts and Culture Committee Briefing - Community Water Fluoridation in Dallas, Texas Follow Up**

The Quality of Life, Arts and Culture Committee has heard from two speaker panels on the topic of community water fluoridation. The following information and attachments are provided in response to questions asked by the Committee during the panel discussions.

## **What are the levels of fluoride in our raw water and tap water?**

Please see Attachment A.

## **What raw water sources does Dallas pull from?**

The City of Dallas draws water from six surface water reservoirs. The East Side Water Treatment Plant, located in Sunnyvale, Texas draws water from Lakes Hubbard, Tawakoni, and Fork while the Elm Fork and Bachman Water Treatment Plants receive water from Lakes Ray Roberts, Lewisville, and Grapevine via the Elm Fork of the Trinity River.

## **Do we have hard water?**

Yes. Dallas' water hardness ranges from 120 to 155 mg/L as CaCO<sub>3</sub>. For your reference, please see the table below on hardness ratings.

### WATER HARDNESS RATING

mg/L as CaCO <sub>3</sub>	Degree of Hardness
0-60	Soft water
61-120	Moderately hard water
121-180	Hard water
>180	Very hard water

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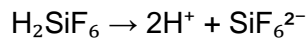
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### **What was the language used in the 1966 special election proposition?**

Please see Attachment B – Special Election Canvassing Report dated January 31, 1966.

### **What is the chemical make-up of the fluoride used by Dallas? When it is dissolved in water, what compounds are released?**

Dallas utilizes hydrofluorosilicic acid (HFS) to fluoridate the drinking water. HFS is one of only three products approved for water fluoridation. HFS is represented by the chemical formula  $H_2SiF_6$ . It is derived from naturally occurring minerals and is a compound consisting of hydrogen, silicon, and fluorine atoms. When Hydrofluorosilicic acid is added to water, it readily breaks down into its component ions. Specifically -



When the  $SiF_6^{2-}$  ion further dissociates in water, it releases fluoride ions. Under typical water pH levels, the silicate part (from the hexafluorosilicate ion) hydrolyzes, and forms hydrated silica. These fluoride ions are left and are responsible for the dental health benefits.

### **Why does Dallas maintain a 0.7 mg/L level?**

The recommended fluoride level for water utilities is typically established by the appropriate state regulatory or health agency. In the absence of an established state limit, the U.S. Department of Health and Human Services (USHHS) recommends a level of 0.7 mg/L.

Please see Attachment C – U.S. Public Health Service Recommendation for Fluoride Concentration in Drinking Water for the Prevention of Dental Caries.

### **Why does 73% of the U.S. population on public water systems have access to fluoridated water?**

[There are a total of 51,373 community water systems in the United States, of which only 9% provide water to over 83% of the population.](#) This means a small number of large community water systems are responsible for serving a large portion of the U.S. population. With the majority of the top 50 U.S. cities fluoridating their water supply or naturally receiving fluoride in their water, the United States has been able to provide a significant portion the population with a fluoridated supply of drinking water.

Also contributing to the number of systems participating in community water fluoridation is 13 states with established requirements for fluoridation of public water supplies including Connecticut, Kentucky, Illinois, Minnesota, Ohio, South Dakota, Georgia, Nebraska, California,

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Delaware, Nevada, Louisiana, and Arkansas. (Source: [Fluoride Legislative User Information Database](#)). Additionally, no State prohibit community water fluoridation.

### **What was the revision process for lowering the recommendation in community water fluoridation levels?**

Please refer to Attachment C – U.S. Public Health Service Recommendation for Fluoride Concentration in Drinking Water for the Prevention of Dental Caries, specifically pages 5 – 7. The attachment explains the process by which the US Department of Human and Health Services and the US Environmental Protection Agency conducted their review outlining their considerations for lowering the recommendation level for community water fluoridation.

### **Of the 50 largest cities, some don't fluoridate their water, how are they able to do so?**

During the briefing, Council members listed a handful of cities not fluoridating their drinking water. This list included Portland, Oregon, San Jose, California, Fresno, California, Albuquerque, New Mexico, Tucson, Arizona and Wichita, Kansas. Below are our findings -

A 2020 [Oregon Public Broadcasting report](#) explained that Portlanders have repeatedly rejected measures to allow fluoride to be added to its water, most recently in 2013. Similarly, residents of [Wichita, Kansas](#) have also repeatedly rejected measures to allow fluoride to be added to is water, most recently in 2012. In both instances, the Cities were able to remain non-fluoridated by a vote.

[A 2015 news article](#) reported that a group of dentists was able to persuade the Tucson City Council in 1992 to fluoridate the local water supply in conjunction with the introduction of Central Arizona Project water. But the fluoridation never happened. Most Tucson Water users — 709,000 people — receive water that averages 0.4 milligrams per liter of naturally occurring fluoride. According to the news article, adding fluoride to Tucson's water now would require a directive from the mayor and council.

According to their [frequently asked questions on their website](#), the City of Fresno no longer fluoridates the water that is delivered to customers.

In [Albuquerque, New Mexico](#), supplemental fluoridation of the City's drinking water was resumed on June 27, 2018. Supplemental fluoridation was the norm in Albuquerque from 1972 until 2011, when the Water Authority suspended the practice pending issuance of new federal recommendations on optimal fluoride levels. At the urging of the New Mexico Department of Health and the New Mexico Dental Association, the Water Authority chose to resume supplemental fluoridation for the sake of the community's dental health.

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According to the [San Jose Water \(SJW\) Website](#), San Jose residents receives water from various water providers, based on location. Some, but not all the water providers deliver a fluoridated supply of water. Parts of San Jose have received fluoridated water since the early 1960s. In 2005, another water provider began fluoridating the water supply and in 2016 another water provider did the same.

The final remaining provider currently blends non-fluoridated treated water and non-fluoridated groundwater. As a result, some customers periodically receive water with fluoride levels slightly below the recommended range. This provider is expected to deliver fluoridated water from all three of its water treatment facilities by 2027. When that occurs, a large majority of San Jose Water's service area will receive a fluoridated water supply.

This action is in compliance with California law (Section 116409-116415 of the California Public Health and Safety Code) which requires water systems to fluoridate their supply if funding for capital and ongoing operation and maintenance of the system is provided by a source other than the utility or its customers. The Santa Clara County Public Health Officer, the Health Trust, and the California Dental Association are providing the necessary funding and to comply with all drinking water laws and regulations, SJW is fluoridating their water supply.

**What specific pediatric dental programs would need to be in place to diminish/eliminate the need for community water fluoridation?**

The Centers for Disease Control and Prevention provide the following regarding oral health practices for children and adults.

[Children's Oral Health](#) –

What Parents and Caregivers Can Do for Babies -

- Wipe gums twice a day with a soft, clean cloth in the morning after the first feeding and right before bed to wipe away bacteria and sugars that can cause cavities.
- When teeth come in, start brushing twice a day with a soft, small-bristled toothbrush and plain water.
- Visit the dentist by your baby's first birthday to spot signs of problems early.
- Talk to your dentist or doctor about putting fluoride varnish on your child's teeth as soon as the first tooth appears.
- For children younger than 2, consult first with your doctor or dentist regarding the use of fluoride toothpaste.

What Parents and Caregivers Can Do for Children -

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- Brush their teeth twice a day with fluoride toothpaste.
- Help your child brush their teeth until they have good brushing skills.
- If your child is younger than 6, watch them brush. Make sure they use a pea-sized amount of toothpaste and always spit it out rather than swallow.
- Ask your child's dentist to apply dental sealants when appropriate.
- Drink tap water that contains fluoride.

#### Adult Oral Health -

- Drink fluoridated water and brush with fluoride toothpaste.
- Practice good oral hygiene. Brush teeth thoroughly twice a day and floss daily between the teeth to remove dental plaque.
- Visit your dentist at least once a year, even if you have no natural teeth or have dentures.
- Do not use any tobacco products. If you smoke, quit.
- Limit alcoholic drinks.
- If you have diabetes, work to maintain control of the disease. This will decrease risk for other complications, including gum disease. Treating gum disease may help lower your blood sugar level.
- If your medication causes dry mouth, ask your doctor for a different medication that may not cause this condition. If dry mouth cannot be avoided, drink plenty of water, chew sugarless gum, and avoid tobacco products and alcohol.
- See your doctor or a dentist if you have sudden changes in taste and smell.
- When acting as a caregiver, help older individuals brush and floss their teeth if they are not able to perform these activities independently.

#### **What alternative products are available for use in community water fluoridation?**

Only three (3) products are approved for adjusting fluoride in drinking water –

- Sodium Fluoride
- Sodium Fluorosilicate
- Fluorosilicic Acid

These are the only products that have American Water Works Association (AWWA) Standards published in conjunction with the National Sanitation Foundation (NSF) International and American National Standards Institute (ANSI) showcasing applicability for use in drinking water treatment.

US Pharmacopeia (USP) prepares standards for products used in pharmaceuticals. USP grade sodium fluoride is only tested for nonspecific heavy metals and has no criteria for arsenic content

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or radiological exposure. USP does not provide certification of quality by an independent third-party credentialing entity to established quality verification. USP also does not have a standard for sodium fluorosilicate or fluorosilicic acid.

On a final note, additional informational provided by the panelists in relation to the briefing discussion have been included as Attachment D.

As a reminder, the State of Texas has designated Dallas a "Superior Public Water System," the highest rating given by the state and continues to provide safe, dependable drinking water that meets or exceeds all regulatory drinking water standards.

If you have questions, please contact me or Sarah Standifer, Director (I) of Dallas Water Utilities at [sarah.standifer@dallas.gov](mailto:sarah.standifer@dallas.gov).



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Biliera Johnson, City Secretary  
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Jack Ireland, Chief Financial Officer  
Genesis D. Gavino, Chief of Staff to the City Manager  
Sarah Standifer, Director (I), Dallas Water Utilities  
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# ATTACHMENT A

**Fluoride Analysis  
FY21 - FY23**

Date	ESWTP		BWTP		EFWTP	
	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)
10/1/2021	0.33	0.70	0.28	0.69	0.28	0.64
10/2/2021	0.27	0.69	0.29	0.71	0.29	0.67
10/3/2021	0.26	0.65	0.27	0.70	0.30	0.68
10/4/2021	0.30	0.67	0.25	0.70	0.29	0.65
10/5/2021	0.30	0.71	0.26	0.70	0.29	0.66
10/6/2021	0.31	0.70	0.28	0.73	0.30	0.65
10/7/2021	0.32	0.70	0.28	0.70	0.28	0.67
10/8/2021	0.37	0.67	0.26	0.70	0.29	0.69
10/9/2021	0.31	0.69	0.30	0.71	0.27	0.69
10/10/2021	0.27	0.67	0.29	0.71	0.28	0.66
10/11/2021	0.27	0.69	0.28	0.71	0.30	0.65
10/12/2021	0.27	0.72	0.29	0.70	0.32	0.73
10/13/2021	0.25	0.70	0.39	0.71	0.28	0.56
10/14/2021	0.26	0.70	0.26	0.69	0.27	0.59
10/15/2021	0.26	0.67	0.26	0.70	0.29	0.68
10/16/2021	0.28	0.68	0.37	0.73	0.29	0.29
10/17/2021	0.28	0.67	0.27	0.71	0.28	0.70
10/18/2021	0.28	0.64	0.26	0.70	0.31	0.78
10/19/2021	0.30	0.67	0.28	0.69	XX	XX
10/20/2021	0.33	0.67	0.25	0.71	XX	XX
10/21/2021	0.31	0.65	0.25	0.71	0.30	0.65
10/22/2021	0.31	0.64	0.28	0.71	0.35	0.70
10/23/2021	0.27	0.66	0.25	0.45	0.35	0.59
10/24/2021	0.22	0.63	0.28	0.70	0.30	0.51
10/25/2021	0.16	0.59	0.34	0.72	0.32	0.62
10/26/2021	0.12	0.52	0.56	0.70	0.31	0.65
10/27/2021	0.14	0.68	0.32	0.56	0.32	0.60
10/28/2021	0.14	0.71	0.24	0.70	0.27	0.50
10/29/2021	0.13	0.64	0.27	0.71	0.31	0.57
10/30/2021	0.15	0.63	0.29	0.71	0.33	0.57
10/31/2021	0.14	0.65	0.26	0.68	0.30	0.53
11/1/2021	0.13	0.65	0.27	0.73	0.32	0.67
11/2/2021	0.15	0.71	0.27	0.70	0.33	0.65



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FY21 - FY23**

Date	ESWTP		BWTP		EFWTP	
	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)
11/3/2021	0.15	0.69	0.26	0.71	0.33	0.61
11/4/2021	0.14	0.66	0.22	0.70	0.30	0.63
11/5/2021	0.13	0.68	0.23	0.69	0.28	0.59
11/6/2021	0.14	0.66	0.26	0.70	0.30	0.62
11/7/2021	0.12	0.66	0.25	0.70	0.31	0.63
11/8/2021	0.13	0.62	0.24	0.71	0.31	0.64
11/9/2021	0.13	0.65	0.23	0.70	0.31	0.66
11/10/2021	0.30	0.76	0.24	0.70	0.34	0.64
11/11/2021	0.24	0.66	0.21	0.75	0.29	0.63
11/12/2021	0.25	0.63	0.23	0.70	0.30	0.60
11/13/2021	0.27	0.63	0.23	0.70	0.30	0.58
11/14/2021	0.26	0.60	0.23	0.70	0.33	0.57
11/15/2021	0.22	0.62	0.23	0.63	0.35	0.72
11/16/2021	0.25	0.59	0.22	0.69	0.30	0.69
11/17/2021	0.25	0.65	0.22	0.77	0.32	0.63
11/18/2021	0.22	0.67	0.26	0.73	0.33	0.55
11/19/2021	0.25	0.65	0.31	0.70	0.34	0.59
11/20/2021	0.25	0.65	0.26	0.70	0.32	0.59
11/21/2021	0.24	0.68	0.26	0.69	0.33	0.59
11/22/2021	0.24	0.62	0.29	0.71	0.33	0.57
11/23/2021	0.30	0.65	0.28	0.71	0.31	0.61
11/24/2021	0.31	0.70	0.26	0.70	0.30	0.62
11/25/2021	0.28	0.66	0.28	0.71	0.32	0.62
11/26/2021	0.28	0.66	0.25	0.71	0.32	0.67
11/27/2021	0.28	0.67	0.28	0.70	0.35	0.63
11/28/2021	0.29	0.64	0.27	0.72	0.34	0.64
11/29/2021	0.32	0.65	0.25	0.73	0.35	0.68
11/30/2021	0.38	0.77	XX	XX	0.32	0.59
12/1/2021	0.32	0.71	XX	XX	0.33	0.57
12/2/2021	0.33	0.65	XX	XX	0.32	0.61
12/3/2021	0.31	0.65	XX	XX	0.36	0.72
12/4/2021	0.30	0.64	XX	XX	0.34	0.62
12/5/2021	0.28	0.65	XX	XX	0.31	0.53

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FY21 - FY23**

Date	ESWTP		BWTP		EFWTP	
	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)
12/6/2021	0.30	0.68	XX	XX	0.30	0.52
12/7/2021	0.32	0.68	XX	XX	0.34	0.60
12/8/2021	0.37	0.71	XX	XX	0.32	0.57
12/9/2021	0.37	0.72	XX	XX	0.30	0.59
12/10/2021	0.38	0.73	XX	XX	0.28	0.56
12/11/2021	0.32	0.71	XX	XX	0.32	0.59
12/12/2021	0.34	0.71	XX	XX	0.33	0.58
12/13/2021	0.31	0.66	XX	XX	0.34	0.72
12/14/2021	0.38	0.73	XX	XX	0.32	0.66
12/15/2021	0.37	0.75	XX	XX	0.33	0.72
12/16/2021	0.32	0.67	XX	XX	0.31	0.64
12/17/2021	0.33	0.71	XX	XX	0.31	0.58
12/18/2021	0.33	0.69	XX	XX	0.33	0.61
12/19/2021	0.25	0.67	XX	XX	0.38	0.62
12/20/2021	0.32	0.65	0.25	0.59	0.35	0.61
12/21/2021	0.32	0.65	0.28	0.68	0.33	0.53
12/22/2021	0.34	0.66	0.30	0.75	0.32	0.58
12/23/2021	0.36	0.66	0.28	0.68	0.32	0.67
12/24/2021	0.30	0.70	0.28	0.70	0.32	0.69
12/25/2021	0.32	0.66	0.30	0.72	0.31	0.65
12/26/2021	0.31	0.63	0.29	0.72	0.31	0.62
12/27/2021	0.32	0.67	0.28	0.70	0.32	0.72
12/28/2021	0.31	0.65	0.30	0.73	0.32	0.69
12/29/2021	0.33	0.71	0.30	0.75	0.33	0.72
12/30/2021	0.33	0.67	0.27	0.71	0.37	0.66
12/31/2021	0.27	0.68	0.29	0.69	0.34	0.63
1/1/2022	0.30	0.66	0.29	0.70	0.35	0.63
1/2/2022	0.36	0.63	0.31	0.72	0.39	0.66
1/3/2022	0.36	0.70	0.29	0.69	0.38	0.63
1/4/2022	0.34	0.74	0.29	0.68	0.39	0.56
1/5/2022	0.36	0.78	0.29	0.70	0.36	0.35
1/6/2022	0.38	0.80	0.29	0.71	0.38	0.65
1/7/2022	0.36	0.78	0.31	0.71	0.37	0.70

**Fluoride Analysis  
FY21 - FY23**

Date	ESWTP		BWTP		EFWTP	
	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)
1/8/2022	0.32	0.72	0.29	0.69	0.36	0.64
1/9/2022	0.35	0.78	0.28	0.72	0.33	0.59
1/10/2022	0.30	0.78	0.29	0.72	0.35	0.66
1/11/2022	XX	XX	0.27	0.70	0.34	0.64
1/12/2022	XX	XX	0.32	0.70	0.35	0.64
1/13/2022	0.39	0.67	0.29	0.71	0.32	0.59
1/14/2022	0.37	0.72	0.28	0.72	0.30	0.53
1/15/2022	0.34	0.70	0.32	0.70	0.31	0.61
1/16/2022	0.35	0.69	0.29	0.71	0.33	0.64
1/17/2022	0.35	0.73	0.27	0.71	0.33	0.60
1/18/2022	0.35	0.66	0.28	0.71	0.35	0.60
1/19/2022	0.39	0.56	0.29	0.70	0.30	0.62
1/20/2022	0.39	0.73	0.28	0.71	XX	XX
1/21/2022	0.29	0.68	0.28	0.71	0.38	0.66
1/22/2022	0.33	0.59	0.32	0.70	0.34	0.63
1/23/2022	0.36	0.72	0.31	0.70	0.36	0.56
1/24/2022	0.35	0.71	0.30	0.71	0.32	0.52
1/25/2022	0.40	0.76	0.36	0.71	0.34	0.53
1/26/2022	0.41	0.73	0.30	0.67	0.36	0.58
1/27/2022	0.45	0.77	0.29	0.72	0.33	0.59
1/28/2022	0.44	0.78	0.31	0.71	0.36	0.62
1/29/2022	0.42	0.77	0.32	0.72	0.38	0.66
1/30/2022	0.39	0.72	0.32	0.72	0.39	0.69
1/31/2022	0.41	0.75	0.30	0.72	0.38	0.62
2/1/2022	0.45	0.78	0.32	0.70	0.38	0.68
2/2/2022	0.42	0.75	0.31	0.70	0.38	0.62
2/3/2022	0.43	0.73	0.30	0.69	0.38	0.49
2/4/2022	0.38	0.69	0.29	0.70	0.35	0.54
2/5/2022	0.35	0.73	0.30	0.71	0.38	0.61
2/6/2022	0.35	0.60	0.32	0.71	0.39	0.63
2/7/2022	0.38	0.72	0.32	0.72	0.43	0.62
2/8/2022	0.34	0.72	0.28	0.70	XX	XX
2/9/2022	0.40	0.71	0.28	0.70	XX	XX

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FY21 - FY23**

Date	ESWTP		BWTP		EFWTP	
	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)
2/10/2022	0.43	0.74	0.29	0.71	XX	XX
2/11/2022	0.40	0.76	0.29	0.70	XX	XX
2/12/2022	0.30	0.74	0.31	0.70	XX	XX
2/13/2022	0.34	0.70	0.29	0.70	XX	XX
2/14/2022	0.30	0.71	0.30	0.69	XX	XX
2/15/2022	0.37	0.71	0.29	0.73	XX	XX
2/16/2022	0.38	0.61	0.28	0.72	XX	XX
2/17/2022	0.40	0.67	0.33	0.70	XX	XX
2/18/2022	0.35	0.77	0.33	0.70	XX	XX
2/19/2022	0.40	0.78	0.32	0.71	XX	XX
2/20/2022	0.42	0.78	0.31	0.71	XX	XX
2/21/2022	0.47	0.84	0.31	0.71	XX	XX
2/22/2022	0.43	0.81	0.32	0.71	XX	XX
2/23/2022	0.49	0.76	0.33	0.70	XX	XX
2/24/2022	0.44	0.69	0.31	0.70	XX	XX
2/25/2022	0.43	0.66	0.30	0.72	XX	XX
2/26/2022	0.40	0.67	0.31	0.70	XX	XX
2/27/2022	0.41	0.64	0.31	0.70	XX	XX
2/28/2022	0.45	0.67	0.27	0.71	XX	XX
3/1/2022	0.42	0.64	0.27	0.71	0.42	0.48
3/2/2022	0.33	0.66	0.28	0.71	0.40	0.54
3/3/2022	0.40	0.59	0.25	0.70	0.35	0.68
3/4/2022	0.43	0.68	0.28	0.70	0.33	0.67
3/5/2022	0.41	0.64	0.30	0.70	0.32	0.65
3/6/2022	0.42	0.70	0.29	0.69	0.31	0.59
3/7/2022	0.29	0.61	0.36	0.70	0.34	0.62
3/8/2022	0.35	0.66	0.29	0.70	0.38	0.71
3/9/2022	XX	XX	0.29	0.70	0.34	0.57
3/10/2022	XX	XX	0.27	0.70	0.33	0.57
3/11/2022	0.53	0.67	0.31	0.68	0.37	0.58
3/12/2022	0.37	0.67	0.33	0.72	0.39	0.90
3/13/2022	0.40	0.65	0.31	0.71	0.38	0.50
3/14/2022	0.34	0.69	0.29	0.71	0.34	0.71

**Fluoride Analysis  
FY21 - FY23**

Date	ESWTP		BWTP		EFWTP	
	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)
3/15/2022	0.34	0.65	0.28	0.70	XX	XX
3/16/2022	0.37	0.64	0.29	0.71	XX	XX
3/17/2022	0.36	0.66	0.29	0.70	XX	XX
3/18/2022	0.37	0.66	0.27	0.69	0.33	0.53
3/19/2022	0.37	0.70	0.30	0.70	0.34	0.52
3/20/2022	0.36	0.62	0.29	0.71	0.33	0.50
3/21/2022	0.44	0.69	0.31	0.70	0.33	0.64
3/22/2022	XX	XX	0.28	0.71	0.33	0.52
3/23/2022	0.40	0.69	0.26	0.69	0.36	0.79
3/24/2022	0.40	0.65	0.29	0.70	0.36	0.67
3/25/2022	0.44	0.73	0.27	0.71	0.36	0.60
3/26/2022	0.40	0.69	0.34	0.71	0.35	0.65
3/27/2022	0.39	0.67	0.29	0.73	0.34	0.59
3/28/2022	0.38	0.69	0.27	0.70	0.33	0.60
3/29/2022	0.38	0.73	0.29	0.70	0.32	0.54
3/30/2022	0.28	0.67	0.24	0.70	0.30	0.55
3/31/2022	0.25	0.55	0.26	0.70	0.32	0.62
4/1/2022	0.38	0.71	0.26	0.68	0.37	0.73
4/2/2022	0.38	0.69	0.29	0.70	0.32	0.50
4/3/2022	0.36	0.65	0.28	0.71	0.31	0.61
4/4/2022	0.37	0.63	0.26	0.70	0.30	0.52
4/5/2022	0.39	0.64	0.21	0.69	0.26	0.56
4/6/2022	0.41	0.71	0.22	0.71	0.26	0.54
4/7/2022	0.38	0.69	0.21	0.70	0.36	0.64
4/8/2022	0.40	0.69	0.22	0.69	0.32	0.53
4/9/2022	0.39	0.74	0.24	0.70	0.32	0.65
4/10/2022	0.40	0.72	0.25	0.71	0.30	0.59
4/11/2022	0.41	0.66	0.22	0.71	0.30	0.60
4/12/2022	0.42	0.67	0.21	0.71	0.29	0.54
4/13/2022	0.38	0.67	0.24	0.72	0.29	0.57
4/14/2022	0.40	0.71	0.25	0.72	0.33	0.66
4/15/2022	0.40	0.66	0.26	0.71	0.34	0.64
4/16/2022	0.38	0.67	0.26	0.71	0.31	0.62

**Fluoride Analysis  
FY21 - FY23**

Date	ESWTP		BWTP		EFWTP	
	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)
4/17/2022	0.39	0.65	0.25	0.72	0.34	0.65
4/18/2022	0.42	0.70	0.25	0.71	0.33	0.65
4/19/2022	0.34	0.69	0.26	0.71	0.32	0.62
4/20/2022	0.34	0.69	0.27	0.60	0.33	0.60
4/21/2022	0.32	0.65	0.24	0.71	0.32	0.64
4/22/2022	0.33	0.72	0.26	0.70	0.31	0.67
4/23/2022	0.42	0.72	0.23	0.70	0.31	0.68
4/24/2022	0.40	0.74	0.27	0.71	0.33	0.62
4/25/2022	0.38	0.70	0.25	0.67	0.31	0.57
4/26/2022	0.22	0.57	0.25	0.73	0.30	0.66
4/27/2022	0.24	0.55	0.25	0.70	0.33	0.62
4/28/2022	0.26	0.58	0.22	0.70	0.34	0.67
4/29/2022	0.27	0.64	0.26	0.70	0.32	0.63
4/30/2022	0.27	0.62	0.27	0.71	0.32	0.63
5/1/2022	0.23	0.54	0.26	0.71	0.34	0.71
5/2/2022	0.24	0.57	0.24	0.69	0.30	0.58
5/3/2022	0.26	0.56	0.27	0.70	0.31	0.60
5/4/2022	0.27	0.58	0.24	0.72	0.30	0.54
5/5/2022	0.27	0.60	0.25	0.70	0.30	0.69
5/6/2022	0.33	0.60	0.27	0.71	0.33	0.73
5/7/2022	0.31	0.61	0.26	0.69	0.31	0.64
5/8/2022	0.30	0.63	0.25	0.70	0.32	0.66
5/9/2022	0.35	0.63	0.25	0.71	0.32	0.66
5/10/2022	0.34	0.65	0.28	0.72	0.30	0.54
5/11/2022	0.27	0.64	0.26	0.72	0.30	0.59
5/12/2022	0.30	0.63	0.25	0.71	0.30	0.52
5/13/2022	0.22	0.61	0.28	0.71	0.28	0.56
5/14/2022	0.26	0.62	0.25	0.70	0.25	0.55
5/15/2022	0.25	0.58	0.26	0.70	0.25	0.53
5/16/2022	0.20	0.58	0.25	0.70	0.31	0.62
5/17/2022	0.28	0.55	0.25	0.71	0.26	0.55
5/18/2022	0.27	0.58	0.26	0.71	0.27	0.74
5/19/2022	0.26	0.57	0.24	0.71	0.25	0.52

**Fluoride Analysis  
FY21 - FY23**

Date	ESWTP		BWTP		EFWTP	
	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)
5/20/2022	0.22	0.57	0.26	0.70	0.26	0.62
5/21/2022	0.24	0.56	0.26	0.70	0.32	0.78
5/22/2022	0.20	0.58	0.26	0.69	0.31	0.78
5/23/2022	0.19	0.50	0.25	0.69	0.29	0.77
5/24/2022	0.19	0.59	0.27	0.70	0.30	0.73
5/25/2022	0.23	0.68	0.25	0.69	0.28	0.67
5/26/2022	0.23	0.72	0.26	0.72	0.27	0.65
5/27/2022	0.21	0.67	0.25	0.71	0.26	0.60
5/28/2022	0.32	0.71	0.26	0.71	0.26	0.62
5/29/2022	0.20	0.64	0.25	0.70	0.25	0.65
5/30/2022	0.24	0.65	0.23	0.72	0.26	0.63
5/31/2022	0.29	0.65	0.28	0.71	0.26	0.61
6/1/2022	0.33	0.74	0.27	0.70	0.26	0.65
6/2/2022	0.29	0.71	0.24	0.70	0.25	0.60
6/3/2022	0.31	0.65	0.21	0.71	0.26	0.59
6/4/2022	0.31	0.67	0.24	0.70	0.28	0.71
6/5/2022	0.30	0.66	0.25	0.72	0.28	0.76
6/6/2022	0.30	0.71	0.26	0.71	0.28	0.81
6/7/2022	0.34	0.48	0.25	0.72	0.25	0.73
6/8/2022	0.29	0.50	0.24	0.72	0.25	0.58
6/9/2022	0.27	0.70	0.26	0.72	0.24	0.69
6/10/2022	0.23	0.68	0.25	0.71	0.27	0.51
6/11/2022	0.33	0.67	0.27	0.71	0.25	0.62
6/12/2022	0.33	0.64	0.26	0.72	0.26	0.56
6/13/2022	0.29	0.62	0.24	0.71	0.24	0.54
6/14/2022	0.27	0.56	0.27	0.70	0.25	0.61
6/15/2022	0.26	0.57	0.26	0.69	0.24	0.50
6/16/2022	0.31	0.68	0.25	0.70	0.27	0.80
6/17/2022	0.30	0.71	0.26	0.72	0.26	0.60
6/18/2022	0.32	0.61	0.24	0.71	0.25	0.63
6/19/2022	0.26	0.70	0.26	0.71	0.28	0.65
6/20/2022	0.32	0.66	0.26	0.71	0.27	0.69
6/21/2022	0.31	0.63	0.27	0.71	0.24	0.45

**Fluoride Analysis  
FY21 - FY23**

Date	ESWTP		BWTP		EFWTP	
	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)
6/22/2022	0.31	0.69	0.26	0.71	0.24	0.53
6/23/2022	0.32	0.76	0.27	0.70	0.23	0.58
6/24/2022	0.33	0.83	0.25	0.71	0.25	0.62
6/25/2022	0.33	0.76	0.26	0.71	0.27	0.64
6/26/2022	0.29	0.57	0.25	0.71	0.29	0.85
6/27/2022	0.30	0.55	0.27	0.71	0.33	0.73
6/28/2022	0.31	0.59	0.26	0.70	0.31	0.74
6/29/2022	0.32	0.54	0.29	0.71	0.33	0.72
6/30/2022	0.31	0.39	0.28	0.70	0.32	0.68
7/1/2022	0.30	0.42	0.29	0.70	0.36	0.71
7/2/2022	0.36	0.34	0.26	0.71	0.31	0.54
7/3/2022	0.33	0.35	0.26	0.71	0.32	0.65
7/4/2022	0.33	0.34	0.26	0.71	0.33	0.66
7/5/2022	0.30	0.33	0.27	0.71	0.32	0.53
7/6/2022	0.31	0.34	0.26	0.71	0.35	0.40
7/7/2022	0.34	0.39	0.30	0.45	0.32	0.36
7/8/2022	0.37	0.39	0.27	0.27	0.31	0.34
7/9/2022	0.36	0.39	0.26	0.27	0.30	0.33
7/10/2022	0.28	0.39	0.25	0.27	0.33	0.31
7/11/2022	0.29	0.34	0.23	0.27	0.33	0.35
7/12/2022	0.32	0.33	0.25	0.27	0.31	0.30
7/13/2022	0.30	0.34	0.23	0.27	0.31	0.31
7/14/2022	0.37	0.40	0.27	0.27	0.49	0.33
7/15/2022	0.37	0.37	0.27	0.27	0.31	0.31
7/16/2022	0.36	0.40	0.27	0.27	0.29	0.28
7/17/2022	0.29	0.38	0.27	0.27	0.30	0.32
7/18/2022	0.35	0.35	0.25	0.48	0.32	0.34
7/19/2022	0.38	0.41	0.28	0.73	0.29	0.29
7/20/2022	0.35	0.40	0.23	0.69	0.26	0.29
7/21/2022	0.40	0.43	0.25	0.71	0.28	0.26
7/22/2022	0.37	0.38	0.27	0.71	0.29	0.28
7/23/2022	0.36	0.36	0.27	0.71	0.30	0.31
7/24/2022	0.34	0.37	0.28	0.71	0.33	0.30



**Fluoride Analysis  
FY21 - FY23**

Date	ESWTP		BWTP		EFWTP	
	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)
7/25/2022	0.37	0.41	0.26	0.70	0.24	0.25
7/26/2022	0.33	0.36	0.25	0.71	0.29	0.28
7/27/2022	0.38	0.38	0.26	0.70	0.31	0.29
7/28/2022	0.40	0.69	0.25	0.71	0.24	0.24
7/29/2022	0.40	0.67	0.30	0.70	0.25	0.26
7/30/2022	0.40	0.66	0.27	0.70	0.30	0.55
7/31/2022	0.42	0.64	0.29	0.71	0.31	0.59
8/1/2022	0.42	0.69	0.26	0.71	0.27	0.60
8/2/2022	0.44	0.72	0.25	0.72	0.27	0.60
8/3/2022	0.53	0.78	0.27	0.71	0.27	0.60
8/4/2022	0.42	0.73	0.26	0.69	0.28	0.62
8/5/2022	0.42	0.72	0.26	0.70	0.32	0.58
8/6/2022	0.41	0.68	0.30	0.71	0.29	0.55
8/7/2022	0.34	0.68	0.29	0.71	0.31	0.59
8/8/2022	0.39	0.65	0.31	0.71	0.31	0.59
8/9/2022	0.41	0.69	0.28	0.69	0.28	0.52
8/10/2022	0.44	0.70	0.28	0.69	0.29	0.56
8/11/2022	0.39	0.72	0.29	0.70	0.32	0.53
8/12/2022	0.42	0.68	0.28	0.70	0.30	0.58
8/13/2022	0.42	0.71	0.28	0.68	0.31	0.70
8/14/2022	0.40	0.69	0.28	0.70	0.34	0.67
8/15/2022	0.43	0.64	0.32	0.72	0.33	0.61
8/16/2022	0.36	0.66	0.27	0.71	0.31	0.63
8/17/2022	0.37	0.65	0.27	0.71	0.32	0.66
8/18/2022	0.39	0.70	0.23	0.70	0.31	0.67
8/19/2022	0.22	0.48	0.29	0.68	0.32	0.69
8/20/2022	0.18	0.41	0.29	0.70	0.33	0.64
8/21/2022	0.16	0.47	0.29	0.70	0.32	0.53
8/22/2022	0.17	0.44	0.28	0.69	0.31	0.63
8/23/2022	0.41	0.68	0.23	0.68	0.31	0.68
8/24/2022	0.46	0.79	0.29	0.70	0.30	0.68
8/25/2022	0.44	0.71	0.17	0.70	0.32	0.71
8/26/2022	0.41	0.67	0.25	0.70	0.35	0.68

**Fluoride Analysis  
FY21 - FY23**

Date	ESWTP		BWTP		EFWTP	
	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)
8/27/2022	0.46	0.70	0.25	0.70	0.34	0.72
8/28/2022	0.35	0.66	0.25	0.70	0.35	0.79
8/29/2022	0.42	0.64	0.26	0.71	0.37	0.88
8/30/2022	0.45	0.70	0.24	0.70	0.33	0.65
8/31/2022	0.51	0.75	0.24	0.68	0.27	0.63
9/1/2022	0.50	0.75	0.23	0.73	0.33	0.61
9/2/2022	0.44	0.69	0.26	0.68	0.30	0.64
9/3/2022	0.48	0.70	0.25	0.71	0.33	0.70
9/4/2022	0.52	0.75	0.25	0.73	0.34	0.64
9/5/2022	0.54	0.72	0.25	0.72	0.38	0.82
9/6/2022	0.47	0.66	0.22	0.70	0.37	0.80
9/7/2022	0.49	0.68	0.26	0.68	0.34	0.75
9/8/2022	0.43	0.63	0.24	0.71	0.39	0.56
9/9/2022	0.37	0.64	0.26	0.70	0.32	0.51
9/10/2022	0.44	0.67	0.26	0.69	0.31	0.52
9/11/2022	0.38	0.65	0.27	0.72	0.35	0.69
9/12/2022	0.29	0.57	0.30	0.71	0.39	0.76
9/13/2022	0.32	0.57	0.27	0.70	0.42	0.79
9/14/2022	0.37	0.59	0.29	0.69	0.44	0.83
9/15/2022	0.38	0.60	0.28	0.72	0.47	0.76
9/16/2022	0.38	0.64	0.29	0.70	0.50	0.46
9/17/2022	0.45	0.70	0.30	0.71	0.47	0.47
9/18/2022	0.49	0.71	0.31	0.71	0.55	0.55
9/19/2022	0.44	0.70	0.27	0.34	0.55	0.56
9/20/2022	0.47	0.67	0.29	0.34	0.31	0.35
9/21/2022	0.43	0.71	0.31	0.33	0.34	0.47
9/22/2022	0.46	0.66	0.30	0.35	0.35	0.64
9/23/2022	0.44	0.67	0.30	0.34	0.38	0.64
9/24/2022	0.42	0.66	0.30	0.34	0.39	0.67
9/25/2022	0.43	0.65	0.31	0.34	0.39	0.75
9/26/2022	0.56	0.72	0.31	0.36	0.43	0.74
9/27/2022	0.43	0.78	0.31	0.37	XX	XX
9/28/2022	0.43	0.67	0.30	0.37	0.44	0.72

**Fluoride Analysis  
FY21 - FY23**

Date	ESWTP		BWTP		EFWTP	
	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)
9/29/2022	0.49	0.69	0.31	0.37	0.45	0.72
9/30/2022	0.45	0.73	0.28	0.37	0.48	0.70
10/1/2022	0.42	0.69	0.31	0.36	0.32	0.45
10/2/2022	0.40	0.65	0.33	0.36	0.45	0.42
10/3/2022	0.42	0.64	0.30	0.36	0.56	0.57
10/4/2022	0.45	0.68	0.31	0.36	0.61	0.63
10/5/2022	0.40	0.66	0.30	0.35	0.30	0.57
10/6/2022	0.48	0.72	0.30	0.35	0.36	0.62
10/7/2022	0.43	0.78	0.30	0.35	0.49	0.87
10/8/2022	0.48	0.71	0.31	0.36	0.33	0.50
10/9/2022	0.40	0.70	0.32	0.37	0.35	0.60
10/10/2022	0.46	0.68	XX	XX	0.33	0.56
10/11/2022	0.44	0.69	XX	XX	0.35	0.60
10/12/2022	0.46	0.68	XX	XX	0.33	0.63
10/13/2022	0.53	0.73	XX	XX	0.34	0.60
10/14/2022	0.52	0.71	XX	XX	0.33	0.58
10/15/2022	0.49	0.75	XX	XX	0.32	0.65
10/16/2022	0.46	0.70	XX	XX	0.33	0.67
10/17/2022	0.55	0.80	XX	XX	0.36	0.72
10/18/2022	0.40	0.75	XX	XX	0.34	0.78
10/19/2022	0.35	0.71	XX	XX	0.33	0.68
10/20/2022	0.56	0.74	XX	XX	0.33	0.67
10/21/2022	0.41	0.75	XX	XX	0.31	0.65
10/22/2022	0.50	0.69	XX	XX	0.31	0.70
10/23/2022	0.46	0.72	XX	XX	0.32	0.62
10/24/2022	0.43	0.74	0.33	0.36	0.31	0.58
10/25/2022	0.59	0.80	0.24	0.32	0.25	0.73
10/26/2022	0.45	0.69	0.20	0.35	0.28	0.71
10/27/2022	0.51	0.70	0.21	0.35	XX	XX
10/28/2022	0.50	0.74	0.26	0.33	0.42	0.80
10/29/2022	0.36	0.62	0.23	0.31	0.28	0.53
10/30/2022	0.41	0.60	0.27	0.30	0.32	0.57
10/31/2022	0.31	0.68	0.22	0.31	0.35	0.56

**Fluoride Analysis  
FY21 - FY23**

Date	ESWTP		BWTP		EFWTP	
	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)
11/1/2022	XX	XX	0.21	0.30	0.35	0.58
11/2/2022	0.46	0.70	0.21	0.29	0.33	0.55
11/3/2022	0.64	0.76	0.23	0.25	0.33	0.67
11/4/2022	0.54	0.75	0.23	0.22	0.34	0.61
11/5/2022	0.58	0.76	0.22	0.23	0.28	0.57
11/6/2022	0.53	0.74	0.23	0.23	0.32	0.61
11/7/2022	0.56	0.75	0.22	0.28	0.31	0.57
11/8/2022	0.54	0.76	0.21	0.73	0.37	0.70
11/9/2022	0.56	0.76	0.23	0.70	0.38	0.69
11/10/2022	0.53	0.72	0.19	0.72	0.33	0.62
11/11/2022	0.46	0.58	0.24	0.74	0.33	0.57
11/12/2022	0.33	0.60	0.24	0.71	0.33	0.63
11/13/2022	0.50	0.58	0.26	0.72	0.34	0.74
11/14/2022	0.51	0.71	0.27	0.72	0.37	0.67
11/15/2022	0.52	0.70	0.27	0.71	0.34	0.64
11/16/2022	0.47	0.59	0.26	0.71	0.37	0.64
11/17/2022	0.57	0.68	0.27	0.70	0.45	0.65
11/18/2022	0.52	0.65	0.29	0.71	0.38	0.64
11/19/2022	0.60	0.72	0.27	0.70	0.42	0.63
11/20/2022	0.52	0.69	0.28	0.71	0.42	0.56
11/21/2022	0.67	0.73	0.26	0.71	0.43	0.61
11/22/2022	0.55	0.70	0.28	0.72	0.42	0.60
11/23/2022	0.60	0.75	0.40	0.71	0.36	0.60
11/24/2022	0.59	0.74	0.32	0.71	0.36	0.59
11/25/2022	0.38	0.62	0.29	0.71	0.34	0.56
11/26/2022	0.48	0.64	0.28	0.70	0.30	0.53
11/27/2022	0.45	0.58	0.28	0.69	0.27	0.51
11/28/2022	0.31	0.62	0.24	0.70	0.28	0.49
11/29/2022	0.53	0.54	0.23	0.68	0.31	0.52
11/30/2022	0.67	0.75	0.23	0.70	0.34	0.54
12/1/2022	0.61	0.83	0.24	0.70	0.42	0.67
12/2/2022	0.59	0.76	0.25	0.69	0.38	0.63
12/3/2022	0.68	0.77	0.27	0.74	0.37	0.67

**Fluoride Analysis  
FY21 - FY23**

Date	ESWTP		BWTP		EFWTP	
	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)
12/4/2022	0.56	0.71	0.25	0.70	0.40	0.83
12/5/2022	0.68	0.76	0.32	0.70	0.36	0.55
12/6/2022	0.65	0.79	0.28	0.71	0.39	0.53
12/7/2022	0.68	0.76	0.28	0.72	0.36	0.54
12/8/2022	0.60	0.75	0.27	0.71	0.34	0.55
12/9/2022	0.38	0.73	0.33	0.68	0.39	0.65
12/10/2022	0.67	0.64	0.34	0.71	0.44	0.75
12/11/2022	0.54	0.67	0.30	0.70	0.40	0.65
12/12/2022	0.56	0.71	0.27	0.70	0.39	0.72
12/13/2022	0.63	0.70	0.28	0.70	0.36	0.66
12/14/2022	0.70	0.71	0.26	0.69	0.41	0.71
12/15/2022	0.61	0.72	0.25	0.73	0.47	0.72
12/16/2022	0.60	0.74	0.26	0.68	0.45	0.59
12/17/2022	0.70	0.75	0.28	0.71	0.41	0.59
12/18/2022	0.38	0.62	0.28	0.70	0.40	0.56
12/19/2022	0.63	0.62	0.22	0.72	0.38	0.55
12/20/2022	0.58	0.64	0.22	0.71	0.38	0.67
12/21/2022	0.54	0.68	0.26	0.71	0.32	0.70
12/22/2022	0.64	0.68	0.27	0.73	0.41	0.62
12/23/2022	0.55	0.66	0.31	0.72	0.38	0.68
12/24/2022	0.67	0.72	0.32	0.71	0.42	0.67
12/25/2022	0.47	0.63	0.32	0.74	0.38	0.68
12/26/2022	0.57	0.67	0.33	0.71	0.36	0.63
12/27/2022	0.50	0.62	0.33	0.67	0.37	0.58
12/28/2022	0.50	0.57	0.32	0.72	XX	XX
12/29/2022	0.60	0.75	0.33	0.70	XX	XX
12/30/2022	0.41	0.74	0.34	0.71	0.50	0.63
12/31/2022	0.63	0.73	0.33	0.71	0.40	0.43
1/1/2023	0.54	0.71	0.34	0.70	0.39	0.39
1/2/2023	0.42	0.70	0.30	0.68	0.37	0.38
1/3/2023	0.47	0.73	0.30	0.71	0.36	0.38
1/4/2023	0.20	0.55	0.31	0.72	0.37	0.38
1/5/2023	0.20	0.40	0.33	0.71	0.40	0.63

**Fluoride Analysis**  
**FY21 - FY23**

Date	ESWTP		BWTP		EFWTP	
	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)
1/6/2023	0.20	0.57	0.33	0.71	0.53	0.78
1/7/2023	0.16	0.71	0.34	0.70	0.40	0.61
1/8/2023	0.16	0.60	0.33	0.71	0.42	0.61
1/9/2023	0.16	0.68	0.31	0.71	0.42	0.61
1/10/2023	0.22	0.71	0.30	0.68	0.38	0.65
1/11/2023	0.19	0.69	0.28	0.71	0.41	0.60
1/12/2023	0.20	0.69	0.30	0.71	0.39	0.64
1/13/2023	0.19	0.67	0.30	0.71	0.37	0.68
1/14/2023	0.21	0.73	0.33	0.71	0.38	0.70
1/15/2023	0.15	0.61	0.33	0.72	0.41	0.67
1/16/2023	0.20	0.71	0.32	0.70	0.37	0.62
1/17/2023	0.18	0.67	0.32	0.70	0.37	0.63
1/18/2023	0.20	0.66	0.34	0.71	0.36	0.64
1/19/2023	0.20	0.65	0.29	0.72	0.31	0.58
1/20/2023	0.22	0.63	0.34	0.72	0.36	0.70
1/21/2023	0.20	0.66	0.33	0.71	0.47	0.75
1/22/2023	0.17	0.28	0.33	0.70	0.41	0.67
1/23/2023	0.19	0.22	0.33	0.72	0.43	0.76
1/24/2023	0.19	0.64	0.34	0.71	0.42	0.72
1/25/2023	0.21	0.65	0.33	0.71	0.40	0.68
1/26/2023	0.60	0.78	0.28	0.68	0.38	0.70
1/27/2023	0.50	0.93	0.25	0.71	0.39	0.62
1/28/2023	0.48	0.74	0.32	0.77	0.37	0.42
1/29/2023	0.55	0.74	0.33	0.69	0.42	0.63
1/30/2023	0.54	0.78	0.29	0.69	0.40	0.62
1/31/2023	0.54	0.78	0.30	0.67	0.39	0.64
2/1/2023	0.58	0.74	0.30	0.70	0.39	0.54
2/2/2023	0.54	0.78	0.28	0.67	0.37	0.57
2/3/2023	0.53	0.73	0.27	0.66	0.42	0.53
2/4/2023	0.52	0.74	0.29	0.71	0.43	0.69
2/5/2023	0.48	0.72	0.32	0.70	0.38	0.57
2/6/2023	0.50	0.71	0.26	0.70	0.37	0.57
2/7/2023	0.49	0.72	0.26	0.69	0.33	0.54

**Fluoride Analysis  
FY21 - FY23**

Date	ESWTP		BWTP		EFWTP	
	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)
2/8/2023	0.51	0.69	0.23	0.69	0.25	0.60
2/9/2023	0.44	0.67	0.22	0.69	0.24	0.61
2/10/2023	0.44	0.67	0.19	0.69	0.28	0.52
2/11/2023	0.44	0.64	0.25	0.74	0.36	0.68
2/12/2023	0.45	0.62	0.27	0.70	0.42	0.73
2/13/2023	0.44	0.70	0.28	0.69	0.37	0.68
2/14/2023	XX	XX	0.27	0.69	0.38	0.55
2/15/2023	XX	XX	0.26	0.69	0.33	0.64
2/16/2023	0.47	0.69	0.24	0.70	0.34	0.63
2/17/2023	0.51	0.68	0.27	0.70	0.44	0.68
2/18/2023	0.58	0.79	0.28	0.71	0.32	0.65
2/19/2023	0.44	0.66	0.28	0.71	0.35	0.60
2/20/2023	0.44	0.74	0.43	0.71	0.32	0.58
2/21/2023	0.48	0.72	0.24	0.71	0.29	0.52
2/22/2023	0.54	0.69	0.32	0.70	0.29	0.58
2/23/2023	0.54	0.71	0.24	0.71	0.33	0.60
2/24/2023	0.51	0.69	0.27	0.69	0.30	0.57
2/25/2023	0.50	0.70	0.29	0.70	0.30	0.59
2/26/2023	0.50	0.71	0.28	0.71	0.29	0.57
2/27/2023	0.48	0.68	0.29	0.70	0.29	0.50
2/28/2023	XX	XX	0.26	0.71	0.35	0.60
3/1/2023	XX	XX	0.29	0.71	0.35	0.64
3/2/2023	0.41	0.71	0.29	0.71	0.33	0.37
3/3/2023	0.50	0.65	0.29	0.71	0.27	0.29
3/4/2023	0.47	0.68	0.28	0.70	0.31	0.31
3/5/2023	0.45	0.68	0.30	0.70	0.29	0.30
3/6/2023	0.49	0.66	0.28	0.71	XX	XX
3/7/2023	0.62	0.74	0.33	0.69	XX	XX
3/8/2023	0.58	0.77	0.32	0.72	XX	XX
3/9/2023	0.53	0.70	0.29	0.64	XX	XX
3/10/2023	0.51	0.65	0.30	0.72	XX	XX
3/11/2023	0.52	0.70	0.31	0.70	XX	XX
3/12/2023	0.48	0.62	0.30	0.71	XX	XX

**Fluoride Analysis  
FY21 - FY23**

Date	ESWTP		BWTP		EFWTP	
	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)
3/13/2023	0.54	0.65	0.33	0.81	XX	XX
3/14/2023	0.55	0.69	0.32	0.71	XX	XX
3/15/2023	0.57	0.69	0.31	0.71	XX	XX
3/16/2023	0.58	0.73	0.29	0.71	XX	XX
3/17/2023	0.50	0.67	0.32	0.70	XX	XX
3/18/2023	0.55	0.70	0.31	0.71	XX	XX
3/19/2023	0.52	0.68	0.31	0.71	XX	XX
3/20/2023	0.54	0.70	0.33	0.71	XX	XX
3/21/2023	0.49	0.67	0.30	0.72	XX	XX
3/22/2023	0.52	0.66	0.28	0.71	XX	XX
3/23/2023	0.49	0.68	0.30	0.72	XX	XX
3/24/2023	0.54	0.73	0.31	0.70	0.38	0.70
3/25/2023	0.55	0.71	0.32	0.70	0.37	0.41
3/26/2023	0.48	0.64	0.30	0.70	0.37	0.38
3/27/2023	0.60	0.73	0.32	0.69	0.35	0.35
3/28/2023	0.57	0.71	0.31	0.70	0.35	0.36
3/29/2023	0.63	0.81	0.30	0.70	0.36	0.44
3/30/2023	0.63	0.82	0.29	0.70	0.35	0.69
3/31/2023	0.42	0.68	0.31	0.71	0.36	0.50
4/1/2023	0.45	0.60	0.32	0.70	0.37	0.73
4/2/2023	0.51	0.60	0.33	0.69	0.39	0.76
4/3/2023	0.46	0.59	0.31	0.70	0.42	0.73
4/4/2023	XX	XX	0.30	0.70	0.39	0.68
4/5/2023	XX	XX	0.31	0.70	0.38	0.66
4/6/2023	0.45	0.64	0.31	0.70	0.37	0.72
4/7/2023	0.43	0.54	0.32	0.72	0.36	0.72
4/8/2023	0.50	0.54	0.30	0.71	0.40	0.73
4/9/2023	0.50	0.59	0.31	0.70	0.40	0.74
4/10/2023	0.41	0.61	0.29	0.69	0.38	0.67
4/11/2023	0.44	0.62	0.27	0.70	0.36	0.69
4/12/2023	0.52	0.66	0.27	0.73	0.36	0.70
4/13/2023	0.51	0.68	0.28	0.68	0.34	0.69
4/14/2023	0.56	0.71	0.30	0.69	0.34	0.67



**Fluoride Analysis  
FY21 - FY23**

Date	ESWTP		BWTP		EFWTP	
	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)
4/15/2023	0.47	0.70	0.29	0.70	0.33	0.61
4/16/2023	0.46	0.64	0.33	0.70	0.33	0.67
4/17/2023	0.39	0.69	0.29	0.70	0.31	0.61
4/18/2023	0.40	0.63	0.33	0.70	0.31	0.60
4/19/2023	0.41	0.61	0.35	0.70	0.30	0.61
4/20/2023	0.41	0.61	0.27	0.70	0.34	0.59
4/21/2023	0.40	0.62	0.30	0.71	0.35	0.64
4/22/2023	0.38	0.56	0.29	0.71	0.36	0.67
4/23/2023	0.39	0.61	0.30	0.70	0.39	0.70
4/24/2023	0.40	0.60	0.30	0.70	0.37	0.70
4/25/2023	0.42	0.60	0.29	0.70	0.36	0.67
4/26/2023	0.44	0.63	0.26	0.71	0.35	0.65
4/27/2023	0.44	0.62	0.22	0.69	0.37	0.64
4/28/2023	0.41	0.63	0.25	0.72	0.33	0.67
4/29/2023	0.42	0.63	0.29	0.69	0.32	0.60
4/30/2023	0.38	0.62	0.30	0.69	0.31	0.64
5/1/2023	0.40	0.60	0.23	0.71	0.32	0.63
5/2/2023	0.43	0.61	0.24	0.68	0.33	0.56
5/3/2023	0.45	0.70	0.32	0.71	0.30	0.57
5/4/2023	0.47	0.74	0.26	0.71	0.30	0.43
5/5/2023	0.44	0.69	0.28	0.70	0.34	0.35
5/6/2023	0.52	0.72	0.30	0.70	0.31	0.33
5/7/2023	0.49	0.74	0.28	0.71	0.33	0.34
5/8/2023	0.44	0.70	0.30	0.74	0.32	0.33
5/9/2023	0.48	0.68	0.26	0.70	0.33	0.32
5/10/2023	0.44	0.73	0.29	0.70	0.29	0.29
5/11/2023	0.43	0.69	0.24	0.66	0.27	0.27
5/12/2023	0.39	0.67	0.30	0.73	0.25	0.25
5/13/2023	0.43	0.69	0.33	0.72	0.25	0.25
5/14/2023	0.44	0.72	0.45	0.70	0.25	0.27
5/15/2023	0.34	0.67	0.42	0.70	0.25	0.24
5/16/2023	0.32	0.59	0.39	0.74	0.27	0.27
5/17/2023	0.45	0.69	0.28	0.70	0.25	0.25

**Fluoride Analysis  
FY21 - FY23**

Date	ESWTP		BWTP		EFWTP	
	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)
5/18/2023	0.47	0.71	0.28	0.70	0.27	0.27
5/19/2023	0.45	0.75	0.24	0.70	0.26	0.26
5/20/2023	0.42	0.68	0.27	0.69	0.23	0.29
5/21/2023	0.40	0.63	0.27	0.70	0.29	0.38
5/22/2023	0.42	0.64	0.28	0.71	0.28	0.39
5/23/2023	0.44	0.72	0.23	0.70	0.27	0.41
5/24/2023	0.45	0.71	0.29	0.70	0.29	0.33
5/25/2023	0.50	0.69	0.27	0.70	0.31	0.61
5/26/2023	0.40	0.65	0.27	0.70	0.34	0.92
5/27/2023	0.48	0.64	0.28	0.74	0.29	0.70
5/28/2023	0.39	0.64	0.27	0.72	0.31	0.56
5/29/2023	0.51	0.73	0.28	0.72	0.26	0.45
5/30/2023	0.52	0.68	0.33	0.70	0.24	0.35
5/31/2023	0.52	0.69	0.31	0.71	0.22	0.31
6/1/2023	0.50	0.73	0.25	0.71	0.24	0.42
6/2/2023	0.53	0.76	0.27	0.72	0.34	0.74
6/3/2023	0.56	0.80	0.31	0.70	0.32	0.70
6/4/2023	0.53	0.68	0.29	0.70	0.33	0.65
6/5/2023	0.56	0.71	0.27	0.69	0.33	0.65
6/6/2023	0.40	0.62	0.26	0.70	0.36	0.62
6/7/2023	0.40	0.64	0.27	0.67	0.26	0.61
6/8/2023	0.40	0.67	0.24	0.71	0.35	0.68
6/9/2023	0.40	0.66	0.26	0.64	0.35	0.63
6/10/2023	0.48	0.70	0.30	0.76	0.33	0.62
6/11/2023	0.43	0.68	0.34	0.69	0.37	0.64
6/12/2023	0.35	0.62	0.26	0.71	0.33	0.70
6/13/2023	0.34	0.58	0.26	0.70	0.33	0.63
6/14/2023	0.53	0.65	0.28	0.71	0.34	0.65
6/15/2023	0.44	0.71	0.28	0.71	0.34	0.66
6/16/2023	0.47	0.68	0.27	0.72	0.34	0.65
6/17/2023	0.48	0.71	0.29	0.72	0.31	0.62
6/18/2023	0.39	0.64	0.30	0.72	0.34	0.63
6/19/2023	0.42	0.64	0.27	0.71	0.31	0.62

**Fluoride Analysis  
FY21 - FY23**

Date	ESWTP		BWTP		EFWTP	
	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)
6/20/2023	0.41	0.64	0.29	0.70	0.29	0.55
6/21/2023	0.44	0.69	0.34	0.70	0.27	0.58
6/22/2023	0.45	0.68	0.29	0.68	0.24	0.49
6/23/2023	0.48	0.73	0.30	0.71	0.23	0.51
6/24/2023	0.43	0.74	0.31	0.65	0.25	0.61
6/25/2023	0.51	0.76	0.30	0.70	0.34	0.84
6/26/2023	0.56	0.83	0.33	0.67	0.33	0.77
6/27/2023	0.57	0.82	0.28	0.65	0.36	0.84
6/28/2023	0.56	0.80	0.32	0.65	0.43	0.78
6/29/2023	0.53	0.73	0.30	0.64	0.32	0.68
6/30/2023	0.35	0.62	0.34	0.59	0.33	0.62
7/1/2023	0.36	0.60	0.28	0.71	0.39	0.69
7/2/2023	0.32	0.56	0.35	0.72	0.47	0.76
7/3/2023	0.40	0.64	0.35	0.71	0.45	0.77
7/4/2023	0.37	0.60	0.31	0.70	0.52	0.74
7/5/2023	0.47	0.69	0.29	0.70	0.48	0.72
7/6/2023	0.46	0.72	0.32	0.71	0.46	0.73
7/7/2023	0.44	0.70	0.33	0.70	0.46	0.76
7/8/2023	0.47	0.70	0.31	0.71	0.47	0.72
7/9/2023	0.43	0.66	0.32	0.71	0.52	0.73
7/10/2023	0.41	0.64	0.33	0.71	0.46	0.80
7/11/2023	0.49	0.69	0.33	0.70	0.45	0.74
7/12/2023	0.46	0.71	0.31	0.71	0.47	0.73
7/13/2023	0.44	0.68	0.30	0.70	0.47	0.70
7/14/2023	0.44	0.69	0.32	0.71	0.47	0.74
7/15/2023	0.45	0.73	0.32	0.71	0.48	0.69
7/16/2023	0.47	0.71	0.31	0.69	0.50	0.72
7/17/2023	0.47	0.72	0.33	0.71	0.45	0.63
7/18/2023	0.45	0.67	0.33	0.70	0.41	0.60
7/19/2023	0.54	0.73	0.33	0.70	0.31	0.44
7/20/2023	0.54	0.73	0.33	0.70	0.31	0.57
7/21/2023	0.62	0.79	0.31	0.71	0.31	0.63
7/22/2023	0.50	0.68	0.31	0.71	0.30	0.63

**Fluoride Analysis  
FY21 - FY23**

Date	ESWTP		BWTP		EFWTP	
	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)
7/23/2023	0.47	0.65	0.29	0.71	0.30	0.59
7/24/2023	0.48	0.67	0.30	0.71	0.29	0.62
7/25/2023	0.44	0.63	0.29	0.72	0.29	0.68
7/26/2023	0.47	0.70	0.32	0.71	0.31	0.62
7/27/2023	0.51	0.68	0.31	0.69	0.31	0.65
7/28/2023	0.45	0.69	0.31	0.70	0.29	0.64
7/29/2023	0.53	0.72	0.30	0.71	0.29	0.64
7/30/2023	0.47	0.65	0.29	0.70	0.30	0.63
7/31/2023	0.45	0.67	0.28	0.71	0.27	0.56
8/1/2023	0.48	0.66	0.29	0.72	0.29	0.60
8/2/2023	0.44	0.65	0.31	0.71	0.33	0.67
8/3/2023	0.47	0.68	0.34	0.72	0.29	0.59
8/4/2023	0.43	0.67	0.32	0.71	0.30	0.53
8/5/2023	0.50	0.70	0.34	0.72	0.26	0.57
8/6/2023	0.41	0.61	0.32	0.71	0.28	0.58
8/7/2023	0.44	0.69	0.32	0.71	0.27	0.60
8/8/2023	0.49	0.70	0.31	0.71	0.30	0.65
8/9/2023	0.48	0.71	0.32	0.71	0.31	0.61
8/10/2023	0.46	0.65	0.32	0.71	0.32	0.72
8/11/2023	0.49	0.70	0.33	0.70	0.33	0.65
8/12/2023	0.50	0.69	0.29	0.70	0.30	0.57
8/13/2023	0.49	0.61	0.32	0.69	0.35	0.63
8/14/2023	0.41	0.57	0.33	0.71	0.38	0.71
8/15/2023	0.49	0.71	0.32	0.72	0.31	0.52
8/16/2023	0.46	0.65	0.33	0.72	0.29	0.50
8/17/2023	0.49	0.68	0.32	0.72	0.33	0.64
8/18/2023	0.50	0.72	0.32	0.71	0.33	0.57
8/19/2023	0.50	0.72	0.30	0.71	0.29	0.52
8/20/2023	0.51	0.66	0.34	0.71	0.33	0.60
8/21/2023	0.50	0.74	0.34	0.70	0.30	0.60
8/22/2023	0.50	0.72	0.33	0.69	0.30	0.52
8/23/2023	0.49	0.76	0.33	0.71	0.29	0.50
8/24/2023	0.44	0.69	0.34	0.71	0.30	0.63

**Fluoride Analysis  
FY21 - FY23**

Date	ESWTP		BWTP		EFWTP	
	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)
8/25/2023	0.42	0.67	0.32	0.70	0.30	0.65
8/26/2023	0.51	0.67	0.29	0.69	0.33	0.63
8/27/2023	0.50	0.74	0.33	0.70	0.33	0.66
8/28/2023	0.60	0.68	0.33	0.71	0.30	0.61
8/29/2023	0.50	0.71	0.31	0.70	0.34	0.65
8/30/2023	0.52	0.77	0.34	0.70	0.33	0.68
8/31/2023	0.43	0.67	0.31	0.71	0.31	0.59
9/1/2023	0.38	0.49	0.34	0.71	0.30	0.56
9/2/2023	0.39	0.49	0.30	0.71	0.30	0.60
9/3/2023	0.33	0.45	0.29	0.71	0.37	0.63
9/4/2023	0.43	0.52	0.31	0.71	0.35	0.59
9/5/2023	0.40	0.53	0.31	0.71	0.33	0.52
9/6/2023	0.36	0.51	0.31	0.70	0.32	0.57
9/7/2023	0.37	0.54	0.31	0.69	0.33	0.64
9/8/2023	0.40	0.58	0.32	0.70	0.31	0.60
9/9/2023	0.36	0.60	0.28	0.70	0.41	0.68
9/10/2023	0.35	0.56	0.31	0.70	0.37	0.64
9/11/2023	0.35	0.55	0.32	0.71	0.32	0.65
9/12/2023	0.33	0.55	0.31	0.71	0.36	0.61
9/13/2023	0.40	0.61	0.34	0.71	0.31	0.58
9/14/2023	0.43	0.63	0.30	0.71	0.33	0.64
9/15/2023	0.41	0.64	0.32	0.69	0.31	0.57
9/16/2023	0.40	0.60	0.30	0.70	0.30	0.57
9/17/2023	0.42	0.64	0.32	0.70	0.38	0.61
9/18/2023	0.43	0.64	0.32	0.70	0.35	0.61
9/19/2023	0.52	0.82	0.32	0.70	0.33	0.66
9/20/2023	0.55	0.84	0.33	0.71	0.28	0.64
9/21/2023	0.50	0.75	0.34	0.71	0.34	0.66
9/22/2023	0.64	0.74	0.32	0.71	0.34	0.66
9/23/2023	0.50	0.66	0.30	0.70	0.34	0.65
9/24/2023	0.52	0.69	0.30	0.70	0.35	0.62
9/25/2023	0.60	0.73	0.30	0.70	0.34	0.61
9/26/2023	0.40	0.62	0.30	0.71	0.35	0.69

**Fluoride Analysis  
FY21 - FY23**

Date	ESWTP		BWTP		EFWTP	
	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)	RAW (mg/L)	TAP (mg/L)
9/27/2023	0.47	0.60	0.24	0.70	0.33	0.64
9/28/2023	0.46	0.65	0.30	0.70	0.32	0.61
9/29/2023	0.44	0.62	0.28	0.71	0.31	0.66
9/30/2023	0.45	0.64	0.31	0.70	0.35	0.73
10/1/2023	0.59	0.79	0.30	0.71	0.32	0.60
10/2/2023	0.49	0.67	0.30	0.70	0.31	0.63
10/3/2023	0.55	0.79	0.31	0.71	0.31	0.65
10/4/2023	0.56	0.73	0.34	0.71	0.32	0.61
10/5/2023	0.56	0.79	0.34	0.71	0.33	0.65
10/6/2023	0.49	0.67	0.33	0.71	0.30	0.58
10/7/2023	0.48	0.32	0.29	0.71	0.33	0.62
10/8/2023	0.46	0.67	0.30	0.70	0.32	0.64
10/9/2023	0.55	0.65	0.31	0.71	0.31	0.62
10/10/2023	0.51	0.72	0.33	0.70	0.31	0.63
10/11/2023	0.55	0.71	0.30	0.70	0.31	0.61
10/12/2023	0.55	0.75	0.29	0.70	0.31	0.62
10/13/2023	0.46	0.64	0.33	0.70	0.31	0.62
10/14/2023	0.40	0.55	0.33	0.71	0.34	0.64
10/15/2023	0.57	0.71	0.33	0.70	0.33	0.63
10/16/2023	0.54	0.70	0.33	0.71	0.32	0.74
10/17/2023	0.59	0.74	0.32	0.72	0.33	0.68
10/18/2023	0.64	0.76	0.32	0.71	0.35	0.64

# **ATTACHMENT B**

January 31, 1966

TO THE HONORABLE MAYOR AND CITY COUNCIL:

WE, the undersigned, your Committee of the City Council, heretofore appointed on January 24, 1966, to canvass the returns of the Special Election held in the City of Dallas on the 29th day of January A.D. 1966, in accordance with Ordinance No. 11317 enacted by the City Council on the 3rd day of January, A.D. 1966, calling said Special Election for the purpose of determining whether or not the Ordinance making it unlawful for the City of Dallas or any of its employees or agents to place any fluoride or fluorine, or any chemical compound containing the same, in water distributed or furnished for domestic use by the Dallas City Waterworks within the City of Dallas be enacted by the City Council of the City of Dallas, hereby report that in accordance with said Ordinance enacted on January 3, 1966, the following proposition was submitted to the qualified voters of the City of Dallas, to-wit:

PROPOSITION

"FOR: THE ORDINANCE MAKING IT UNLAWFUL FOR THE CITY OF DALLAS OR ANY OF ITS EMPLOYEES OR AGENTS TO PLACE ANY FLUORIDE OR FLUORINE, OR ANY CHEMICAL COMPOUND CONTAINING THE SAME, IN WATER DISTRIBUTED OR FURNISHED FOR DOMESTIC USE BY THE DALLAS CITY WATERWORKS WITHIN THE CITY OF DALLAS.

AGAINST: THE ORDINANCE MAKING IT UNLAWFUL FOR THE CITY OF DALLAS OR ANY OF ITS EMPLOYEES OR AGENTS TO PLACE ANY FLUORIDE OR FLUORINE, OR ANY CHEMICAL COMPOUND CONTAINING THE SAME, IN WATER DISTRIBUTED OR FURNISHED FOR DOMESTIC USE BY THE DALLAS CITY WATERWORKS WITHIN THE CITY OF DALLAS.

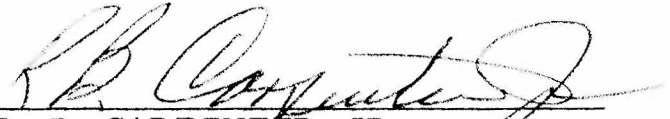
We beg to state that we have carefully canvassed the returns of said Special Election and find that the following number of votes were cast respectively "for" and "against" said proposition:

"FOR:"	<u>10,940</u>
"AGAINST"	<u>27,089</u>


That the votes by Precincts "FOR" and "AGAINST" said Proposition are tabulated as follows:



From the foregoing tabulation it appears that said Proposition did not receive the favorable vote of the majority of the votes cast by the qualified voters of the City of Dallas participating in said Special Election, and therefore failed to carry.

  
R. B. CARPENTER, JR.

  
MRS. C. F. HAMILTON

  
WILLIAM E. COTHRUM

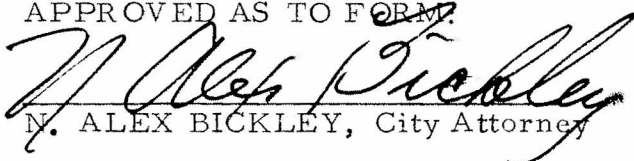
WHEREAS, the Canvassing Committee of the City Council has filed its Report canvassing the Special Election held under authority of Ordinance No. 11317, and the City Council in Regular Session has duly examined said Report of the Canvassing Committee and finds that it is in all things correct and that said Special Election was regularly held as provided by law; Now, Therefore,

BE IT RESOLVED BY THE CITY COUNCIL OF THE CITY OF DALLAS:

SECTION 1. That the proposition set forth in Ordinance No. 11317, passed by the City Council on the 3rd day of January A. D. 1966, was duly submitted to the qualified voters of the City of Dallas on the 29th day of January A. D. 1966 at a Special Election called by said Ordinance No. 11317 for the purpose of determining whether or not an ordinance should be enacted by the City Council of the City of Dallas making it unlawful for the City of Dallas or any of its employees or agents to place any fluoride or fluorine, or any chemical compound containing the same, in water distributed or furnished for domestic use by the Dallas City Waterworks within the City of Dallas, and that said proposition failed to receive a majority of the votes cast at said Election, and that said proposition is, therefore, declared void.



SECTION 2. That this resolution shall take effect from and after its passage as in the Charter in such cases is made and provided.

APPROVED AS TO FORM:

  
N. ALEX BICKLEY, City Attorney

APPROVED BY  
CITY COUNCIL

JAN 31 1966

  
  
Harold B. Pham  
City Secretary

PROPOSITION

THE ORDINANCE making it unlawful for the City of Dallas or any of its employees or agents to place any Fluoride or Fluorine, or any chemical compound containing the same, in water distributed or furnished for domestic use by the Dallas City Waterworks within the City of Dallas.

	FOR	AGAINST	
108	21	35	1
109	71	244	2
110	177	964	3
111	170	880	4
112	59	407	5
113	70	467	6
114	82	213	7
115	71	252	8
116	80	441	9
117	57	511	10
118	65	537	11
119	30	102	12
120	90	280	13
121	60	214	14
126	200	547	15
127	86	317	16
128	36	54	17
129	56	279	18
134	72	90	19
138	86	72	20
139	70	228	21
144	40	83	22
145	114	188	23
146	126	360	24
147	123	231	25
148	112	164	26
149	131	434	27
150	114	160	28
151	60	135	29
152	87	130	30
153	40	137	31
154	49	69	32
155	106	82	33
156	71	96	34
157	30	20	35
158	60	146	36
159	40	91	37
160	37	76	38
161	41	69	39
162	36	85	40
163	16	21	41
164	18	27	42
165	29	27	43
166	17	37	44
167	154	473	45

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434	162	262
436	215	726
437	125	346
440	141	187
441	147	530
442	84	101
444	46	21
445	117	328
446	28	34
ABSENTEE	69	167

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**TOTAL**                      10940      27089

*William J. Hamilton*  
*K. P. Carpenter*  
*Wm. C. B. Hamilton*

# ATTACHMENT C

# U.S. Public Health Service Recommendation for Fluoride Concentration in Drinking Water for the Prevention of Dental Caries

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U.S. DEPARTMENT OF  
HEALTH AND HUMAN  
SERVICES FEDERAL PANEL  
ON COMMUNITY WATER  
FLUORIDATION

Through this final recommendation, the U.S. Public Health Service (PHS) updates and replaces its 1962 Drinking Water Standards related to community water fluoridation—the controlled addition of a fluoride compound to a community water supply to achieve a concentration optimal for dental caries prevention.<sup>1</sup> For these community water systems that add fluoride, PHS now recommends an optimal fluoride concentration of 0.7 milligrams/liter (mg/L). In this guidance, the optimal concentration of fluoride in drinking water is the concentration that provides the best balance of protection from dental caries while limiting the risk of dental fluorosis. The earlier PHS recommendation for fluoride concentrations was based on outdoor air temperature of geographic areas and ranged from 0.7–1.2 mg/L. This updated guidance is intended to apply to community water systems that currently fluoridate, or that will initiate fluoridation, and is based on considerations that include:

- Scientific evidence related to the effectiveness of water fluoridation in caries prevention and control across all age groups,
- Fluoride in drinking water as one of several available fluoride sources,
- Trends in the prevalence and severity of dental fluorosis, and
- Current evidence on fluid intake of children across various outdoor air temperatures.

## BACKGROUND

Because fluoridation of public drinking water systems had been demonstrated as effective in reducing dental caries, PHS provided recommendations regarding optimal fluoride concentrations in drinking water for community water systems in 1962.<sup>2,3</sup> The U.S. Department of Health and Human Services (HHS) is releasing this updated PHS recommendation because of new data that address changes in the prevalence of dental fluorosis, the relationship between water intake and outdoor temperature in children, and the contribution of fluoride in drinking water to total fluoride exposure in the United States. Although PHS recommends community water fluoridation as an effective public health intervention, the decision to fluoridate water systems is made by state and local governments.

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As of December 31, 2012, the U.S. Centers for Disease Control and Prevention (CDC) estimated that approximately 200 million people in the United States were served by 12,341 community water systems that added fluoride to water or purchased water with added fluoride from other systems. For many years, nearly all of these fluoridated systems used fluoride concentrations ranging from 0.8 to 1.2 mg/L; fewer than 1% of these systems used a fluoride concentration at 0.7 mg/L (Unpublished data, Water Fluoridation Reporting System, CDC, 2010). When water systems that add fluoride implement the new PHS recommendation (0.7 mg/L), the fluoride concentration in these systems will be reduced by 0.1–0.5 mg/L, and fluoride intake from water will decline among most people served by these systems.

It is expected that implementation of the new recommendation will lead to a reduction of approximately 25% (range: 12%–42%) in fluoride intake from drinking water alone and a reduction of approximately 14% (range: 5%–29%) in total fluoride intake. These estimates are based on intake among young children at the 90th percentile of drinking water intake for whom drinking water accounts for 40%–70% of total fluoride intake.<sup>4</sup> Furthermore, these estimates are based on a weighted mean fluoride concentration of 0.94 mg/L in systems that added fluoride (or purchased water from systems that added fluoride) in 2009 (Unpublished data, Water Fluoridation Reporting System, CDC, 2009). Community water systems that contain naturally occurring fluoride at concentrations >0.7 mg/L (estimated to serve about 11 million people) will not be directly affected by the new PHS recommendation.

Under the Safe Drinking Water Act, the U.S. Environmental Protection Agency (EPA) sets standards for drinking water quality.<sup>5</sup> EPA is in the process of reviewing the maximum amount of fluoride allowed in drinking water. Upon completion of its review, the EPA will determine if it is appropriate to revise the drinking water standard for fluoride. Currently, the enforceable standard is set at 4.0 mg/L to protect against severe skeletal fluorosis (i.e., a bone disease caused by excessive fluoride intake for a long period of time that in advanced stages can cause pain or damage to bones and joints), which is a rare condition in the United States.<sup>6,7</sup> If the EPA determines that it is appropriate to revise the standard, any revisions could affect certain community water systems that have naturally occurring fluoride. More information about EPA's existing drinking water standards for fluoride can be found on the EPA's website.<sup>8</sup>

## RECOMMENDATION

For community water systems that add fluoride to their water, PHS recommends a fluoride concentration of 0.7 mg/L (parts per million [ppm]) to maintain caries prevention benefits and reduce the risk of dental fluorosis.

### Rationale

**Importance of community water fluoridation.** Community water fluoridation is a major factor responsible for the decline in prevalence (occurrence) and severity of dental caries (tooth decay) during the second half of the 20th century.<sup>9</sup> For adolescents, the prevalence of dental caries in at least one permanent tooth (excluding third molars) decreased from 90% among those aged 12–17 years in the 1960s to 60% among those aged 12–19 years in 1999–2004; during that interval, the number of permanent teeth affected by dental caries (i.e., decayed, missing, and filled) declined from 6.2 to 2.6, respectively.<sup>10,11</sup> Adults also have benefited from community water fluoridation; the average number of affected teeth decreased from 18 among 35- to 44-year-old adults in the 1960s to 10 among 35- to 49-year-old adults in 1999–2004.<sup>11,12</sup> Although data were not age-adjusted, age groups in the 1999–2004 survey used a higher upper age limit, and both caries prevalence and number of teeth affected increased with age; thus, these comparisons may underestimate caries decline over time.

Although there have been notable declines in tooth decay, it remains one of the most common chronic diseases of childhood.<sup>1,13</sup> In 2009–2010, national survey data showed that untreated dental caries among children varied by race/ethnicity and federal poverty level. About one in four children living below 100% of the federal poverty level had untreated tooth decay,<sup>14</sup> which can result in pain, school absences, and poorer school performance.<sup>15–18</sup>

Systematic reviews of the scientific evidence related to fluoride have concluded that community water fluoridation is effective in decreasing dental caries prevalence and severity.<sup>19–26</sup> Effects included significant increases in the proportion of children who were caries-free and significant reductions in the number of teeth or tooth surfaces with caries in both children and adults.<sup>20,22,24–26</sup> When analyses were limited to studies conducted after the introduction of other sources of fluoride, especially fluoride toothpaste, beneficial effects across the lifespan from community water fluoridation were still apparent.<sup>20,24,27</sup>

Fluoride in saliva and dental plaque works to prevent dental caries primarily through topical remineralization



of tooth surfaces.<sup>28,29</sup> Consuming fluoridated water and beverages, and foods prepared or processed with fluoridated water, throughout the day maintains a low concentration of fluoride in saliva and plaque that enhances remineralization. Although other fluoride-containing products are available and contribute to the prevention and control of dental caries, community water fluoridation has been identified as the most cost-effective method of delivering fluoride to all members of the community regardless of age, educational attainment, or income level.<sup>9,30</sup> Studies continue to find that community water fluoridation is cost saving.<sup>21,31–33</sup>

**Trends in availability of fluoride sources.** Community water fluoridation and fluoride toothpaste are the most common sources of non-dietary fluoride in the United States.<sup>34</sup> Community water fluoridation began in 1945, reaching 49% of the U.S. population by 1975 and 67% by 2012.<sup>35,36</sup> Toothpaste containing fluoride was first marketed in the United States in 1955.<sup>37</sup> By 1983, more than 90% of children and adolescents 5–19 years of age, and almost 70% of young children 2–4 years of age, reportedly used fluoride toothpaste.<sup>38</sup> By 1986, more than 90% of young children 2–4 years of age were reported to use fluoride toothpaste.<sup>39</sup> And by the 1990s, fluoride toothpaste accounted for more than 90% of the toothpaste market.<sup>40</sup> Other products that provide fluoride now include mouth rinses, dietary fluoride supplements, and professionally applied fluoride compounds. More detailed explanations of these products are published elsewhere.<sup>34,41,42</sup>

More information on major sources of ingested fluoride and their relative contributions to total fluoride exposure in the United States is presented in an EPA report.<sup>4</sup> To protect the majority of the population, EPA uses the 90th percentile of drinking water intake for all age groups to calculate the relative contribution for each fluoride source. The EPA definition of “drinking water” includes tap water ingested alone or with beverages and certain foods reconstituted in the home. Among children aged 6 months to 14 years, drinking water accounts for 40%–70% of total fluoride intake; for adults, drinking water provides 60% of total fluoride intake. Toothpaste that has been swallowed inadvertently is estimated to account for about 20% of total fluoride intake in very young children (1–3 years of age).<sup>4</sup> Other major contributors to total daily fluoride intake are commercial beverages and solid foods.

**Dental fluorosis.** Fluoride ingestion while teeth are developing can result in a range of visually detectable changes in the tooth enamel called dental fluorosis.<sup>43</sup> Changes range from barely visible lacy white markings in milder cases to pitting of the teeth in the rare,

severe form. The period of possible risk for fluorosis in the permanent teeth (excluding the third molars) extends from birth through 8 years of age when the pre-eruptive maturation of tooth enamel is complete.<sup>34,44,45</sup> The risk for and severity of dental fluorosis depends on the amount, timing, frequency, and duration of the exposure.<sup>34</sup> When communities first began adding fluoride to their public water systems in 1945, drinking water and local foods and beverages prepared with fluoridated water were the primary sources of fluoride for most children.<sup>7,46</sup> At that time, only a few systems fluoridated their water, minimizing the amount of fluoride contributed by processed water to commercial foods and beverages. Since the 1940s, other sources of ingested fluoride such as fluoride toothpaste (if swallowed) and dietary fluoride supplements have become available. Fluoride intake from these products, in addition to water, other beverages, and infant formula prepared with fluoridated water, have been associated with increased risk of dental fluorosis.<sup>47–53</sup> Both the 1962 PHS recommendations and the current updated recommendation for fluoride concentration in community drinking water were set to achieve reduction in dental caries while minimizing the risk of dental fluorosis.

Results of two national surveys indicate that the prevalence of dental fluorosis has increased since the 1980s, but mostly in very mild or mild forms. Data on the prevalence of dental fluorosis come from the National Health and Nutrition Examination Survey (NHANES) 1999–2004. NHANES assessed the prevalence and severity of dental fluorosis among people aged 6–49 years. Twenty-three percent (95% confidence interval [CI] 20.1, 26.1) had dental fluorosis, of which the vast majority was very mild or mild. Approximately 2% (95% CI 1.5, 2.5) of people had moderate dental fluorosis, and fewer than 1% (95% CI 0.1, 0.4) had severe fluorosis. The prevalence of dental fluorosis that was very mild or greater was higher among young people and ranged from 41% (95% CI 36.3, 44.9) among adolescents aged 12–15 years to 9% (95% CI 6.1, 11.4) among adults aged 40–49 years.<sup>54</sup>

The prevalence and severity of dental fluorosis among 12- to 15-year-olds in 1999–2004 also were compared with estimates from the Oral Health of United States Children survey, 1986–1987, which was the first national survey to include measures of dental fluorosis.<sup>55</sup> Although these two national surveys differed in sampling and representation (household vs. schoolchildren), findings support the hypothesis that there was an increase in dental fluorosis that was very mild or greater during the time between the two surveys. In 1986–1987 and 1999–2004, the prevalence

of dental fluorosis was 23% and 41%, respectively, among adolescents aged 12–15 years.<sup>54</sup> Similarly, the prevalence of very mild fluorosis (17.2% and 28.5%), mild fluorosis (4.1% and 8.6%), and moderate and severe fluorosis combined (1.3% and 3.6%) among 12- to 15-year-old adolescents during 1986–1987 and 1999–2004, respectively, all showed increases. Estimates limited to severe fluorosis among adolescents in both surveys, however, were statistically unreliable because there were too few cases among survey participants examined. The higher prevalence of dental fluorosis in young people in 1999–2004 may reflect increases in fluoride exposures (intake) across the U.S. population.

Children are at risk for fluorosis in the permanent teeth from birth through 8 years of age. Adolescents who were 12–15 years of age when they participated in the national surveys of 1986–1987 and 1999–2004 would have been at risk for dental fluorosis during 1971–1983 and 1984–2000, respectively.

By 1969, the percentage of the U.S. population receiving fluoridated water was 44% ( $n=88,475,684$ ). By 1985, this percentage increased about 10 percentage points to 55% ( $n=130,172,334$ ). By 2000, this percentage was 57% ( $n=161,924,080$ ). Although the percentage point increases in more recent years appear small (2 percentage points from 1985 to 2000), it is important to note that the total size of the U.S. population also continued to expand during the time period. As a result, the 10-percentage-point increase from 1969 to 1985 reflects an increase of more than 40 million people receiving fluoridated water, whereas the 2-percentage-point increase from 1985 to 2000 represents an increase of more than 30 million people.<sup>36</sup>

Available data do not support additional detailed examination of changes in the percentage of children and adolescents using fluoride toothpaste. As mentioned previously, by 1983, more than 90% of children and adolescents 5–19 years of age, and almost 70% of young children 2–4 years of age, were reportedly using fluoride toothpaste; by 1986, more than 90% of young children were also using fluoride toothpaste.<sup>38,39</sup> As mentioned, recent EPA estimates indicate that toothpaste swallowed inadvertently accounts for about 20% of total fluoride intake in very young children.<sup>4</sup>

More information on fluoride concentrations in drinking water and the risk of severe dental fluorosis in children is presented in an EPA report.<sup>7</sup> EPA's scientific assessments considered new data on dental fluorosis and updated exposure estimates to reflect current conditions. Based on original data from a study that predated widespread water fluoridation in the United States, EPA determined that the benchmark dose for a 0.5% prevalence of severe dental fluorosis was a drink-

ing water fluoride concentration of 2.14 mg/L, with a lower 95% CI of 1.87 mg/L.<sup>7</sup> Categorical regression modeling also indicated that the concentration of fluoride in water associated with a 1% prevalence of severe dental fluorosis decreased over time (1940–2000).<sup>56</sup> These findings are consistent with an increase in exposures from other sources of fluoride and support the conclusion that a fluoride concentration in drinking water of 0.7 mg/L would reduce the chance of dental fluorosis—especially severe dental fluorosis—in the current context of multiple fluoride sources.

The two EPA assessments of fluoride published in 2010 responded to earlier findings of the National Research Council (NRC) of the National Academies of Science, published in 2006.<sup>4,6,7</sup> The NRC had reviewed new data on fluoride at EPA's request and in 2006 recommended that EPA update health and exposure assessments to consider all sources of fluoride and to take into account dental effects—specifically, pitting of teeth (i.e., severe dental fluorosis) in children. The NRC identified severe dental fluorosis as an adverse health effect, because pitting of the enamel compromises its protective function. The NRC's report focused on the potential for adverse effects from naturally occurring fluoride at 2–4 mg/L in drinking water; it did not examine benefits or risks that might occur at lower concentrations typically used for community water fluoridation (0.7–1.2 mg/L).<sup>6</sup> For this PHS recommendation, panel scientists did review the balance of benefits and potential for unwanted effects of water fluoridation at those lower levels.<sup>7</sup>

**Relationship between dental caries and fluorosis at varying water fluoridation concentrations.** The 1986–1987 Oral Health of United States Children survey has been the only national survey that assessed the child's water fluoride exposure, thus allowing linkage of that exposure to measures of caries and fluorosis.<sup>55</sup> An additional analysis of data from this survey examined the relationship between dental caries and fluorosis at varying water fluoride concentrations for children and adolescents. Findings indicate that there was a gradual decline in dental caries as fluoride content in water increased from negligible to 0.7 mg/L. Reductions plateaued at concentrations from 0.7–1.2 mg/L. In contrast, the percentage of children with at least very mild dental fluorosis increased from 13.5% (standard error [SE] = 1.9) to 41.4% (SE=4.4) as fluoride concentrations in water increased from <0.3 mg/L to >1.2 mg/L.<sup>57</sup>

In Hong Kong, a small decrease of about 0.2 mg/L in the mean fluoride concentration in drinking water in 1978 (from 0.82 mg/L to 0.64 mg/L) was associated with a detectable reduction in fluorosis prevalence by

the mid-1980s, from 64% (SE=4.1) to 47% (SE=4.5), based on the upper right central incisor only. Across all age groups, more than 90% of fluorosis cases were very mild or mild.<sup>58</sup> The study did not include measures of fluoride intake. Concurrently, dental caries prevalence did not increase.<sup>59</sup> Although not fully generalizable to the current U.S. context, these findings, along with findings from the 1986–1987 survey of U.S. schoolchildren, suggest that the risk of fluorosis can be reduced and caries prevention maintained toward the lower end (i.e., 0.7 mg/L) of the 1962 PHS recommendations for community water fluoridation.

***Relationship of water intake and outdoor temperature among children and adolescents in the United States.*** The 1962 PHS recommendations stated that community drinking water should contain 0.7–1.2 mg/L (ppm) fluoride, depending on the outdoor air temperature of the area. These temperature-related guidelines were based on studies conducted in two communities in California in the early 1950s. Findings indicated that a lower fluoride concentration was appropriate for communities in warmer climates because children drank more water on warm days.<sup>60–62</sup> Social and environmental changes, including increased use of air conditioning and more sedentary lifestyles, have occurred since the 1950s; thus, the assumption that children living in warmer regions drink more tap water than children in cooler regions may no longer be valid.<sup>63</sup>

Studies conducted since 2001 suggest that children's water intake does not increase with increases in outdoor air temperature.<sup>64,65</sup> One study conducted among children using nationally representative data from NHANES 1988–1994 did not find an association between either total or plain water intake and outdoor air temperature.<sup>64</sup> Although a similar study using nationally representative data from NHANES 1999–2004 also found no association between total water intake and outdoor temperature among children or adolescents, additional analyses of these data detected a small but statistically significant association between plain water intake and outdoor temperature.<sup>65,66</sup> Temperature explained less than 1% of the variation in plain water intake; thus, these findings support the use of one target concentration for community water fluoridation in all temperature zones of the United States, a standard far simpler to implement than the 1962 temperature-based recommendations. In these analyses, "plain water" was defined as from the tap or bottled water, and "total water" included water from or mixed with other beverages, such as juice, soda, sport drinks, and nondairy milk, as well as water from or mixed with foods.<sup>66</sup>

## PROCESS

HHS convened a federal interdepartmental, inter-agency panel of scientists to review scientific evidence relevant to the 1962 PHS Drinking Water Standards for fluoride concentrations in drinking water in the United States and to update these recommendations based on current science. Panelists included representatives from CDC, the National Institutes of Health, the U.S. Food and Drug Administration, the Agency for Healthcare Research and Quality, the Office of the Assistant Secretary for Health, EPA, and the U.S. Department of Agriculture.

The panel evaluated recent systematic reviews of the effectiveness of fluoride in drinking water to prevent dental caries, as well as published reports about the epidemiology of dental caries and fluorosis in the United States and the relationship of these conditions with varying water fluoridation concentrations. The panel also reviewed existing recommendations for fluoride in drinking water and newer data on the relationship between water intake in children and outdoor air temperature in the United States—a relationship that had served as the basis for the 1962 recommendations.

Recent systematic reviews of evidence on the effectiveness of community water fluoridation were from the Community Preventive Services Task Force, first published in 2001 and updated in 2013, and the Australian National Health and Medical Research Council in 2007.<sup>21,23,25,26</sup> Both reviews were updates of a comprehensive systematic review of water fluoridation completed by the National Health Service Centre for Reviews and Dissemination, University of York, in 2000.<sup>19,20</sup> In these reviews, estimates of fluoridation effectiveness in preventing caries were limited to children and adolescents and based on comparative studies. Random assignment of individuals usually is not feasible for studies of water fluoridation, because the intervention occurs in the community water system. Another systematic review examined the effectiveness of water fluoridation in preventing dental caries in adults. Findings were based primarily on cross-sectional studies of lifelong residents of communities with fluoridated or non-fluoridated water.<sup>24</sup> Studies in these systematic reviews were not limited to the United States.

Panel scientists accepted an extensive review of fluoride in drinking water by the NRC as the summary of hazard.<sup>6</sup> The NRC review focused on potential adverse effects of naturally occurring fluoride at 2–4 mg/L in drinking water; it found no evidence substantial enough to support effects other than severe dental fluorosis at these levels. A majority of NRC committee members also concluded that lifetime exposure to

fluoride at a drinking water concentration of 4.0 mg/L (the enforceable standard established by EPA) is likely to increase bone fracture rates in the population, compared with exposure at 1.0 mg/L.<sup>6</sup> Fluoride concentrations used for water fluoridation have been substantially lower than the enforceable standard EPA established to protect against severe skeletal fluorosis.<sup>2,6</sup>

Conclusions of the panel were summarized, along with their rationale, in the *Federal Register*.<sup>67</sup> PHS guidance is advisory, not regulatory, in nature.

## OVERVIEW OF PUBLIC COMMENTS

The public comment period for the Proposed Recommendation for Fluoride Concentration in Drinking Water for the Prevention of Dental Caries lasted for 93 days; it began with publication of the *Federal Register* notice on January 13, 2011, and was extended from its original deadline of February 14, 2011, to April 15, 2011, to allow adequate time for interested organizations and members of the public to respond. Duplicate comments (e.g., electronic and paper submissions from the same source) were counted as one comment. Although the 51 responses received electronically or postmarked after the deadline (midnight ET, April 15, 2011) were not reviewed, all other comments were considered carefully.

Approximately 19,300 responses were received; of these responses, approximately 18,500 (96%) were nearly identical to a letter submitted by an organization opposing community water fluoridation, often originating from the website of that organization; hereafter, these responses are called “standard letters.” Of the remaining 746 unique responses, 79 anecdotes described personal experiences, often citing potentially harmful effects, and 18 consisted of attachments only. Attachments to the unique submissions were examined to ensure that they addressed the recommendation and to determine whether they supported it, opposed it as too low, or opposed it as too high. Although nearly all responses came from the general public, comments also were submitted by organizations, such as those representing dental, public health, or water supply professionals; those that advocate cessation of community water fluoridation; or commercial companies.

Of the unique responses, most opposed the recommendation as still too high and presented multiple concerns. Four CDC scientists (who did not serve on the interagency federal panel) reviewed all unique responses and used an electronic list of descriptors to categorize their contents. Comments were summarized and reported to the full federal panel, along with examples reflecting a range of differing opinions

regarding the new recommendation. The following sections summarize frequent comments and provide the federal panel’s response, divided into three categories: comments that opposed the recommendation as still too high, comments that opposed the recommendation as too low to achieve prevention of dental caries, and comments that supported the recommendation. Data on the approximate numbers of comments received in support of and opposed to the new recommendation are provided for informational purposes. Responses to these comments are based primarily on conclusions of evidence-based reviews and/or expert panels that reviewed and evaluated the best available science.

### Comments that opposed the recommendation as too high

Nearly all submissions opposed community water fluoridation at any concentration; they stated that the new recommendation remains too high, and most asked that all fluoride be removed from drinking water. These submissions included standard letters (about 18,500) and unique responses (about 700 said the new level was too high; of these responses, about 500 specifically asked for all fluoride to be removed). Nearly all of these submissions listed possible adverse health effects as concerns, specifically, severe dental fluorosis, bone fractures, skeletal fluorosis, carcinogenicity, lowered IQ and other neurological effects, and endocrine disruption.

In response to these concerns, PHS again reviewed the scientific information cited to support actions announced in January 2011 by HHS and EPA—and again considered carefully whether or not the proposed recommendations and standards on fluoride in drinking water continue to provide the health benefits of community water fluoridation while minimizing the chance of unwanted health effects from too much fluoride.<sup>4,7,67</sup> After a thorough review of the comments opposing the recommendation, the panel did not identify compelling new information to alter its assessment that the recommended fluoride concentration (0.7 mg/L) provides the best balance of benefit to potential harm.

**Dental fluorosis.** The standard letters stated that the new recommendation would not eliminate dental fluorosis and cited its current prevalence among U.S. adolescents. In national surveys cited by the initial *Federal Register* notice, however, more than 90% of dental fluorosis in the United States is the very mild or mild form, most often appearing as barely visible lacy white markings or spots on the enamel.<sup>54</sup> EPA considers the severe form of dental fluorosis, with staining and pitting of the tooth surface, as the “adverse health effect” to be

prevented.<sup>7</sup> Severe dental fluorosis is rare in the United States, and its prevalence could not be estimated among adolescents in a national survey because there were too few cases among the survey participants examined to achieve statistical reliability.<sup>54</sup> The NRC review noted that prevalence of severe dental fluorosis was near zero at fluoride concentrations <2 mg/L.<sup>6</sup> In addition, the most recent review of community water fluoridation by the Community Preventive Services Task Force concluded that “there is no evidence that [community water fluoridation] results in severe dental fluorosis.”<sup>26</sup>

Standard letter submissions also expressed concern that infants fed formula reconstituted with fluoridated drinking water would receive too much fluoride. If an infant is consuming only infant formula mixed with fluoridated water, there may be an increased chance for permanent teeth (when they erupt at about age 6) to have mild dental fluorosis.<sup>68</sup> To lessen this chance, parents may choose to use low-fluoride bottled water some of the time to mix infant formula (e.g., bottled waters labeled as deionized, purified, demineralized, or distilled, and without any fluoride added after purification treatment; the U.S. Food and Drug Administration requires the label to indicate when fluoride is added). Such guidance currently is found on the websites of both CDC and the American Dental Association.<sup>69,70</sup> The PHS recommendation to lower the fluoride concentration for community water fluoridation should decrease fluoride exposure during the time of enamel formation, from birth through 8 years of age for most permanent teeth, and further lessen the chance for children’s teeth to have dental fluorosis, while keeping the decay prevention benefits of fluoridated water.<sup>34,44,45</sup>

**Bone fractures and skeletal fluorosis.** Some unique comments (about 100) cited fractures or other pathology of bone, while the standard letters expressed concern about skeletal fluorosis and suggested that symptoms of stage II skeletal fluorosis (i.e., a clinical stage associated with chronic pain) are identical to those of arthritis (i.e., sporadic pain and stiffness of the joints). The NRC review found no recent studies to evaluate the prevalence of skeletal fluorosis in U.S. populations exposed to fluoride at the current maximum level of 4.0 mg/L. On the basis of existing epidemiologic literature, the NRC concluded that stage III skeletal fluorosis (i.e., a clinical stage associated with significant bone or joint damage) “appears to be a rare condition in the United States” and stated that the committee “could not determine whether stage II skeletal fluorosis is occurring in U.S. residents who drink water with fluoride at 4 mg/L.”<sup>26</sup>

The NRC also recommended that EPA consider additional long-term effects on bones in adults—stage II

skeletal fluorosis and bone fractures—as well as the health endpoint that had been evaluated previously (i.e., stage III skeletal fluorosis).<sup>6</sup> In response, the EPA Dose–Response Analysis for Non-Cancer Effects noted that, although existing data were inadequate to model the relationship of fluoride exposure and its impact on bone strength, skeletal effects among adults are unlikely to occur at the fluoride intake level estimated to protect against severe dental fluorosis among children. The EPA report concluded that exposure to concentrations of fluoride in drinking water of  $\geq 4$  mg/L appears to be positively associated with the increased relative risk of bone fractures in susceptible populations when compared with populations consuming fluoride concentrations of 1 mg/L.<sup>7</sup> Recently, a large cohort study of older adults in Sweden reported no association between long-term exposure to drinking water with fluoride concentrations up to 2.7 mg/L and hip fracture.<sup>71</sup>

The fluoride intake estimated by EPA to protect against severe dental fluorosis among children during the critical period of enamel formation was determined to be “likely also protective against fluoride-related adverse effects in adults, including skeletal fluorosis and an increased risk of bone fractures.” EPA compared its own risk assessments for skeletal effects with those made both by the NRC in 2006 and by the World Health Organization in 2002.<sup>72</sup> EPA concluded that its own dose recommendation is protective compared with each of these other benchmarks and, thus, is “applicable to the entire population since it is also protective for the endpoints of severe fluorosis of primary teeth, skeletal fluorosis, and increased risk of bone fractures in adults.”<sup>7</sup>

**Carcinogenicity.** Some unique comments (about 100) mentioned concerns regarding fluoride as a carcinogen, and the standard letters called attention to one study that reported an association between osteosarcoma (i.e., a type of bone cancer) among young males and estimated fluoride exposure from drinking water, based on residence history.<sup>73</sup> The study examined an initial set of cases from a hospital-based case-control study of osteosarcoma and fluoride exposure. Findings from subsequent cases were published in 2011. This later study assessed fluoride exposure using actual bone fluoride concentration—a more accurate and objective measure than previous estimates based on reported fluoride concentrations in drinking water at locations in the reported residence history. The later study showed no significant association between bone fluoride levels and osteosarcoma risk.<sup>74</sup> This finding is consistent with systematic reviews and three recent ecological studies that found no association between

incidence of this rare cancer and the fluoride content of community water.<sup>20,23,25,75–78</sup> Although study authors acknowledged the statistical and methodological limitations of ecological analyses, they also noted that their findings were consistent with the hypothesis that low concentrations of fluoride in water do not increase the risk of osteosarcoma development.

A critical review of fluoride and fluoridating agents of drinking water, accepted by the European Commission's Scientific Committee on Health and Environmental Risks (SCHER) in 2011, used a weight-of-evidence approach and concluded that epidemiological studies did not indicate a clear link between fluoride in drinking water and osteosarcoma or cancer in general. In addition, the committee found that the available data from animal studies, in combination with the epidemiology results, did not support classifying fluoride as a carcinogen.<sup>79</sup> Finally, the Proposition 65 Carcinogen Identification Committee, convened by the Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, determined in 2011 that fluoride and its salts have not clearly been shown to cause cancer.<sup>80</sup>

***IQ and other neurological effects.*** The standard letters and approximately 100 unique responses expressed concern about fluoride's impact on the brain, specifically citing lower IQ in children. Several Chinese studies considered in detail by the NRC review reported lower IQ among children exposed to fluoride in drinking water at mean concentrations of 2.5–4.1 mg/L—several times higher than concentrations recommended for community water fluoridation.<sup>81–83</sup> The NRC found that “the significance of these Chinese studies is uncertain” because important procedural details were omitted, but also stated that findings warranted additional research on the effects of fluoride on intelligence.<sup>6</sup>

Based on animal studies, the NRC committee speculated about potential mechanisms for nervous system changes and called for more research “to clarify the effect of fluoride on brain chemistry and function.” These recommendations should be considered in the context of the NRC review, which limited its conclusions regarding adverse effects to water fluoride concentrations of 2–4 mg/L and did “not address the lower exposures commonly experienced by most U.S. citizens.”<sup>6</sup> A recent meta-analysis of studies conducted in rural China, including those considered by the NRC report, identified an association between high fluoride exposure (i.e., drinking water concentrations ranging up to 11.5 mg/L) and lower IQ scores; study authors noted the low quality of included studies and the inability to rule out other explanations.<sup>84</sup> A subsequent review cited this meta-analysis to support its identification of

“raised fluoride concentrations” in drinking water as a developmental neurotoxicant.<sup>85</sup>

A review by SCHER also considered the neurotoxicity of fluoride in water and determined that there was not enough evidence from well-controlled studies to conclude if fluoride in drinking water at concentrations used for community fluoridation might impair the IQ of children. The review also noted that “a biological plausibility for the link between fluoridated water and IQ has not been established.”<sup>79</sup> Findings of a recent prospective study of a birth cohort in New Zealand did not support an association between fluoride exposure, including residence in an area with fluoridated water during early childhood, and IQ measured repeatedly during childhood and at age 38 years.<sup>86</sup>

***Endocrine disruption.*** All of the standard letters and some of the unique comments (about 100) expressed concern that fluoride disrupts endocrine system function, especially for young children or for individuals with high water intake. The 2006 NRC review considered a potential association between fluoride exposure (2–4 mg/L) and changes in the thyroid, parathyroid, and pineal glands in experimental animals and humans. The report noted that available studies of the effects of fluoride exposure on endocrine function have limitations. For example, many studies did not measure actual hormone concentrations, and several studies did not report nutritional status or other factors likely to confound findings. The NRC called for better measurement of exposure to fluoride in epidemiological studies and for further research “to characterize the direct and indirect mechanisms of fluoride's action on the endocrine system and factors that determine the response, if any, in a given individual.”<sup>6</sup> A 2007 review did not find evidence that consuming drinking water with fluoride at the level used in community water fluoridation presents health risks for people with chronic kidney disease.<sup>87</sup>

***Effectiveness of community water fluoridation in caries prevention.*** In addition to citing potential adverse health effects, the standard letters stated that the benefits of community water fluoridation have never been documented in any randomized controlled trial. 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.<sup>20,21,23,25,26</sup> 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.

Standard letters also stated that African American and low-income children would not be protected by the recommendation, as they have experienced more tooth decay than other racial/ethnic groups, despite exposure to fluoride through drinking water and other sources. Data from NHANES do not support this statement and, instead, document a decline in the prevalence and severity of dental caries (tooth decay) across racial/ethnic groups. For example, in 1999–2004, compared with 1988–1994, the percentage of adolescents aged 12–19 years who had experienced dental caries in their permanent teeth, by race/ethnicity, was 54% in African American (down from 63%), 58% in non-Hispanic white (down from 68%), and 64% in Mexican American (down from 69%) adolescents.<sup>11</sup> For adolescents whose family income was less than 100% of the federal poverty level, a similar decline occurred: 66% had experienced dental caries in 1999–2004, down from 72% in 1988–1994. Although disparities in caries prevalence among these adolescent groups remain, the prevalence for each group was lower in 1999–2004 than in 1988–1994. Concurrent with these reductions in the prevalence of dental caries, the percentage of the U.S. population receiving fluoridated water increased from 56% ( $n=144,217,476$ ) in 1992 to 62% ( $n=180,632,481$ ) in 2004. This change represented an increase of more than 36 million people.<sup>36</sup>

**Cost-effectiveness of community water fluoridation.** Some unique comments (about 200) called attention to the cost of water fluoridation or stated that it was unnecessary or inefficient given the availability of other fluoride modalities and the amount of water used for purposes other than drinking. Cost-effectiveness studies that included costs incurred in treating all community water with fluoride additives still found fluoridation to be cost saving.<sup>21,88</sup> Although the annual per-person cost varied by size of the water system (from \$0.50 in communities of  $\geq 20,000$  to \$3.70 for communities of  $\leq 5,000$ , updated to 2010 dollars using the Consumer Price Index [CPI]), it remains only a fraction of the cost of one dental filling. The annual per-person cost savings for those aged 6–65 years ranged from \$35.90 to \$28.70 for larger and smaller communities, respectively (updated to 2010 dollars using CPI dental services).<sup>88</sup> Studies in the United States and Australia also have documented the cost-effectiveness of community water fluoridation.<sup>21,31–33</sup>

**Safety of fluoride additives.** Unique comments (about 300) expressed concern that fluoride is a poison and an industrial waste product; standard letters noted

the lack of specific data on the safety of silicofluoride compounds used by many water systems for community water fluoridation. All additives used to treat water, including those used for community water fluoridation, are subject to a system of standards, testing, and certification involving participation of the American Water Works Association, NSF International, and the American National Standards Institute (ANSI)—entities that are nonprofit, nongovernmental organizations. Most states require that water utilities use products that have been certified against ANSI/NSF Standard 60: Drinking Water Treatment Chemicals—Health Effects (hereinafter, Standard 60) by an ANSI-accredited laboratory. All fluoride products evaluated against Standard 60 are tested to ensure that the levels of regulated impurities present in the product will not contribute to the treated drinking water more than 10% of the corresponding maximum contaminant level established by EPA for that contaminant.<sup>89</sup> Results from 2000–2011, reported on the NSF International website, found that no contaminants exceeded the concentration allowed by Standard 60.<sup>90</sup>

Although commenters expressed concerns about silicofluorides, studies have shown that these compounds achieve virtually complete dissolution and ionic disassociation at concentrations added to drinking water and, thus, are comparable to the fluoride ion produced by other additives, such as sodium fluoride.<sup>89,91,92</sup> At the pH of drinking water, usually 6.5–8.5, and at a fluoride concentration of 1 mg/L, the degree of hydrolysis of hexafluorosilicic acid has been described as “essentially 100%.”<sup>89</sup> Standard 60 provides criteria to develop an allowable concentration when no maximum contaminant level has been established by the EPA. Using this protocol, NSF International calculations showed that a sodium fluorosilicate concentration needed to achieve 1.2 mg/L would result in 0.8 mg/L of silicate, or about 5% of the allowable concentration calculated by NSF International.<sup>90</sup>

SCHER also considered health and environmental risks associated with the use of silicofluoride compounds in community water fluoridation and concurred that in water they are rapidly hydrolyzed to fluoride, and that concentrations of contaminants in drinking water are well below guideline values established by the World Health Organization.<sup>79</sup>

**Ethics of community water fluoridation.** All standard letters and some unique comments (about 200) stated that water fluoridation is unethical mass medication of the population. To determine if a public health action that may encroach on individual preferences is ethical, a careful analysis of its benefits and risks must occur. In the case of water fluoridation, the literature offers

clear evidence of its benefits in reducing dental decay, with documented risk limited to dental fluorosis.<sup>4,7,19–26</sup>

Several aspects of decision-making related to water fluoridation reflect careful analysis and lend support to viewing the measure as a sound public health intervention. State and local governments decide whether or not to implement water fluoridation after considering evidence regarding its benefits and risks. Often, voters themselves make the final decision to adopt or retain community water fluoridation. Although technical support is available from HHS, federal agencies do not initiate efforts to fluoridate individual water systems. In addition, court systems in the United States have thoroughly reviewed legal challenges to community water fluoridation and have viewed it as a proper means of furthering public health and welfare.<sup>93</sup>

#### **Comments that opposed the recommendation as too low**

Several unique comments said that 0.7 mg/L is too low to offer adequate protection against tooth decay. Evidence, however, does suggest that 0.7 mg/L will maintain caries preventive benefits. Analysis of data from the 1986–1987 Oral Health of United States Children survey found that reductions in dental caries plateaued at 0.7–1.2 mg/L of fluoride.<sup>57</sup> In addition, fluoride in drinking water is only one of several available fluoride sources, such as toothpaste, mouth rinses, and professionally applied fluoride compounds.

#### **Comments that supported the recommendation**

Some submissions specifically endorsed lowering the concentration of fluoride in drinking water for the prevention of dental caries. Other commenters asked for guidance on the operational range for implementing the recommended concentration of 0.7 mg/L and on consistent messaging regarding the recommended change. Currently, CDC is reviewing available data and collaborating with organizations of water supply professionals to update operational guidance. In addition, CDC continues to support local and state infrastructure needed to implement and monitor the recommendation. Examples of this support include maintenance of the Water Fluoridation Reporting System; provision of training opportunities for water supply professionals; assisting state and local health agencies with health promotion and public education related to water fluoridation; and funding research and surveillance activities related to dental caries, dental fluorosis, and fluoride intake (in coordination with other federal agencies, including the National Institute of Dental and Craniofacial Research).

#### **MONITORING IMPLEMENTATION OF THE NEW RECOMMENDATION**

Unpublished data from the Water Fluoridation Reporting System show how rapidly the proposed change in recommended concentration has already gained acceptance. In December 2010, about 63% of the population on water systems adjusting fluoride (or buying water from such systems) was at  $\geq 1.0$  mg/L and fewer than 1% were at 0.7 mg/L. By summer 2011—only six months after publication of the draft notice—68% of that population was at 0.7 mg/L and about 28% was at  $\geq 1.0$  mg/L.

Following broad implementation of the new recommendation, enhanced surveillance during the next decade will detect changes in the prevalence and severity of dental caries and of dental fluorosis that is very mild or greater, nationally and for selected sociodemographic groups. For example, the 2011–2012 NHANES included clinical examination of children and adolescents by dentists to assess decayed, missing, and filled teeth; presence of dental sealants; and dental fluorosis. The 2013–2014 examination added fluoride content of home water (assessed using water taken from a faucet in the home), residence history (needed to estimate fluoride content of home tap water for each child since birth), and questions on use of other fluoride modalities (e.g., toothpaste, prescription drops, and tablets). As findings from these and future examinations become available, they can be accessed through the CDC website.<sup>94</sup>

Definitive evaluation of changes in dental fluorosis prevalence or severity associated with reduction in fluoride concentration in drinking water cannot occur until permanent teeth erupt in the mouths of children who drank that water during the period of tooth development. HHS agencies continue to give priority to the development of valid and reliable measures of fluorosis, as well as technologies that could assess individual fluoride exposure precisely. A recent study documented the validity of fingernail fluoride concentrations at age 2–7 years as a biomarker for dental fluorosis of the permanent teeth at age 10–15 years.<sup>95</sup>

#### **CONCLUSIONS**

PHS acknowledges the concerns of commenters and appreciates the efforts of all who submitted responses to the *Federal Register* notice describing its recommendation to lower the fluoride concentration in drinking water for the prevention of dental caries. The full federal panel considered these responses in the context of best available science but did not alter its recommendation that the optimal fluoride concentration



in drinking water for prevention of dental caries in the United States be reduced to 0.7 mg/L, from the previous range of 0.7–1.2 mg/L, based on the following information:

- Community water fluoridation remains an effective public health strategy for delivering fluoride to prevent tooth decay and is the most feasible and cost-effective strategy for reaching entire communities.
- In addition to drinking water, other sources of fluoride exposure have contributed to the prevention of dental caries and an increase in dental fluorosis prevalence.
- Caries preventive benefits can be achieved and the risk of dental fluorosis reduced at 0.7 mg/L.
- Recent data do not show a convincing relationship between water intake and outdoor air temperature. Thus, recommendations for water fluoride concentrations that differ based on outdoor temperature are unnecessary.

Surveillance of dental caries, dental fluorosis, and fluoride intake will monitor changes that might occur, following implementation of the recommendation.

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# ATTACHMENT D

**Dr. Nguyen provided the following in response to the discussion on dose versus toxicity -**

Swiss physician and chemist Paracelsus expressed the basic principle of toxicology 500 years ago as: “All things are poison, and nothing is without poison; only the dose makes a thing not a poison.” This is often condensed to: “The dose makes the poison” as the basic tenet of toxicology. It means that any substance containing toxic properties can cause harm only if it occurs in a high enough concentration. In other words, any chemical—even water and oxygen—can be toxic if too much is ingested or absorbed into the body, especially in acute exposure over a short period of time.

The toxicity of a specific substance depends on a variety of factors, including the weight of the person, how much of the substance a person is exposed to, duration of exposure, as well as the route of exposure. Toxicity from an acute massive ingestion of fluoride can lead to nausea, vomiting, as well as low calcium in the blood because the fluoride anion chelates calcium cations in the blood, potentially resulting in acute medical complications from low blood calcium such as cardiac rhythm disturbance, seizures, muscle spasm.

The concentration of fluoride in toothpaste is >2000X fold higher than that in fluoridated drinking water (optimally set at 0.7 ppm or 0.7 mg/L); therefore, any acute overdose related to fluoride from fluoridated drinking water is next to impossible, and a person would more likely get water toxicity from drinking too much water as opposed to fluoride toxicity. A 70-kg adult eating **eight** 6-oz tubes of a standard fluoridated toothpaste in one-sitting, such as from an intentional overdose or suicide, can experience medical complications because such an acute massive ingestion of fluoride could overwhelm the body’s normal ability to excrete it and lead to significant amounts of calcium being chelated. In contrast, twice-daily usage of fluoride toothpaste for dental hygiene is thousands of orders of magnitudes lower in exposure; therefore, even accidentally ingesting fluoride toothpaste residues while brushing teeth daily would not be expected to produce any clinically significant toxicity given such a low exposure amount.

With respect to chronic toxicity, long-term chronic exposure to significantly elevated fluoride concentration in water over a lifetime can potentially increase the risk for skeletal fluorosis, which is rare in the United States (US). As a result, the United States Environmental Protection Agency (EPA), has set an enforceable maximum contaminant level (MCL) of fluoride in public drinking water systems at 4 mg/dL to protect against severe skeletal fluorosis. Moreover, below the secondary contaminant level that is set at 2 mg/L, severe dental fluorosis is near

zero, which is three times the level of fluoride that's in water fluoridation. Consequently, the lower fluoride concentration of 0.7 mg/dL that is recommended for fluoridation of drinking water for dental health would not be expected to result in any clinically significant skeletal or dental fluorosis.

**Dr. Johnson provided the following in response to his comment on the national survey, NHANES -**

“The National Health and Nutrition Examination Survey ([NHANES](#))”

The National Health and Nutrition Examination Survey (NHANES) is a program of studies designed to assess the health and nutritional status of adults and children in the United States. The survey is unique in that it combines interviews and physical examinations. NHANES is a major program of the National Center for Health Statistics (NCHS). NCHS is part of the Centers for Disease Control and Prevention (CDC) and has the responsibility for producing vital and health statistics for the Nation.

The NHANES program began in the early 1960s and is an ongoing program.

1. “Water Fluoridation and Dental Caries in U.S. Children and Adolescents”, Slade et al, [J Dent Res](#). 2018 Sep;97(10):1122-1128

A 2018 study on water fluoridation and dental caries in U.S. children and adolescents utilized fluoridation levels, by county as reported to the CDC and merged it with National Health and Nutrition Examination Survey (NHANES) data from a 10 year period. This study included 7,000 children 2-8 years old were used to calculate the cavities prevented by fluoridation in their primary teeth (baby teeth) and 12,604 children ages 6-17 years old were used to calculate cavity reductions in their permanent teeth. It is a current study and shows how well fluoridation reduces cavities in children and young adults.

**Results:**

- Primary teeth – 30% cavity reductions in fluoridated vs non-fluoridated communities;
  - Permanent teeth: 12% cavity reductions in permanent teeth by age 17 years old.
2. “Recent trends in dental caries in U.S. children and the effect of water fluoridation”, Brunelle, Carlos, [J Dent Res](#). 1990 Feb:69 Spec No:723-7. This is the largest study ever conducted; had a sample size of 39,207 U.S. school children ages 5-17.
    - The chart below shows that, on average, children who lived their whole lives in fluoridated communities experienced less tooth decay in their



permanent teeth than did children of the same age who lived their whole lives in non-fluoridated communities. In general, the amount of tooth decay prevented by water fluoridation increased with age, from an average of 0.2 fewer decayed, missing, or filled permanent tooth surfaces (DMFS) at age 7 to an average of 1.6 fewer DMFS at age 17.

- **Note:** The chart shows the difference between cavity experiences in those who grew up with water fluoridation minus those without water fluoridation. The differences become more pronounced as the children approached 17 years of age. The slope of the difference begins to rise exponentially, meaning that the benefits of fluoridation increase as we continue to reap the benefits as adults.
- The reason for this is that the first permanent molars come in around 6 years of age. The second permanent molars come in around 12 years of age. The most common surface for cavities to occur on these permanent molars is on their chewing surfaces. As can be seen from the chart, the cavity rate of the permanent first molars is low when they first come in. However, by the time that they've been in the mouth at age 12 years old (when the permanent second molars come in), the cavity differences have increased steadily. When the permanent second molars come in (~age 12 years), they have very few cavities. However, by 17 years old, both the permanent first and second permanent molars begin to have an exponential rise in the difference between those who grew up with fluoridation vs those who did not grow up with fluoridation.
- This accounts for the 12% cavity reductions in the study listed in A above. The longer that these teeth are in, the more the cavity rate approaches the 25% cavity reductions that we see prevented by community water fluoridation for adults and children.

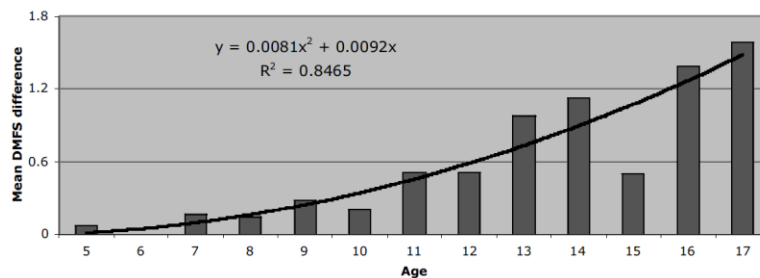


Figure 1—Data from Table 6 of Brunelle and Carlos.<sup>16</sup> Difference in Mean Decayed, Missing Due to Caries and Filled Permanent Tooth Surfaces (DMFS) between Children with Continuous Exposure to Water Fluoridation and Those with No Such Exposure.

**The panelists provided the following list in response to the inquiry of studies from the last 10 years on community fluoridation -**

*Studies demonstrating increases in cavities when fluoridation was stopped:*

- Israel; [2022](#)
- Calgary/Edmonton, Alberta; [2022](#)
- Juneau/Anchorage, Alaska [2022](#)
- Windsor/Tecumseh, Ontario; [2018](#)

*Studies showing cavity reductions from the start-up of fluoridation vs non-fluoridating communities:*

- England, [2022](#)
- Australia, [2015](#)
- Systematic review; [2013](#)

*Studies providing reviews or systematic reviews of the benefits of fluoridation in communities with and without water fluoridation:*

- Systematic Review; [2023](#)
- Systematic Review; [2022](#)
- Systematic Review, U.S.; [2020](#)
- U.S.; [2018](#)
- Korea; [2017](#)
- Australia; [2016](#)
- Systematic Review; [2015](#)
- Ireland, [2015](#)
- Public Health England, [2014](#)
- Australia; [2013](#)

**Dr. Johnny Johnson provided the following information on alternative fluoridation practices.**

Approximately 13.5 million people in Europe are served by community water fluoridation. At 6 million, just over 10% of the United Kingdom receives a fluoridated supply of water. The United Kingdom has recently moved fluoridation decisions to the equivalent to our Department of Health and it is uncertain as to how this will impact number moving forward. At nearly 4.2 million, approximately 10% of Spain's population is now receiving a fluoridated supply of water. The Irish Republic has 3.4 million, or nearly 2/3<sup>rd</sup> of their population being served by community fluoridation. Other countries with community water fluoridation programs include Australia, New Zealand, Israel, Singapore, Hong Kong, and Brunei.

Alternatives such as salt and milk are utilized worldwide. Approximately 60 million people across Europe use fluoridated salt (2013 data, [American Dental Association](#)) whereas nearly 1 million children, worldwide received fluoridated milk (2013 data – [American Dental Association](#)). Additionally, school-based topical fluoride rinses and toothpaste, in-class instructions on oral hygiene, coupled with free dental care for the school children during the school year.

**Dr. Staniland provided the following testimony of his presentation and articles of interest -**

First, this is a list of the studies that were printed and submitted to the Committee prior to the presentations beginning on November 7, 2023.

Fluoride Exposure and Age of Menarche: Potential Differences Among Adolescent Girls and Women in the United States

<https://fluoridealert.org/wp-content/uploads/malin-2021.pdf>

Fluoride exposure and sleep patterns among older adolescents in the United States: a cross-sectional study of NHANES 2015–2016

<https://ehjournal.biomedcentral.com/articles/10.1186/s12940-019-0546-7>

Plasma and water fluoride levels and hyperuricemia among adolescents: A cross-sectional study of a nationally representative sample of the United States for 2013–2016

<https://fluoridealert.org/wp-content/uploads/wei-2020.pdf>

Fluoride exposure and kidney and liver function among adolescents in the United States: NHANES, 2013–2016

<https://www.sciencedirect.com/science/article/pii/S0160412019309274>

Fluoride in Drinking Water, Diet, and Urine in Relation to Bone Mineral Density and Fracture Incidence in Postmenopausal Women

<https://ehp.niehs.nih.gov/doi/10.1289/EHP7404>

Fluoride exposure and thyroid function among adults living in Canada: Effect modification by iodine status

<https://pubmed.ncbi.nlm.nih.gov/30316182/>

Fluoride exposure from infant formula and child IQ in a Canadian birth cohort

<https://www.sciencedirect.com/science/article/pii/S0160412019326145?via=ihub>

Prenatal Fluoride Exposure and Cognitive Outcomes in Children at 4 and 6–12 Years of Age in Mexico

<https://ehp.niehs.nih.gov/doi/10.1289/ehp655>

Prenatal fluoride exposure and attention deficit hyperactivity disorder (ADHD) symptoms in children at 6–12 years of age in Mexico City

<https://www.sciencedirect.com/science/article/pii/S0160412018311814?via%3Dihub&eType=EmailBlastContent&eld=ba3191f8-9c43-47c3-ac2d-9131ae604322>

Association Between Maternal Fluoride Exposure During Pregnancy and IQ Scores in Offspring in Canada

<https://jamanetwork.com/journals/jamapediatrics/fullarticle/2748634?eType=EmailBlast>

[Content&eld=ba3191f8-9c43-47c3-ac2d-9131ae604322](https://www.sciencedirect.com/science/article/pii/S0160412019326145?via%3Dihub&type=EmailBlastContent&eld=ba3191f8-9c43-47c3-ac2d-9131ae604322)

Fluoride exposure from infant formula and child IQ in a Canadian birth cohort  
<https://www.sciencedirect.com/science/article/pii/S0160412019326145?via%3Dihub&type=EmailBlastContent&eld=ba3191f8-9c43-47c3-ac2d-9131ae604322>

Iodine Status Modifies the Association between Fluoride Exposure in Pregnancy and Preschool Boys' Intelligence  
<https://pubmed.ncbi.nlm.nih.gov/35889877/>

**The panel presentations were cut short and no questions were asked about these or any other health issues. These studies were copied from this document...**

Teeth are obviously not the only tissues in the body that are harmed by, or accumulate, fluoride. NHANES data has been used in recent published peer-reviewed studies to link fluoridated water with a number of additional side effects, including earlier [onset of menstruation](#) for black teens, [sleep disorders](#) in adolescents, [increased uric acid levels](#) in the blood, and [kidney and liver impairment](#) in adolescents.

Additional studies on fluoridation have also recently found [higher rates of hip fractures](#), disruption of the [endocrine system](#), and increased rates of [hypothyroidism](#).

There is also now a large body of government-funded studies linking early life exposure to [neurotoxicity](#), including [during infancy](#). The CDC must take action to warn both pregnant women and parents of formula-fed infants about the recent NIH-sponsored research indicating that fluoride in drinking water poses a risk to the developing brain at the exposure levels experienced in fluoridated communities, both in utero and during early infancy. The CDC's lack of appropriate action to protect children is an alarming disregard for science and disrespect for the welfare of U.S. citizens.

It has now been six years since the first high quality US-government funded study ([Bashash et al., 2017](#)) found an association between fetal exposure to fluoride and lowered IQ, five years since a government-funded study found an increase in ADHD symptoms associated with in utero exposure to fluoride ([Bashash et al., 2018](#)), four years since the findings in Bashash's study were repeated by another US-government funded study ([Green et al., 2019](#)), and 3 years since a third US-government-funded study ([Till et al., 2020](#)) found that bottle-fed infants in fluoridated communities in Canada had a significantly lowered IQ compared to bottle-fed infants in non-fluoridated communities.

**More recently, the CDC has ignored:**

- A study from Canada published last summer in the journal *Nutrients* found that pregnant women who had low iodine levels and elevated fluoride had boys who suffered an average IQ loss of 9.3 IQ points [[Goodman 2022](#)]. Artificially

fluoridated drinking water was the main source of the fluoride. To put this huge 9-point IQ loss from fluoride into perspective, studies show that a pregnant woman smoking 20+ cigarettes each and every day during their pregnancy can cause less of an IQ loss for the child than fluoride, averaging [about 6.2-points lost](#).

- Experts in environmental toxins, including the former Director of the National Toxicology Program, Dr. Linda Birnbaum, [published an op-ed](#) calling for policy makers to look at the science and take action to protect pregnant women and their children.
- Famed Harvard researcher Phillippe Grandjean, known for helping warn the world about the effects of arsenic, mercury, and PFOAs, conducted the first benchmark dose analysis in 2020 on maternal fluoride exposure and neurotoxicity to the fetus, which was published in the journal Risk Analysis ([Grandjean, 2021](#)). Benchmark dose analyses are used by the Environmental Protection Agency (EPA) and toxicologists to determine at what level a substance starts to cause harm. The analysis confirmed that extremely low fluoride exposure during pregnancy impairs fetal brain development, finding that a maternal urine fluoride concentration of only 0.2mg/L — which coincides with the level in the water (0.2ppm) — was enough to lower IQ by at least 1 point.

(Abstract) Grandjean

A Benchmark Dose Analysis for Maternal Pregnancy Urine-Fluoride and IQ in Children

<https://onlinelibrary.wiley.com/doi/abs/10.1111/risa.13767>

**On to the Questions cited in this Draft:**

***What is the chemical make-up of the fluoride used by Dallas? When it is dissolved in water, what compounds are released?***

The following phrase/paragraph should be amended:

*“These fluoride ions are left and are responsible for the dental health benefits.”*

Rather, it should include all the information below:

These fluoride ions are left and the fluorine ions are responsible for potential dental health benefits **and for potential adverse health effects**.

Fluorine is the most reactive element on the Periodic Table of Chemistry. Thus, it can react with many body processes, not just the teeth and bones. The fluorine ions can go anywhere in the body, including the brain.

<https://www.britannica.com/science/fluorine/Production-and-use>

The EPA classifies Hydrofluorosilicic Acid as a Hazardous Waste. The EPA imposes large fines for those entities which improperly store, transport or dispose of Hydrofluorosilicic Acid because it is a Hazardous Waste.

The EPA Website:

**EPA guidance manual on the RCRA "Regulation of Recycled Hazardous Waste"**

Page 206 -207 – (Handwritten numeral 2-170) – Hydrofluorosilicic Acid

<https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=2000EG87.TXT>

<https://nepis.epa.gov/Exe/ZyPDF.cgi/2000KO9S.PDF?Dockey=2000KO9S.PDF>

Screenshot

Image: <https://imageprocessor.digital.vistaprint.com/crop/0,0,1147x1918/maxLength/1000/https://uploads.documents.cimpress.io/v1/uploads/8bc7fcf6-27f2-4583-ad3f-7c099cba0ba1~110/original?tenant=vbu-digital>

At The Texas Dental Association website on “Community Water Fluoridation Facts and Strategy” in “Topic 5 – The Process of Water Fluoridation”, Hydrofluorosilicic Acid is discussed by quoting Mosaic.

<https://www.tda.org/member-center/fluoride-facts>

Mosaic was once a vendor of Hydrofluorosilicic Acid for Dallas Water Utilities. Around that time period, the shipments to DWU had an accompanying sheet showing the quantities of major contaminants.

When the phosphate ore is processed to extract uranium and/or phosphate for fertilizer, toxic fluorine gases and other contaminants are emitted. These gases are scrubbed with water to prevent them from entering the atmosphere. This resulting slurry sludge is Hydrofluorosilicic Acid, along with many contaminants. These contaminants include arsenic, lead, mercury, radioactive particulates, cadmium, and more.

In 2015, Mosaic was fined 2 billion dollars for improperly handling massive amounts its hazardous waste. A Google search will also reveal stories about Mosaic and radioactive water.

PFAS and Hydrofluorosilicic Acid

Hydrofluorosilicic Acid is an extremely acidic, highly reactive, highly corrosive, hazardous toxic waste substance; one that melts glass, melts concrete, and melts metal.

Because Hydrofluorosilicic Acid is so very corrosive (melting glass, concrete and steel), **it must be transported and stored in specially lined containers.**

Often, PFAS “Forever Chemicals” are utilized as a coating to guard against corrosion.

**This raises a serious concern...**

**Can parts of these PFAS “Forever Chemicals” flake off into solution with the Hydrofluorosilicic Acid?**

If so, then PFAS chemicals are being inserted to the drinking water during water fluoridation.

Fluorosilicates are a breed of chemical with special properties and interactions. Many studies have been done on fluorosilicates, some with alarming health implications (e.g. lead, aluminum.)

One example is this article with the linked scientific study:

**Chloramine + Lead Pipes + Fluoride = Contaminated tap water**

<https://www.ewg.org/news-insights/news/chloramine-lead-pipes-fluoride-contaminated-tap-water#.WzTEE6-0VKZ>

***Additionally: What is the chemical make-up of the fluoride used by Dallas? When it is dissolved in water, what compounds are released?***

The claims that the hydrofluorosilicic acid (HFS) is natural and **entirely dissociates are false**. The HFS is primarily the waste product of phosphate fertilizer plants in the U.S. and Mexico and metal factories in China. The fluoride product is invariably polluted with arsenic, lead, aluminum, barium, cadmium, etc., all of which are dangerous to consumer health and the environment. Some of the chemical does not dissociate and some of the chemical that does dissociate binds with other metal contaminants to create new toxins, like Aluminum Fluoride which is implicated in neurodegenerative conditions like dementia.

- Russ TC, et al. (2020) [Aluminium and fluoride in drinking water in relation to later dementia risk](#). The British Journal of Psychology
- Mullenix PJ. (2014) [A new perspective on metals and other contaminants in fluoridation chemicals](#). International Journal of Occupational and Environmental Health.
- Sauerheber R. (2013) [Physiologic Conditions Affect Toxicity of Ingested Industrial Fluoride](#). Journal of Environmental and Public Health.
- Sawan RM, et al. (2010) [Fluoride increases lead concentrations in whole blood and in calcified tissues from lead-exposed rats](#). Toxicology.
- Varner JA, et al. (1998) [Chronic administration of aluminum–fluoride or sodium–fluoride to rats in drinking water: alterations in neuronal and cerebrovascular integrity](#). Brain Research. V
- Westendorf, Johannes. (1975) [The Kinetics of Acetylcholinesterase Inhibition and the Influence of Fluoride and Fluoride Complexes on the Permeability of Erythrocyte Membranes](#) [English Translation]
- Barber JC, Farr. TD (1970) [Fluoride Recovery From Phosphorus Production](#). Chemical Engineering Progress

***Why does Dallas maintain a 0.7 mg/L level?***

Although there are policy and regulations regarding the community fluoridation with a water concentration of 0.7 ppm, it is an optional policy based on several assumptions, i.e. that the individual adult dose will not exceed 0.7 mg/L per day and that fluoride exposure while young hardens the teeth and makes them more impervious to decay. Moreover, the assumptions that this small exposure has a topical benefit and has no



confirmed adverse effects are discredited. In other words, all of the underlying assumptions for fluoridation are faulty.

Consider that diabetics, kidney patients, athletes and others who routinely consume triple (or more) water, consume triple (or more) the individual dose which means their dose is 2.1 mg/L or more. (A mg/L dose is the equivalent of a ppm water concentration.) The scientific evidence is that these doses are harmful to brains, kidneys, thyroids, and even teeth.

Moreover, recent studies determine that 0.28 mg/L is harmful to the fetus in the womb. It has been known since the 1950s and confirmed in this century that fluoride not only passes into the womb, but concentrates in the placenta and amniotic fluid.

- Grandjean P, et al. (2023) [Dose dependence of prenatal fluoride exposure associations with cognitive performance at school age in three prospective studies](#). Eur J Public Health
- Kheradpisheh Z, et al. (2018) [Impact of Drinking Water Fluoride on Human Thyroid Hormones: A Case- Control Study](#). Scientific Reports.
- Martín-Pardillos A, et al. (2014) [Effect of water fluoridation on the development of medial vascular calcification in uremic rats](#). Toxicology.
- Müller F, et al. (2010) [Elemental depth profiling of fluoridated hydroxyapatite: saving your dentition by the skin of your teeth?](#) Langmuir.
- Gardner DE, et al. (1952) [The fluoride concentration of placental tissue as related to fluoride content in drinking water](#). Science.

### ***What was the revision process for lowering the recommendation in community water fluoridation levels?***

The EPA sets the SMCL at 2.0 ppm to protect teeth. The 2015 reduction to 0.7 ppm is a primarily a political move to address the epidemic of dental fluorosis which afflicts more than half of American teens, albeit much of it on back teeth. Approximately 12% of the population will seek costly cosmetic dentistry to address those stained and brittle teeth that are visible, per both the prestigious 2000 York Review and 2015 Cochrane Report, neither of which found evidence of fluoridation benefit to adults or SES benefit.

Even the pro-fluoridation researchers in the Iowa Fluoride Study (IFS) have repeatedly found that determining an optimal concentration is problematic and “achieving a caries-free status may have relatively little to do with fluoride intake, while fluorosis is clearly more dependent on fluoride intake.”

- Umar MF. (2023) [A Systematic Review on Water Fluoride Levels Causing Dental Fluorosis](#). Sustainability
- Wiener RC, et al. (2018) [Dental Fluorosis over Time: A comparison of National Health and Nutrition Examination Survey data from 2001-2002 and 2011-2012](#). J Dent Hyg

- Ihezor-Ejiofor Z, (2015) et al. [Water fluoridation for the prevention of dental caries](#). Cochrane Database of Systematic Reviews
- Warren JJ, et al. (2008) [Considerations on Optimal Fluoride Intake using Dental Fluorosis and Dental Caries Outcomes – A Longitudinal Study](#). Journal of public health dentistry
- McDonagh MS, et al. (2000) [Systematic review of water fluoridation](#). BMJ

***Why is dose important when determining toxicity?***

Dr. Nguyen is apparently ignorant of the toxicity of fluoride, the difference between toxic and lethal, and the stages of skeletal fluorosis.

Dental fluorosis is indisputable visible proof of fluoride poisoning while young, and about half of those with dental fluorosis also have heart irregularities. Many thyroid and kidney doctors advise their patients to avoid fluoridated water and foods prepared with fluoridated water and have done so for decades since it is well known that fluoride interferes with thyroid hormones and is toxic to kidneys.

Researchers in a Public Health Service controlled dose study advised the PHS in 1956 and 1961 that a portion of the “allergic” population would have acute reactions to even low dose exposure which would include gastrointestinal illness, rashes, and neurological symptoms. Those researchers advised the PHS that it is one thing to drop a subject from a controlled dose trial, but it would be an entirely other thing for people to avoid fluoride if their water supply was fluoridated.

Americans suffer arthritis at younger ages and knee replacement (as I commented in my presentation) due to crippling osteoarthritis is also common. These are consistent with skeletal fluorosis which literally means bones poisoned by fluoride.

The 2006 National Research Council advised that the EPA MCL/MCLG was not protective of the population and should be lowered. They also advised that there was no evidence of safety of susceptible populations (as mentioned above) at any dose. The EPA has failed to act. However, based on science published in recent years, the EPA is being sued in federal court in a case that will be heard the first two weeks of February specific to the evidence of pre- and post-natal developmental neurotoxicity from fluoride exposure. An [October 2023 Interview](#) with Attorney Michael Connett included clips of depositions with EPA, CDC and NSF leadership who admitted they did not have safety data.

- Veneri F, et al (2023) [Fluoride exposure and cognitive neurodevelopment: Systematic review and dose- response meta-analysis](#). Environ Res.
- Lindsay SE (2023). [Community Water Fluoridation and Rate of Pediatric Fractures](#). J Am Acad Orthop Surg Glob Res Rev
- Meng X et al. (2023) [Relatively low fluoride in drinking water increases risk of knee osteoarthritis \(KOA\): a population-based cross-sectional study in China](#). Environ Geochem Health

- Garcia ALH, et al. (2022) [Fluorosilicic acid and cotinine, separately and in combination, induce genotoxicity and telomeric reduction in human osteoblast cell line MG63](#). Mutat Res Genet Toxicol Environ Mutagen
- Zhou J, et al. (2022) [Necessity to Pay Attention to the Effects of Low Fluoride on Human Health: an Overview of Skeletal and Non-skeletal Damages in Epidemiologic Investigations and Laboratory Studies](#). Biol Trace Elem Res.
- Helte E, et al. (2021) [Fluoride in Drinking Water, Diet, and Urine in Relation to Bone Mineral Density and Fracture Incidence in Postmenopausal Women](#). Environ Health Perspect
- Selmin Karademir, et al. (2011). [Effects of fluorosis on QT dispersion, heart rate variability and echocardiographic parameters in children](#). Anatol J Cardiol.
- [Fluoride in Drinking Water: A Scientific Review of EPA's Standards](#). Washington, DC: The National Academies Press 2006.
- Feltman R, Kosel G. (1961) [Prenatal and postnatal ingestion of fluorides - Fourteen years of investigation - Final report](#). Journal of Dental Medicine
- Feltman R. (1956) [Prenatal and postnatal ingestion of fluorides - A Progress Report](#). Dental Digest.

**ADDITIONALLY, Why is dose important when determining toxicity?  
“With respect to chronic toxicity....”**

All recipients and customers of Dallas Water Utilities drinking water must absolutely be made aware of this non-controversial fact presented by the National Institutes of Health (NIH) and many studies:

***“In adults, about 50% of absorbed fluoride is retained, and bones and teeth store about 99% of fluoride in the body. The other 50% is excreted in urine. In young children, up to 80% of absorbed fluoride is retained because more is taken up by bones and teeth than in adults.”***

<https://ods.od.nih.gov/factsheets/Fluoride-HealthProfessional/>

**Consumed fluoride accumulates in the body, building up year after year. It is next to impossible to remove this fluoride build-up.**

**EPA - Substantial Evidence of Developmental Neurotoxicity - Fluoride**

Around 2008, the EPA started a science inventory with “Building a Database of Developmental Neurotoxicants: Evidence from Human and Animal Studies”. Fluoride was and is listed in **“Chemicals with Substantial Evidence of Developmental Neurotoxicity”**.

2013 version: <https://fluoridealert.org/wp-content/uploads/mundy-epa.neurotoxicant.pdf>

Screenshot image: <https://uploads.documents.cimpress.io/v1/uploads/4ef614c9-bef9-41c8-b569-e29786fe398d~110/original?tenant=vbu-digital>

**There is not a single study conducted in pregnant women showing safety for water fluoridation.**

**It is deceptive to say that fluoridation is safe.**

**Studies with pregnant women have demonstrated that fluoride presents an unreasonable risk.**

## **EPA Fluoride Lawsuit**

This is the first time in history that a citizens group has ever taken the EPA to trial under TSCA (Toxic Substances Control Act.)

<https://fluoridealert.org/articles/fluoride-lawsuit-update-nov-2023-new-details-trial-dates/>

Among the expert witnesses for the final trial dates are some world famous toxicologists, including Dr. Philippe Grandjean and Dr. Bruce Lanphear.

Most egregious is the fact that the Quality of Life, Arts and Culture Committee never once asked the November 7 panel about fluoride's toxicity.

Dr. Bruce Lanphear is world famous. Google search his name and news. The Quality of Life, Arts and Culture Committee missed an opportunity of a lifetime not questioning him. Dr. Lanphear was able to hear the Chairman, his responses could have been made in writing, but none were asked for the record.

### ***Are there any national surveys showcasing a comparison of communities with water fluoridation vs. those without?***

It has been known at least since the 1980s that non-fluoridated communities and fluoridated communities have basically equivalent cavity experience. However, **there is always the opportunity to cherry pick and misrepresent findings.**

For example, the dental research team of Slade & Sanders who used NHANES data from the CDC to document a comparative benefit also address the absolute benefit when they write, "When considered at the level of an individual, these effect estimates represent clinical benefits that are either small (1.3 fewer dfs per child) or negligible (0.3 fewer DMFS per child)." These **authors try to argue that multiplying out the "negligible" cavity savings of a fraction of a single surface is considerable. That logic is the same as claiming if one woman can make a baby in nine months, nine women can make a baby in one month.**

**This same team did another NHANES study that found that children drinking tap water in fluoridated communities have high lead levels in their bodies. Then they argued that cavities are a more immediate and tangible concern than lead poisoning which takes years to manifest.**

- G.D. Slade, W.B. Grider, W.R. Maas, A.E. Sanders. (2018) [Water Fluoridation and Dental Caries in U.S. Children and Adolescents](#). Journal of Dental Research.
- Sanders AE, Slade GD. (2017) [Blood Lead Levels and Dental Caries in U.S. Children Who Do Not Drink Tap Water](#). American Journal of Preventive Medicine.

Two 1980s studies used the same deceptive logic to advance their agenda. See: "[New Studies Cast Doubt on Fluoridation Benefits](#)" by Bette Hileman, Chemical & Engineering News.1989.

- In a sample of 1,500 school children, there was no difference in cavity experience in grades 2 through 5, but a small difference in the lowest SES of first graders. The first graders in the non-fluoridated community had an average of two cavities while those in the fluoridated communities had an average of one and a half cavities. A half cavity difference in a subset of perhaps twenty children out of 1,500 and the claim was “25% reduction.”
- A national survey of 39,000 American school children resulted in an odd report on a subset that found an 18% reduction or 25% increase. When a researcher managed to get ahold of the entire set of data, he found there was natural variation between the communities. Some non-fluoridated communities did much better than fluoridated communities.

The NHANES data is more important for the differences it shows between fluoridated and non-fluoridated communities that has little or nothing to do with teeth. See excerpts from peer-reviewed studies published in medical and scientific journals using those national surveys of Americans:

1. **INFLAMMATION**: “Our finding that neutrophils and monocytes are associated with higher plasma fluoride in U.S. children and adolescents is consistent with animal data showing fluoride related effects of increased inflammation.” (Den Besten et al. 2022)
2. **KIDNEYS**: “Water fluoridation results in higher plasma fluoride levels in those with lower renal function. How routine water fluoridation may affect the many millions of Americans with Chronic Kidney Disease, who are particularly susceptible to heavy metal and mineral accumulation, needs to be further investigated.” (Danzinger et al. 2022)
3. **LIVERS & KIDNEYS**: “Fluoride exposure may contribute to complex changes in kidney and liver related parameters among U.S. adolescents. As the study is cross-sectional, reverse causality cannot be ruled out; therefore, altered kidney and/or liver function may impact bodily fluoride absorption and metabolic processes.” (Malin et al. 2019)
4. **BLOOD PRESSURE**: “This study suggested that fluoride exposure may affect childhood blood pressure.” (Guo et al. 2022)
5. **LOW BIRTH WEIGHT & ENVIRONMENTAL JUSTICE**: “Our findings suggest a significant association between excess water fluoride exposure (>0.7 ppm) and LBW weight in Hispanic women, independent of established LBW risk factors.” (Arun 2022)
6. **DENTAL FLUOROSIS**: “The prevalence of dental fluorosis was 70% in the U.S. children and adolescents in survey of NHANES 2015–2016.” (Dong et al. 2021)
7. **REPRODUCTIVE HEALTH**: “Median (IQR) water and plasma fluoride levels were 0.48 (0.53) mg/L and 0.34 (0.30)  $\mu$ mol/L respectively. An IQR increase in water fluoride was associated with a 3.3 month earlier first menstrual period (B= -0.28, 95%CI: -0.54, -0.02, p = 0.05). Additionally, we observed a significant interaction between plasma fluoride and race/ ethnicity in association with age of menarche (p = 0.01). For non-Hispanic black females, each IQR increase in

plasma fluoride was associated with a 5-month earlier age of menarche (B=-0.42, 95%CI: -0.61, -0.23, p < 0.001).” (Maiin et al. 2021)

8. **SLEEP PATTERNS**: “Fluoride exposure may contribute to changes in sleep cycle regulation and sleep behaviors among older adolescents in the US. Additional prospective studies are warranted to examine the effects of fluoride on sleep patterns and determine critical windows of vulnerability for potential effects.” (Malin et al. 2019)

**But as far as cavities go, I suggest looking at these studies:**

1. British Columbia ([2001](#)): This Canadian study of over 6,000 school children found that “Decay **decreased over time in the fluoridation-ended community while remaining unchanged in the fluoridated community**”
2. Germany ([2000](#)): Only a handful of towns were ever fluoridated in Germany as an experiment, but community fluoridation was discontinued. The dental researchers reported: “**In contrast to the anticipated increase in dental caries following the cessation of water fluoridation, a significant fall in caries prevalence was observed. This trend corresponded to the national caries decline and appeared to be a new population-wide phenomenon.**”
3. Cuba ([2000](#)): Study performed seven years after cessation among low income children found a dramatic decrease in cavities and an increase in the number of children with no cavities. **The hypothesis for their findings was the twice a month fluoride rinses in school were more beneficial than fluoridation.**
4. North Carolina ([2000](#)): Researchers followed up on children between age 1 and 3 after an eleven month gap in fluoridation. **They found no increase in cavities, but a significant decrease in dental fluorosis.**
5. Finland ([1998](#)): **There was no increased decay after three years. Moreover, the researchers checked with dental offices and found that topical treatments had “decreased sharply.”**
6. Australia ([1991](#)): This study looked at the impact of cessation of fluoride supplementation in non-fluoridated communities. It found, ““Dental fluorosis seems to have fallen parallel with a reduction of discretionary intake from supplements and toothpaste. **No increase in dental caries experience was recorded.**”

***Are there any studies within the last 10 years that showcase the impact of community fluoridation?***

Per answers above, there are NHANES and scores of other studies showing the damage caused by fluoridation to bodies, bones and brains. But there are also government reports and questionable studies by biased actors that claim safety and dental benefit. **The following refutes two of them:**

- Calgary ([2017](#)): **Even the pro-fluoridation author admitted that cavities increased in primary teeth in both cities and decreased in permanent teeth in the fluoridation ended city:** “Some of the coverage was positive and accurate, but in other cases the study findings were mis-reported and the

conclusions overstated; for example, suggesting that 'cavities spiked since fluoridation was stopped'. There was no spike but rather a gradual increase, and the trend observed was not since fluoridation was stopped, but rather over a time period during which cessation occurred: 2004/05 to 2013/14 (cessation occurred in 2011). In terms of permanent teeth, caries experience actually decreased (improved) in Calgary during the time frame of the study." - Lindsay McLaren in MOSAIC Fall 2017 newsletter.

- Juneau ([2018](#)): There was no control cited in this dissertation and the author used Medicaid reimbursements as a proxy for cavities. **Medicaid payments and dentists accepting Medicaid had apparently increased in the nine years between the two measurements.** It may have got the author her degree, but it is a typical "garbage in - garbage out" study.

**Ultimately**, as far as cavities go the only things we can say with certainty are that 1) cavity rates decreased in the 20th century regardless of fluoridation status and are increasing in the 21st century regardless of fluoridation status, and that 2) **fluoride is a poison even in low doses which exerts sinister effects on thyroids, kidneys, hearts, bones, guts, and brains.**

**It is my considered expert medical opinion, shared by many other doctors, dentists and scientists who have done their due diligence, that fluoridation damages the health of consumers.**

# Association Between Maternal Fluoride Exposure During Pregnancy and IQ Scores in Offspring in Canada

Rivka Green, MA; Bruce Lanphear, MD; Richard Hornung, PhD; David Flora, PhD; E. Angeles Martinez-Mier, DDS; Raichel Neufeld, BA; Pierre Ayotte, PhD; Gina Muckle, PhD; Christine Till, PhD

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**IMPORTANCE** The potential neurotoxicity associated with exposure to fluoride, which has generated controversy about community water fluoridation, remains unclear.

**OBJECTIVE** To examine the association between fluoride exposure during pregnancy and IQ scores in a prospective birth cohort.

**DESIGN, SETTING, AND PARTICIPANTS** This prospective, multicenter birth cohort study used information from the Maternal-Infant Research on Environmental Chemicals cohort. Children were born between 2008 and 2012; 41% lived in communities supplied with fluoridated municipal water. The study sample included 601 mother-child pairs recruited from 6 major cities in Canada; children were between ages 3 and 4 years at testing. Data were analyzed between March 2017 and January 2019.

**EXPOSURES** Maternal urinary fluoride ( $MUF_{SG}$ ), adjusted for specific gravity and averaged across 3 trimesters available for 512 pregnant women, as well as self-reported maternal daily fluoride intake from water and beverage consumption available for 400 pregnant women.

**MAIN OUTCOMES AND MEASURES** Children's IQ was assessed at ages 3 to 4 years using the Wechsler Primary and Preschool Scale of Intelligence-III. Multiple linear regression analyses were used to examine covariate-adjusted associations between each fluoride exposure measure and IQ score.

**RESULTS** Of 512 mother-child pairs, the mean (SD) age for enrollment for mothers was 32.3 (5.1) years, 463 (90%) were white, and 264 children (52%) were female. Data on  $MUF_{SG}$  concentrations, IQ scores, and complete covariates were available for 512 mother-child pairs; data on maternal fluoride intake and children's IQ were available for 400 of 601 mother-child pairs. Women living in areas with fluoridated tap water ( $n = 141$ ) compared with nonfluoridated water ( $n = 228$ ) had significantly higher mean (SD)  $MUF_{SG}$  concentrations (0.69 [0.42] mg/L vs 0.40 [0.27] mg/L;  $P = .001$ ; to convert to millimoles per liter, multiply by 0.05263) and fluoride intake levels (0.93 [0.43] vs 0.30 [0.26] mg of fluoride per day;  $P = .001$ ). Children had mean (SD) Full Scale IQ scores of 107.16 (13.26), range 52-143, with girls showing significantly higher mean (SD) scores than boys: 109.56 (11.96) vs 104.61 (14.09);  $P = .001$ . There was a significant interaction ( $P = .02$ ) between child sex and  $MUF_{SG}$  (6.89; 95% CI, 0.96-12.82) indicating a differential association between boys and girls. A 1-mg/L increase in  $MUF_{SG}$  was associated with a 4.49-point lower IQ score (95% CI, -8.38 to -0.60) in boys, but there was no statistically significant association with IQ scores in girls ( $B = 2.40$ ; 95% CI, -2.53 to 7.33). A 1-mg higher daily intake of fluoride among pregnant women was associated with a 3.66 lower IQ score (95% CI, -7.16 to -0.14) in boys and girls.

**CONCLUSIONS AND RELEVANCE** In this study, maternal exposure to higher levels of fluoride during pregnancy was associated with lower IQ scores in children aged 3 to 4 years. These findings indicate the possible need to reduce fluoride intake during pregnancy.

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For decades, community water fluoridation has been used to prevent tooth decay. Water fluoridation is supplied to about 66% of US residents, 38% of Canadian residents, and 3% of European residents.<sup>1</sup> In fluoridated communities, fluoride from water and beverages made with tap water makes up 60% to 80% of daily fluoride intake in adolescents and adults.<sup>2</sup>

Fluoride crosses the placenta,<sup>3</sup> and laboratory studies show that it accumulates in brain regions involved in learning and memory<sup>4</sup> and alters proteins and neurotransmitters in the central nervous system.<sup>5</sup> Higher fluoride exposure from drinking water has been associated with lower children's intelligence in a meta-analysis<sup>6</sup> of 27 epidemiologic studies and in studies<sup>7,8</sup> including biomarkers of fluoride exposure. However, most prior studies were cross-sectional and conducted in regions with higher water fluoride concentrations (0.88–31.6 mg/L; to convert to millimoles per liter, multiply by 0.05263) than levels considered optimal (ie, 0.7 mg/L) in North America.<sup>9</sup> Further, most studies did not measure exposure during fetal brain development. In a longitudinal birth cohort study involving 299 mother-child pairs in Mexico City, Mexico, a 1-mg/L increase in maternal urinary fluoride (MUF) concentration was associated with a 6-point (95% CI, –10.84 to –1.74) lower IQ score among school-aged children.<sup>10</sup> In this same cohort, MUF was also associated with more attention-deficit/hyperactivity disorder-like symptoms.<sup>11</sup> Urinary fluoride concentrations among pregnant women living in fluoridated communities in Canada are similar to concentrations among pregnant women living in Mexico City.<sup>12</sup> However, it is unclear whether fluoride exposure during pregnancy is associated with cognitive deficits in a population receiving optimally fluoridated water.

This study examined whether exposure to fluoride during pregnancy was associated with IQ scores in children in a Canadian birth cohort in which 40% of the sample was supplied with fluoridated municipal water.

## Methods

### Study Cohort

Between 2008 and 2011, the Maternal-Infant Research on Environmental Chemicals (MIREC) program recruited 2001 pregnant women from 10 cities across Canada. Women who could communicate in English or French, were older than 18 years, and were within the first 14 weeks of pregnancy were recruited from prenatal clinics. Participants were not recruited if there was a known fetal abnormality, if they had any medical complications, or if there was illicit drug use during pregnancy. Additional details are in the cohort profile description.<sup>13</sup>

A subset of 610 children in the MIREC Study was evaluated for the developmental phase of the study at ages 3 to 4 years; these children were recruited from 6 of 10 cities included in the original cohort: Vancouver, Montreal, Kingston, Toronto, Hamilton, and Halifax. Owing to budgetary restraints, recruitment was restricted to the 6 cities with the most participants who fell into the age range required for the testing during the data collection period. Of the 610 children, 601

## Key Points

**Question** Is maternal fluoride exposure during pregnancy associated with childhood IQ in a Canadian cohort receiving optimally fluoridated water?

**Findings** In this prospective birth cohort study, fluoride exposure during pregnancy was associated with lower IQ scores in children aged 3 to 4 years.

**Meaning** Fluoride exposure during pregnancy may be associated with adverse effects on child intellectual development, indicating the possible need to reduce fluoride intake during pregnancy.

(98.5%) completed neurodevelopmental testing; 254 (42.3%) of these children lived in nonfluoridated regions and 180 (30%) lived in fluoridated regions; for 167 (27.7%) fluoridation status was unknown owing to missing water data or reported not drinking tap water (Figure 1).

This study was approved by the research ethics boards at Health Canada, York University, and Indiana University. All women signed informed consent forms for both mothers and children.

### Maternal Urinary Fluoride Concentration

We used the mean concentrations of MUF measured in urine spot samples collected across each trimester of pregnancy at a mean (SD) of 11.57 (1.57), 19.11 (2.39), and 33.11 (1.50) weeks of gestation. Owing to the variability of urinary fluoride measurement and fluoride absorption during pregnancy,<sup>14</sup> we only included women who had all 3 urine samples. In our previous work, these samples were moderately correlated; intraclass correlation coefficient (ICC) ranged from 0.37 to 0.40.<sup>12</sup>

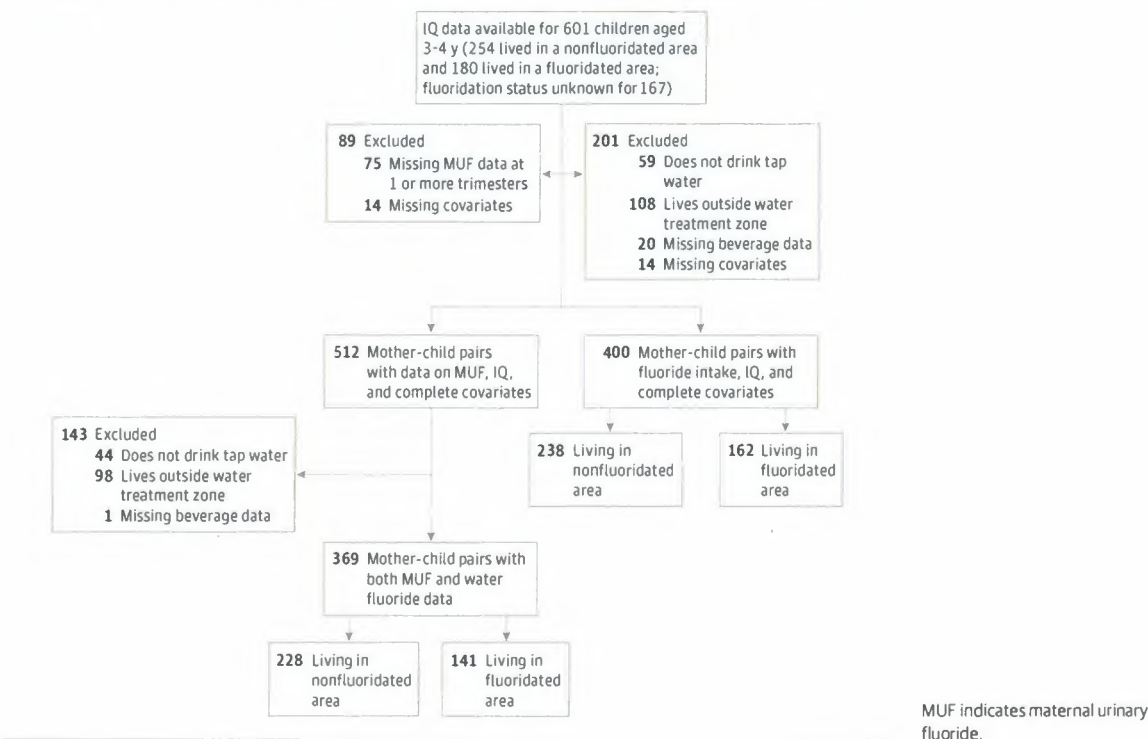
Urinary fluoride concentration was analyzed at the Indiana University School of Dentistry using a modification of the hexamethyldisiloxane (Sigma Chemical Co) microdiffusion procedure<sup>15</sup> and described in our previous work.<sup>12</sup> Fluoride concentration could be measured to 0.02 mg/L. We excluded 2 samples (0.002%) because the readings exceeded the highest concentration standard (5 mg/L) and there was less certainty of these being representative exposure values.

To account for variations in urine dilution at the time of measurement, we adjusted MUF concentrations for specific gravity (SG) using the following equation:  $MUF_{SG} = MUF_i \times (SG_M - 1) / (SG_i - 1)$ , where  $MUF_{SG}$  is the SG-adjusted fluoride concentration (in milligrams of fluoride per liter),  $MUF_i$  is the observed fluoride concentration,  $SG_i$  is the SG of the individual urine sample, and  $SG_M$  is the median SG for the cohort.<sup>16</sup> For comparison, we also adjusted MUF using the same creatinine adjustment method that was used in the 2017 Mexican cohort.<sup>10</sup>

### Water Fluoride Concentration

Water treatment plants measured fluoride levels daily if fluoride was added to municipal drinking water and weekly or monthly if fluoride was not added to water.<sup>12</sup> We matched participants' postal codes with water treatment plant zones, allowing an estimation of water fluoride concentration for each

Figure 1. Flowchart of Inclusion Criteria



woman by averaging water fluoride concentrations (in milligrams per liter) during the duration of pregnancy. We only included women who reported drinking tap water during pregnancy.

#### Daily Fluoride Intake in Mothers

We obtained information on consumption of tap water and other water-based beverages (tea and coffee) from a self-report questionnaire completed by mothers during the first and third trimesters. This questionnaire was used in the original MREC cohort and has not been validated. Also, for this study, we developed methods to estimate and calculate fluoride intake that have not yet been validated. To estimate fluoride intake from tap water consumed per day (milligrams per day), we multiplied each woman's consumption of water and beverages by her water fluoride concentration (averaged across pregnancy) and multiplied by 0.2 (fluoride content for a 200-mL cup). Because black tea contains a high fluoride content (2.6 mg/L),<sup>17,18</sup> we also estimated the amount of fluoride consumed from black tea by multiplying each cup of black tea by 0.52 mg (mean fluoride content in a 200-mL cup of black tea made with deionized water) and added this to the fluoride intake variable. Green tea also contains varying levels of fluoride; therefore, we used the mean for the green teas listed by the US Department of Agriculture (1.935 mg/L).<sup>18</sup> We multiplied each cup of green tea by 0.387 mg (fluoride content in a 200-mL cup of green tea made with deionized water) and added this to the fluoride intake variable.

#### Primary Outcomes

We assessed children's intellectual abilities with the Wechsler Preschool and Primary Scale of Intelligence, Third Edition. Full Scale IQ (FSIQ), a measure of global intellectual functioning, was the primary outcome. We also assessed verbal IQ (VIQ), representing verbal reasoning and comprehension, and performance IQ (PIQ), representing nonverbal reasoning, spatial processing, and visual-motor skills.

#### Covariates

We selected covariates from a set of established factors associated with fluoride metabolism (eg, time of void and time since last void) and children's intellectual abilities (eg, child sex, maternal age, gestational age, and parity) (Table 1). Mother's race/ethnicity was coded as white or other, and maternal education was coded as either bachelor's degree or higher or trade school diploma or lower. The quality of a child's home environment was measured by the Home Observation for Measurement of the Environment (HOME)-Revised Edition<sup>19</sup> on a continuous scale. We also controlled for city and, in some models, included self-reported exposure to secondhand smoke (yes/no) as a covariate.

#### Statistical Analyses

In our primary analysis, we used linear regression analyses to estimate the associations between our 2 measures of fluoride exposure (MUF<sub>SG</sub> and fluoride intake) and children's FSIQ scores. In addition to providing the coefficient correspond-

**Table 1. Demographic Characteristics and Exposure Outcomes for Mother-Child Pairs With MUF<sub>SG</sub> (n = 512) and Fluoride Intake Data (n = 400) by Fluoridated and Nonfluoridated Status<sup>a</sup>**

Variable <sup>b</sup>	No. (%)		
	MUF <sub>SG</sub> Sample (n = 512) <sup>c</sup>	Maternal-Child Pairs With Fluoride Intake, IQ, and Complete Covariate Data (n = 400)	
		Nonfluoridated (n = 238)	Fluoridated (n = 162)
<b>Mothers</b>			
Age of mother at enrollment, mean (SD), y	32.33 (5.07)	32.61 (4.90)	32.52 (4.03)
Prepregnancy BMI, mean (SD)	25.19 (6.02)	25.19 (6.35)	24.33 (5.10)
Married or common law	497 (97)	225 (95)	159 (98)
Born in Canada	426 (83)	187 (79)	131 (81)
White	463 (90)	209 (88)	146 (90)
<b>Maternal education</b>			
Trade school diploma/high school	162 (32)	80 (34)	38 (24)
Bachelor's degree or higher	350 (68)	158 (66)	124 (76)
Employed at time of pregnancy	452 (88)	205 (86)	149 (92)
Net income household > \$70 000 CAD	364 (71)	162 (68)	115 (71)
HOME total score, mean (SD)	47.32 (4.32)	47.28 (4.48)	48.14 (3.90)
Smoked in trimester 1	12 (2)	7 (3)	2 (1)
Secondhand smoke in the home	18 (4)	9 (4)	2 (1)
<b>Alcohol consumption, alcoholic drink/mo</b>			
None	425 (83)	192 (81)	136 (84)
<1	41 (8)	23 (10)	11 (7)
≥1	46 (9)	23 (10)	15 (9)
Parity (first birth)	233 (46)	119 (50)	71 (44)
<b>Children</b>			
Female	264 (52)	118 (50)	83 (51)
Age at testing, mean (SD), y	3.42 (0.32)	3.36 (0.31)	3.49 (0.29)
Gestation, mean (SD), wk	39.12 (1.57)	39.19 (1.47)	39.17 (1.81)
Birth weight, mean (SD), kg	3.47 (0.49)	3.48 (0.48)	3.47 (0.53)
FSIQ	107.16 (13.26)	108.07 (13.31)	108.21 (13.72)
Boys <sup>d</sup>	104.61 (14.09)	106.31 (13.60)	104.78 (14.71)
Girls <sup>d</sup>	109.56 (11.96)	109.86 (12.83)	111.47 (11.89)
<b>Exposure variables</b>			
<b>MUF<sub>SG</sub> concentration, mg/L<sup>e</sup></b>			
No.	512	228	141
Mean (SD)	0.51 (0.36)	0.40 (0.27)	0.69 (0.42)
<b>Fluoride intake level per day, mg</b>			
No.	369 <sup>a</sup>	238	162
Mean (SD)	0.54 (0.44)	0.30 (0.26)	0.93 (0.43)
<b>Water fluoride concentration, mg/L</b>			
No.	369 <sup>a</sup>	238	162
Mean (SD)	0.31 (0.23)	0.13 (0.06)	0.59 (0.08)

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); CAD, Canadian dollars; FSIQ, Full Scale IQ; HOME, Home Observation for Measurement of the Environment; MUF<sub>SG</sub>, maternal urinary fluoride adjusted for specific gravity.

SI conversion factor: To convert fluoride to millimoles per liter, multiply by 0.05263.

<sup>a</sup> Owing to missing water treatment plant data and/or MUF data, the samples are distinct with some overlapping participants in both groups (n = 369).

<sup>b</sup> All of the listed variables were tested as potential covariates, as well as the following: paternal variables (age, education, employment status, smoking status, and race/ethnicity); maternal chronic condition during pregnancy and birth country; breastfeeding duration; and time of void and time since last void.

<sup>c</sup> Maternal urinary fluoride (averaged across all 3 trimesters) and corrected for specific gravity.

<sup>d</sup> The FSIQ score has a mean (SD) of 100 (15); US population norms used.

<sup>e</sup> Owing to missing water treatment plant data, the sample in the fluoridated and nonfluoridated regions do not add up to the MUF sample size.

ing to a 1-mg difference in fluoride exposure, we also estimated coefficients corresponding to a fluoride exposure difference spanning the 25th to 75th percentile range (which corresponds to a 0.33 mg/L and 0.62 mg F/d difference in MUF<sub>SG</sub> and fluoride intake, respectively) as well as the 10th to 90th percentile range (which corresponds to a 0.70 mg/L and

1.04 mg F/d difference in MUF<sub>SG</sub> and fluoride intake, respectively).

We retained a covariate in the model if its *P* value was less than .20 or its inclusion changed the regression coefficient of the variable associated factor by more than 10% in any of the IQ models. Regression diagnostics confirmed that there were

no collinearity issues in any of the IQ models with  $MUF_{SG}$  or fluoride intake (variance inflation factor <2 for all covariates). Residuals from each model had approximately normal distributions, and their Q-Q plots revealed no extreme outliers. Plots of residuals against fitted values did not suggest any assumption violations and there were no substantial influential observations as measured by Cook distance. Including quadratic or natural-log effects of  $MUF_{SG}$  or fluoride intake did not significantly improve the regression models. Thus, we present the more easily interpreted estimates from linear regression models. Additionally, we examined separate models with 2 linear splines to test whether the  $MUF_{SG}$  association significantly differed between lower and higher levels of  $MUF_{SG}$  based on 3 knots, which were set at 0.5 mg/L (mean  $MUF_{SG}$ ), 0.8 mg/L (threshold seen in the Mexican birth cohort),<sup>10</sup> and 1 mg/L (optimal concentration in the United States until 2015).<sup>20</sup> For fluoride intake, knots were set at 0.4 mg (mean fluoride intake), 0.8 mg, and 1 mg (in accordance with  $MUF_{SG}$ ). We also examined sex-specific associations in all models by testing the interactions between child sex and each fluoride measure.

In sensitivity analyses, we tested whether the associations between  $MUF_{SG}$  and IQ were confounded by maternal blood concentrations of lead,<sup>21</sup> mercury,<sup>21</sup> manganese,<sup>21,22</sup> perfluoro-octanoic acid,<sup>23</sup> or urinary arsenic.<sup>24</sup> We also conducted sensitivity analyses by removing IQ scores that were greater than or less than 2.5 standard deviations from the sample mean. Additionally, we examined whether using  $MUF$  adjusted for creatinine instead of SG affected the results.

In additional analyses, we examined the association between our 2 measures of fluoride exposure ( $MUF_{SG}$  and fluoride intake) with VIQ and PIQ. Additionally, we examined whether water fluoride concentration was associated with FSIQ, VIQ, and PIQ scores.

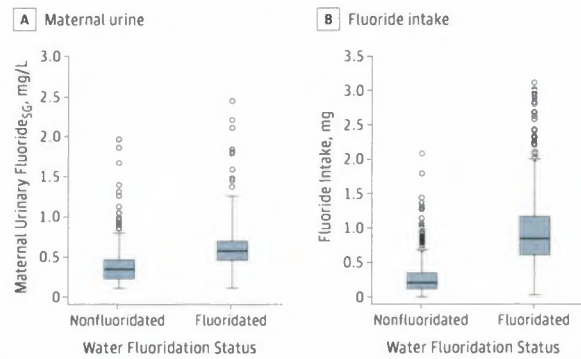
For all analyses, statistical significance tests with a type I error rate of 5% were used to test sex interactions, while 95% confidence intervals were used to estimate uncertainty. Analyses were conducted using R software (the R Foundation).<sup>25</sup> The *P* value level of significance was .05, and all tests were 2-sided.

## Results

For the first measure of fluoride exposure,  $MUF_{SG}$ , 512 of 601 mother-child pairs (85.2%) who completed the neurodevelopmental visit had urinary fluoride levels measured at each trimester of the mother's pregnancy and complete covariate data (Figure 1); 89 (14.8%) were excluded for missing  $MUF_{SG}$  at 1 or more trimesters (*n* = 75) or missing 1 or more covariates included in the regression (*n* = 14) (Figure 1). Of the 512 mother-child pairs with  $MUF_{SG}$  data (and all covariates), 264 children were female (52%).

For the second measure of fluoride exposure, fluoride intake from maternal questionnaire, data were available for 400 of the original 601 mother-child pairs (66.6%): 201 women (33.4%) were excluded for reporting not drinking tap water (*n* = 59), living outside of the predefined water treatment plant zone (*n* = 108), missing beverage consumption data (*n* = 20), or missing covariate data (*n* = 14) (Figure 1).

Figure 2. Distribution of Fluoride Levels in Maternal Urine and for Estimated Fluoride Intake by Fluoridation Status



To convert fluoride to millimoles per liter, multiply by 0.05263.

Children had mean FSIQ scores in the average range (population normed) (mean [SD], 107.16 [13.26], range = 52-143), with girls (109.56 [11.96]) showing significantly higher scores than boys (104.61 [14.09]; *P* < .001) (Table 1). The demographic characteristics of the 512 mother-child pairs included in the primary analysis were not substantially different from the original MIREC cohort or subset of mother-child pairs without 3 urine samples (eTable 1 in the Supplement). Of the 400 mother-child pairs with fluoride intake data (and all covariates), 118 of 238 (50%) in the group living in a nonfluoridated region were female and 83 of 162 (51%) in the group living in a fluoridated region were female.

### Fluoride Measurements

The median  $MUF_{SG}$  concentration was 0.41 mg/L (range, 0.06-2.44 mg/L). Mean  $MUF_{SG}$  concentration was significantly higher among women (*n* = 141) who lived in communities with fluoridated drinking water (0.69 [0.42] mg/L) compared with women (*n* = 228) who lived in communities without fluoridated drinking water (0.40 [0.27] mg/L; *P* < .001) (Table 1; Figure 2).

The median estimated fluoride intake was 0.39 mg per day (range, 0.01-2.65 mg). As expected, the mean (SD) fluoride intake was significantly higher for women (162 [40.5%]) who lived in communities with fluoridated drinking water (mean [SD], 0.93 [0.43] mg) than women (238 [59.5%]) who lived in communities without fluoridated drinking water (0.30 [0.26] mg; *P* < .001) (Table 1; Figure 2). The  $MUF_{SG}$  was moderately correlated with fluoride intake (*r* = 0.49; *P* < .001) and water fluoride concentration (*r* = 0.37; *P* < .001).

### Maternal Urinary Fluoride Concentrations and IQ

Before covariate adjustment, a significant interaction (*P* for interaction = .03) between  $MUF_{SG}$  and child sex (*B* = 7.24; 95% CI, 0.81-13.67) indicated that  $MUF_{SG}$  was associated with FSIQ in boys; an increase of 1 mg/L  $MUF_{SG}$  was associated with a 5.01 (95% CI, -9.06 to -0.97, *P* = .02) lower FSIQ score in boys. In contrast,  $MUF_{SG}$  was not significantly associated with FSIQ score in girls (*B* = 2.23; 95% CI, -2.77 to 7.23; *P* = .38) (Table 2).

Table 2. Unadjusted and Adjusted Associations Estimated From Linear Regression Models of Fluoride Exposure Variables and FSIQ Scores

Variable	Difference (95% CI)		Adjusted Estimates, Regression Coefficients Indicate Change in Outcome per <sup>a</sup>		
	Unadjusted		1 mg	25th to 75th Percentiles	10th to 90th Percentiles
MUF <sub>SG</sub> <sup>b,c</sup>	-2.60 (-5.80 to 0.60)		-1.95 (-5.19 to 1.28)	-0.64 (-1.69 to 0.42)	-1.36 (-3.58 to 0.90)
Boys	-5.01 (-9.06 to -0.97)		-4.49 (-8.38 to -0.60)	-1.48 (-2.76 to -0.19)	-3.14 (-5.86 to -0.42)
Girls	2.23 (-2.77 to 7.23)		2.40 (-2.53 to 7.33)	0.79 (-0.83 to 2.42)	1.68 (-1.77 to 5.13)
Fluoride intake <sup>d,e</sup>	-3.19 (-5.94 to -0.44)		-3.66 (-7.16 to -0.15)	-2.26 (-4.45 to -0.09)	-3.80 (-7.46 to -0.16)

Abbreviations: FSIQ, Full Scale IQ; HOME, Home Observation for Measurement of the Environment; MUF<sub>SG</sub>, maternal urinary fluoride adjusted for specific gravity.

<sup>a</sup> Adjusted estimates pertain to predicted FSIQ difference for a value spanning the interquartile range (25th to 75th percentiles) and 80th central range (10th to 90th percentiles): (1) MUF<sub>SG</sub>: 0.33 mg/L, 0.70 mg/L, respectively; (2) fluoride intake: 0.62 mg, 1.04 mg, respectively.

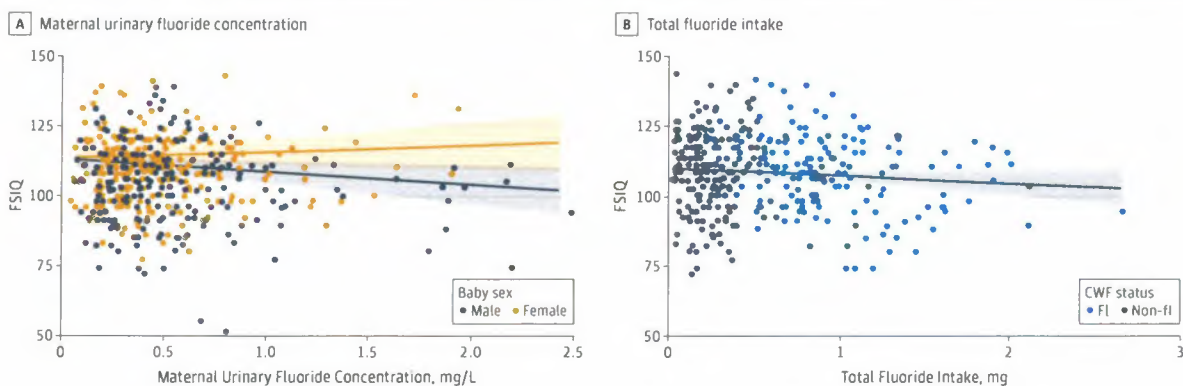
<sup>b</sup> n = 512.

<sup>c</sup> Adjusted for city, HOME score, maternal education, race/ethnicity, and including child sex interaction.

<sup>d</sup> n = 400.

<sup>e</sup> Adjusted for city, HOME score, maternal education, race/ethnicity, child sex, and prenatal secondhand smoke exposure.

Figure 3. Covariate Results of Multiple Linear Regression Models of Full Scale IQ (FSIQ) from Maternal Urinary Fluoride Concentration by Child Sex (n = 512) and Total Fluoride Intake Estimated from Daily Maternal Beverage Consumption (n = 400)



B, Community fluoridation status (CWF) is shown for each woman; black dots represent women living in nonfluoridated (non-FI) communities and blue dots represent women living in fluoridated (FI) communities.

Adjusting for covariates, a significant interaction ( $P$  for interaction = .02) between child sex and MUF<sub>SG</sub> ( $B = 6.89$ ; 95% CI, 0.96-12.82) indicated that an increase of 1 mg/L of MUF<sub>SG</sub> was associated with a 4.49 (95% CI, -8.38 to -0.60;  $P = .02$ ) lower FSIQ score for boys. An increase from the 10th to 90th percentile of MUF<sub>SG</sub> was associated with a 3.14 IQ decrement among boys (Table 2; Figure 3). In contrast, MUF<sub>SG</sub> was not significantly associated with FSIQ score in girls ( $B = 2.43$ ; 95% CI, -2.51 to 7.36;  $P = .33$ ).

#### Estimated Fluoride Intake and IQ

A 1-mg increase in fluoride intake was associated with a 3.66 (95% CI, -7.16 to -0.15;  $P = .04$ ) lower FSIQ score among boys and girls (Table 2; Figure 3). The interaction between child sex and fluoride intake was not statistically significant ( $B = 1.17$ ; 95% CI, -4.08 to 6.41;  $P$  for interaction = .66).

#### Sensitivity Analyses

Adjusting for lead, mercury, manganese, perfluorooctanoic acid, or arsenic concentrations did not substantially change the overall estimates of MUF<sub>SG</sub> for boys or girls (eTable 2 in the Supplement). Use of MUF adjusted for creatinine did not sub-

stantially alter the associations with FSIQ (eTable 2 in the Supplement). Including time of void and time since last void did not substantially change the regression coefficient of MUF<sub>SG</sub> among boys or girls.

Estimates for determining the association between MUF<sub>SG</sub> and PIQ showed a similar pattern with a statistically significant interaction between MUF<sub>SG</sub> and child sex ( $P$  for interaction = .007). An increase of 1 mg/L MUF<sub>SG</sub> was associated with a 4.63 (95% CI, -9.01 to -0.25;  $P = .04$ ) lower PIQ score in boys, but the association was not statistically significant in girls ( $B = 4.51$ ; 95% CI, -1.02 to 10.05;  $P = .11$ ). An increase of 1 mg/L MUF<sub>SG</sub> was not significantly associated with VIQ in boys ( $B = -2.85$ ; 95% CI, -6.65 to 0.95;  $P = .14$ ) or girls ( $B = 0.55$ ; 95% CI, -4.28 to 5.37;  $P = .82$ ); the interaction between MUF<sub>SG</sub> and child sex was not statistically significant ( $P$  for interaction = .25) (eTable 3 in the Supplement).

Consistent with the findings on estimated maternal fluoride intake, increased water fluoride concentration (per 1 mg/L) was associated with a 5.29 (95% CI, -10.39 to -0.19) lower FSIQ score among boys and girls and a 13.79 (95% CI, -18.82 to -7.28) lower PIQ score (eTable 4 in the Supplement).

## Discussion

Using a prospective Canadian birth cohort, we found that estimated maternal exposure to higher fluoride levels during pregnancy was associated with lower IQ scores in children. This association was supported by converging findings from 2 measures of fluoride exposure during pregnancy. A difference in MUF<sub>SG</sub> spanning the interquartile range for the entire sample (ie, 0.33 mg/L), which is roughly the difference in MUF<sub>SG</sub> concentration for pregnant women living in a fluoridated vs a non-fluoridated community, was associated with a 1.5-point IQ decrement among boys. An increment of 0.70 mg/L in MUF<sub>SG</sub> concentration was associated with a 3-point IQ decrement in boys; about half of the women living in a fluoridated community have a MUF<sub>SG</sub> equal to or greater than 0.70 mg/L. These results did not change appreciably after controlling for other key exposures such as lead, arsenic, and mercury.

To our knowledge, this study is the first to estimate fluoride exposure in a large birth cohort receiving optimally fluoridated water. These findings are consistent with that of a Mexican birth cohort study that reported a 6.3 decrement in IQ in preschool-aged children compared with a 4.5 decrement for boys in our study for every 1 mg/L of MUF.<sup>10</sup> The findings of the current study are also concordant with ecologic studies that have shown an association between higher levels of fluoride exposure and lower intellectual abilities in children.<sup>7,8,26</sup> Collectively, these findings support that fluoride exposure during pregnancy may be associated with neurocognitive deficits.

In contrast with the Mexican study,<sup>10</sup> the association between higher MUF<sub>SG</sub> concentrations and lower IQ scores was observed only in boys but not in girls. Studies of fetal and early childhood fluoride exposure and IQ have rarely examined differences by sex; of those that did, some reported no differences by sex.<sup>10,27-29</sup> Most rat studies have focused on fluoride exposure in male rats,<sup>30</sup> although 1 study<sup>31</sup> showed that male rats were more sensitive to neurocognitive effects of fetal exposure to fluoride. Testing whether boys are potentially more vulnerable to neurocognitive effects associated with fluoride exposure requires further investigation, especially considering that boys have a higher prevalence of neurodevelopmental disorders such as ADHD, learning disabilities, and intellectual disabilities.<sup>32</sup> Adverse effects of early exposure to fluoride may manifest differently for girls and boys, as shown with other neurotoxicants.<sup>33-36</sup>

The estimate of maternal fluoride intake during pregnancy in this study showed that an increase of 1 mg of fluoride was associated with a decrease of 3.7 IQ points across boys and girls. The finding observed for fluoride intake in both boys and girls may reflect postnatal exposure to fluoride, whereas MUF primarily captures prenatal exposure. Importantly, we excluded women who reported that they did not drink tap water and matched water fluoride measurements to time of pregnancy when estimating maternal fluoride intake. None of the fluoride concentrations measured in municipal drinking water were greater than the maximum acceptable concentra-

tion of 1.5 mg/L set by Health Canada; most (94.3%) were lower than the 0.7 mg/L level considered optimal.<sup>37</sup>

Water fluoridation was introduced in the 1950s to prevent dental caries before the widespread use of fluoridated dental products. Originally, the US Public Health Service set the optimal fluoride concentrations in water from 0.7 to 1.2 mg/L to achieve the maximum reduction in tooth decay and minimize the risk of enamel fluorosis.<sup>38</sup> Fluorosis, or mottling, is a symptom of excess fluoride intake from any source occurring during the period of tooth development. In 2012, 68% of adolescents had very mild to severe enamel fluorosis.<sup>39</sup> The higher prevalence of enamel fluorosis, especially in fluoridated areas,<sup>40</sup> triggered renewed concern about excessive ingestion of fluoride. In 2015, in response to fluoride overexposure and rising rates of enamel fluorosis,<sup>39,41,42</sup> the US Public Health Service recommended an optimal fluoride concentration of 0.7 mg/L, in line with the recommended level of fluoride added to drinking water in Canada to prevent caries. However, the beneficial effects of fluoride predominantly occur at the tooth surface after the teeth have erupted.<sup>43</sup> Therefore, there is no benefit of systemic exposure to fluoride during pregnancy for the prevention of caries in offspring.<sup>44</sup> The evidence showing an association between fluoride exposure and lower IQ scores raises a possible new concern about cumulative exposures to fluoride during pregnancy, even among pregnant women exposed to optimally fluoridated water.

## Strengths and Limitations

Our study has several strengths and limitations. First, urinary fluoride has a short half-life (approximately 5 hours) and depends on behaviors that were not controlled in our study, such as consumption of fluoride-free bottled water or swallowing toothpaste prior to urine sampling. We minimized this limitation by using 3 serial urine samples and tested for time of urine sample collection and time since last void, but these variables did not alter our results. Second, although higher maternal ingestion of fluoride corresponds to higher fetal plasma fluoride levels,<sup>45</sup> even serial maternal urinary spot samples may not precisely represent fetal exposure throughout pregnancy. Third, while our analyses controlled for a comprehensive set of covariates, we did not have maternal IQ data. However, there is no evidence suggesting that fluoride exposure differs as a function of maternal IQ; our prior study did not observe a significant association between MUF levels and maternal education level.<sup>12</sup> Moreover, a greater proportion of women living in fluoridated communities (124 [76%]) had a university-level degree compared with women living in non-fluoridated communities (158 [66%]). Nonetheless, despite our comprehensive array of covariates included, this observational study design could not address the possibility of other unmeasured residual confounding. Fourth, fluoride intake did not measure actual fluoride concentration in tap water in the participant's home; Toronto, for example, has overlapping water treatment plants servicing the same household. Similarly, our fluoride intake estimate only considered fluoride from beverages; it did not include fluoride from other sources such as dental products or food. Furthermore, fluoride intake data were limited by self-report of mothers' recall of beverage consump-

tion per day, which was sampled at 2 points of pregnancy, and we lacked information regarding specific tea brand.<sup>17,18</sup> In addition, our methods of estimating maternal fluoride intake have not been validated; however, we show construct validity with MUF. Fifth, this study did not include assessment of postnatal fluoride exposure or consumption. However, our future analyses will assess exposure to fluoride in the MIREC cohort in infancy and early childhood.

sociated with lower IQ scores in children measured at age 3 to 4 years. These findings were observed at fluoride levels typically found in white North American women. This indicates the possible need to reduce fluoride intake during pregnancy.

## Conclusions

In this prospective birth cohort study from 6 cities in Canada, higher levels of fluoride exposure during pregnancy were as-

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**Administrative, technical, or material support:** Green, Lanphear, Martinez-Mier, Ayotte, Till.

**Supervision:** Flora, Till.

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## Article

# Iodine Status Modifies the Association between Fluoride Exposure in Pregnancy and Preschool Boys' Intelligence

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**Abstract:** In animal studies, the combination of in utero fluoride exposure and low iodine has greater negative effects on offspring learning and memory than either alone, but this has not been studied in children. We evaluated whether the maternal urinary iodine concentration (MUIC) modifies the association between maternal urinary fluoride (MUF) and boys' and girls' intelligence. We used data from 366 mother–child dyads in the Maternal–Infant Research on Environmental Chemicals Study. We corrected trimester-specific MUF and MUIC for creatinine, and averaged them to yield our exposure variables (MUF<sub>CRE</sub>, mg/g; MUIC<sub>CRE</sub>, µg/g). We assessed children's full-scale intelligence (FSIQ) at 3 to 4 years. Using multiple linear regression, we estimated a three-way interaction between MUF<sub>CRE</sub>, MUIC<sub>CRE</sub>, and child sex on FSIQ, controlling for covariates. The MUIC<sub>CRE</sub> by MUF<sub>CRE</sub> interaction was significant for boys ( $p = 0.042$ ), but not girls ( $p = 0.190$ ). For boys whose mothers had low iodine, a 0.5 mg/g increase in MUF<sub>CRE</sub> was associated with a 4.65-point lower FSIQ score (95% CI:  $-7.67, -1.62$ ). For boys whose mothers had adequate iodine, a 0.5 mg/g increase in MUF<sub>CRE</sub> was associated with a 2.95-point lower FSIQ score (95% CI:  $-4.77, -1.13$ ). These results suggest adequate iodine intake during pregnancy may minimize fluoride's neurotoxicity in boys.

**Keywords:** fluoride; iodine; intelligence; neurodevelopment; pregnancy

## 1. Introduction

Fluoride exposure during early brain development has been associated with diminished intelligence quotient (IQ) scores among children living in areas with high levels of naturally occurring fluoride in drinking water (~3 mg/L) [1–3] and in areas where fluoride is added to public water supplies or salt for caries prevention [4–6]. The mechanism(s) underlying fluoride-associated cognitive deficits are not well understood, but changes to the thyroid function may be one such mechanism [7–11]. In 2006, the National Research Council (NRC) classified fluoride as an endocrine disruptor and recommended more research to understand fluoride's effects on the thyroid gland, especially in iodine deficient pregnant women [12].

Iodine is an essential nutrient for thyroid hormone synthesis and normal thyroid function [13]. Sufficient iodine intake is critical for optimal maternal and fetal thyroid function and normal fetal neurodevelopment [14–17]. Even mild to moderate iodine deficiency in pregnancy has been linked to diminished cognitive abilities in children [14,18–24], though not in all studies [25,26]. The inconsistent results may reflect differences in the severity of maternal iodine deficiency, methodology, age at outcome assessment, or other biological co-factors.

Studies conducted in China examined whether fluoride exposure and iodine deficiency combine to impart adverse effects on children's intelligence. Notably, school-aged children living in endemic fluoride and iodine-deficient areas had lower IQ scores than those living in endemic fluoride areas alone or iodine-deficient areas alone [27,28]. Fluoride in drinking water was reported to exacerbate the adverse effects of low iodine on child neurodevelopment and central nervous system function more broadly [28]. However, these studies were cross-sectional and did not account for potential confounders. In experimental studies, rat offspring exposed to both high fluoride and low iodine in utero showed greater deficits in learning and memory compared with those exposed to either high fluoride or low iodine [29,30].

Given the ubiquity of fluoride exposure, along with recent trends showing mild-to-moderate iodine deficiency in pregnant women [17,31,32], we evaluated whether the maternal iodine status modifies the association between prenatal fluoride exposure and children's intelligence. We hypothesized that low urinary iodine concentrations in Canadian pregnant women would exacerbate the fluoride-associated intellectual deficits observed in their children. We further hypothesized that the effects would be stronger in boys than girls given previous findings of sex differences in the neurotoxicity of prenatal fluoride exposure [33].

## 2. Materials and Methods

### 2.1. Participants

Participants included mother–child dyads enrolled in the Canadian Maternal–Infant Research on Environmental Chemicals (MIREC) study. Between 2008 and 2011, 2001 pregnant women were recruited from 10 cities across Canada to participate in a longitudinal cohort study. The inclusion criteria were as follows: women who were 18 years of age or older who could provide consent, communicate in English or French, and were <14 weeks' gestation. Participants were excluded if they had any medical complications, any known fetal abnormalities, or if there was illicit drug or alcohol abuse during pregnancy. Additional details of the MIREC study can be found in the cohort profile [34].

A subset of 808 women provided consent to participate with their child in the MIREC-Child Development Plus (CD Plus) follow-up study. Due to budgetary constraints, recruitment for MIREC CD Plus was limited to six of the ten cities from the original cohort, namely Vancouver, Toronto, Hamilton, Montreal, Kingston, and Halifax. The inclusion criteria for mother–child dyads in MIREC-CD Plus were as follows: mothers of singleton children born >28 weeks' gestation who were between the ages of 3 and 4 at time of the study and had no congenital abnormalities, major neurological disorders, or history of convulsions. Among the 808 women who consented, 610 agreed to child IQ testing (76%), 601 of whom completed the neurodevelopmental testing. The latter subset of 601 mother–child dyads provided data for the current study.

Of the 601 children who completed IQ testing, 366 had complete data on maternal urinary fluoride (MUF), maternal urinary iodine concentration (MUI), urinary creatinine (CRE), and covariates (See Figure 1); 235 were excluded for missing (i) creatinine data at all three trimesters ( $n = 175$ ), (ii) a valid MUF measure available at all three trimesters ( $n = 9$ ), (iii)  $MUI_{CRE} < 600 \mu\text{g/g}$  data at trimesters 1 and 2 ( $n = 40$ ), and (4) covariate data ( $n = 11$ ). Women with  $MUI_{CRE}$  values greater than or equal to  $600 \mu\text{g/g}$  ( $n = 37$ ) were excluded from the analyses because excess iodine levels have been linked to diminished intelligence [35], and we were specifically interested in comparing women with “low”

levels of iodine with those with “adequate” levels of iodine, rather than “excess” levels of iodine. We considered  $MUIC_{CRE}$  values greater than or equal to  $600 \mu\text{g/g}$  to be “higher than adequate” as opposed to the WHO cut-off of  $500 \mu\text{g/L}$  for unadjusted MUIC [36], given that we used MUIC values corrected for creatinine, and  $MUIC_{CRE}$  values increase from trimester 1 to 2 [37].

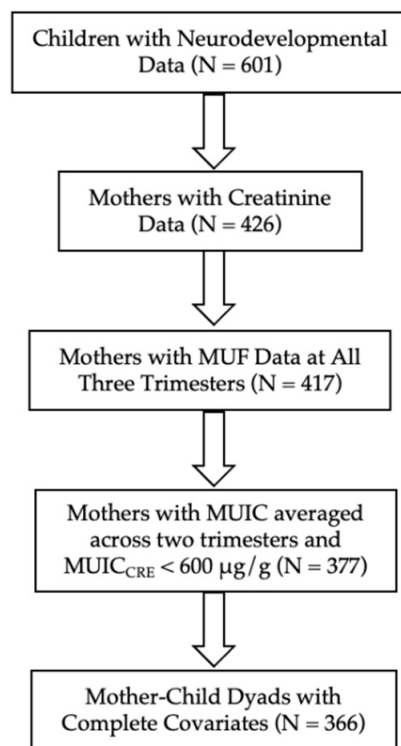


Figure 1. Sample flow chart.

The present study was approved by the research ethics boards at Health Canada and York University. The MIREC study was also approved by the research ethics boards at all participating recruitment sites and at Health Canada. All participants provided their informed consent.

## 2.2. Urine Collection

Urine was collected in Nalgene<sup>®</sup> containers, labeled with a unique identification, aliquoted into smaller Cryovials<sup>®</sup>, and stored at appropriate temperatures until they were shipped for fluoride or iodine analysis. Spot samples were collected in each trimester of pregnancy at a mean  $\pm$  SD of  $11.57 \pm 1.57$ ,  $19.11 \pm 2.39$ , and  $33.11 \pm 1.50$  weeks' gestation.

## 2.3. Maternal Urinary Fluoride Concentration

We derived maternal urinary fluoride (MUF, mg/L) concentrations by averaging fluoride concentrations across trimesters. We previously found a moderate correlation between the three samples, with intraclass correlation coefficients ranging from 0.37 to 0.40 [38]. Urine samples were analyzed at the Indiana University School of Dentistry through a modification of the hexamethyldisiloxane (HMDS; Sigma Chemical Co., St. Louis, MO, USA) micro-diffusion procedure described previously [38,39]. In neutral solutions, fluoride concentrations were measured down to  $0.02 \text{ mg/L}$ . Two of the spot urine samples (0.04%) were excluded from the first trimester as the readings surpassed the highest concentration standard of the instrument ( $5 \text{ mg/L}$ ).

#### 2.4. Maternal Urinary Iodine Concentration

We derived the maternal urinary iodine concentration (MUIC,  $\mu\text{g}/\text{L}$ ) by averaging iodine concentrations from two spot urine samples collected in the first and second trimester. MUIC is considered a reliable biomarker of recent iodine intake and reflects total iodine intake from all dietary sources [40]. MUIC was measured by the accredited Toxicology Laboratory at the Institut National de Santé Publique du Québec (INSPQ) using inductively coupled plasma mass spectrometry (ICP-MS). Values below the limit of detection (LOD,  $38 \mu\text{g}/\text{L}$ ) were replaced with the LOD divided by the square root of 2 (Hornung and Reed, 1990); 180 (9.74%) and 79 (4.56%) mothers had a MUIC below the LOD in trimesters 1 and 2, respectively.

#### 2.5. Correcting for Variability in Urinary Dilution

To account for variability in urine dilution at time of measurement, MUF and MUIC were corrected for creatinine (CRE) measured in the same spot sample using the following equations:

$$\text{MUF}_{\text{CRE}} (\text{mg}/\text{g}) = \frac{(\text{MUF}_{\text{T1}}/\text{CRE}_{\text{T1}}) + (\text{MUF}_{\text{T2}}/\text{CRE}_{\text{T2}}) + (\text{MUF}_{\text{T3}}/\text{CRE}_{\text{T3}})}{3},$$

$$\text{MUIC}_{\text{CRE}} (\mu\text{g}/\text{g}) = \frac{(\text{MUIC}_{\text{T1}}/\text{CRE}_{\text{T1}}) + (\text{MUIC}_{\text{T2}}/\text{CRE}_{\text{T2}})}{2},$$

where  $\text{MUF}_{\text{T1}}$  is the observed fluoride concentration,  $\text{MUIC}_{\text{T1}}$  is the observed MUIC, and  $\text{CRE}_{\text{T1}}$  is the observed creatinine concentration for that individual in trimester 1.  $\text{MUF}_{\text{T2}}$  is the observed fluoride concentration,  $\text{MUIC}_{\text{T2}}$  is the observed MUIC, and  $\text{CRE}_{\text{T2}}$  is the observed creatinine concentration for that individual in trimester 2.  $\text{MUF}_{\text{T3}}$  is the observed fluoride concentration, and  $\text{CRE}_{\text{T3}}$  is the observed creatinine concentration for that individual in trimester 3. The measurement of urinary creatinine was previously described [38]. In pregnant women, the iodine to creatinine ratio (MUIC/CRE) is moderately correlated with 24 h urinary iodine excretion, the gold standard measure of iodine status relative to uncorrected urinary iodine concentration [14,41].

#### 2.6. Children's Full-Scale Intelligence Quotient

We assessed children's intellectual abilities at 3 to 4 years of age using the Wechsler Preschool and Primary Scale of Intelligence-III with Canadian age-standardized norms (mean = 100, SD = 15). The test was administered in children's homes in either English or French by qualified research professionals who were blinded to gestational iodine status or fluoride exposure in pregnancy. We used full-scale intelligence (FSIQ), a measure of global intellectual and cognitive functioning, as our primary outcome.

#### 2.7. Covariates

We selected covariates a priori based on prior work with the same study cohort examining fluoride exposure and children's intellectual abilities [6]. Covariates included maternal education (dichotomized as bachelor's degree or higher), maternal race (White/non-White), study site, and a continuous measure of the quality of the home environment using the Home Observation for Measurement of the Environment (HOME)—Revised Edition [42] at the time of the home visit when children were aged 3 to 4 years old.

#### 2.8. Statistical Analyses

We used chi-square tests for categorical covariates and t-tests for continuous covariates to test for sampling differences between those with complete data and those without complete data (i.e., without  $\text{MUF}_{\text{CRE}}$  or  $\text{MUIC}_{\text{CRE}}$  but with FSIQ data). For descriptive purposes,  $\text{MUIC}_{\text{CRE}}$  was stratified into those with low ( $<200 \mu\text{g}/\text{g}$ ) and adequate  $\geq 200$  and  $<600 \mu\text{g}/\text{g}$  urinary iodine. Independent sample t-tests were used to test for differences

between boys and girls for exposure and outcome variables. Welch's correction was applied for t-tests to account for unequal variance.

We used multiple linear regression to estimate a model with a three-way interaction between  $MUF_{CRE}$ ,  $MUIC_{CRE}$ , and child sex in predicting children's FSIQ scores while controlling for maternal education, maternal race, study site, and the HOME score; this model included all constituent two-way interaction terms and first-order effects. To determine whether  $MUF_{CRE}$  and  $MUIC_{CRE}$  interact as a function of sex without stratifying the sample, we then examined the model-implied  $MUF_{CRE}$  by  $MUIC_{CRE}$  two-way interaction within each sex. To facilitate the interpretation of coefficients, we centered  $MUIC_{CRE}$  (i.e., subtracted a constant from every value of  $MUIC_{CRE}$ ) around a "low" level (i.e., 147  $\mu\text{g/g}$  which corresponds to the 10th percentile value for  $MUIC_{CRE}$ ) and an "adequate" level (i.e., 294  $\mu\text{g/g}$  which corresponds to the 50th percentile value for  $MUIC_{CRE}$ ) [43,44]. We then re-ran the model using  $MUIC_{CRE}$  centered around the "low" and "adequate" levels of iodine, separately, with boys coded as the reference. The model was re-estimated with girls coded as the reference to interpret the association between  $MUF_{CRE}$  and FSIQ for a girl whose mother had a low or adequate level of iodine during pregnancy. All models were evaluated for linearity, homoscedasticity, and normality and model assumptions were sufficiently met. No influential outliers were detected according to Cook's distance.

We used STATA version 16.1 (STATA corporation) for data analysis. The level of significance was 0.05, and all statistical tests were two-tailed. All coefficients were reported for every 0.5 mg/g increase in  $MUF_{CRE}$  (approximately the IQR).

### 3. Results

Most participants included in the present study were married or in a common-law relationship, had a bachelor's degree or higher, and were White (Table 1). Mother-child dyads with complete data did not significantly differ from those without complete data on any of the demographic characteristics, except a greater proportion of mothers with complete data were White.

**Table 1.** Demographic Characteristics of those with Complete Data ( $N = 366$ ) and Incomplete Data ( $N = 211$ ).

Demographic Characteristic (Mean $\pm$ SD or N (%))	Complete Data ( $N = 366$ )	Incomplete Data ( $N = 211$ )	<i>p</i>
Mothers			
Maternal Age (years)	32.50 $\pm$ 4.51	32.55 $\pm$ 4.62	0.899
Married or Common Law	353 (96.54)	205 (97.16)	0.646
White	334 (91.26)	181 (85.78)	0.041
Bachelor's Degree or Higher	243 (66.39)	142 (67.30)	0.824
Taking a prenatal multivitamin	319 (87.40)	175 (82.94)	0.140
HOME Score	47.23 $\pm$ 4.44	47.40 $\pm$ 4.10	0.649
Children			
Male	186 (50.82)	98 (46.44)	0.311
Age at Testing (years)	3.44 $\pm$ 0.32	3.40 $\pm$ 0.31	0.144

Abbreviations: HOME = Home Observation Measurement of the environment.

The median (IQR)  $MUF_{CRE}$  and  $MUIC_{CRE}$  were 0.61 (0.49) mg/g and 294 (181)  $\mu\text{g/g}$ , respectively. Boys and girls did not differ significantly in  $MUF_{CRE}$  concentration or  $MUIC_{CRE}$  (Table 2). Children's FSIQ scores were in the average range, with girls scoring significantly higher than boys ( $t(364) = -3.17$ ,  $p = 0.002$ ; Table 2).

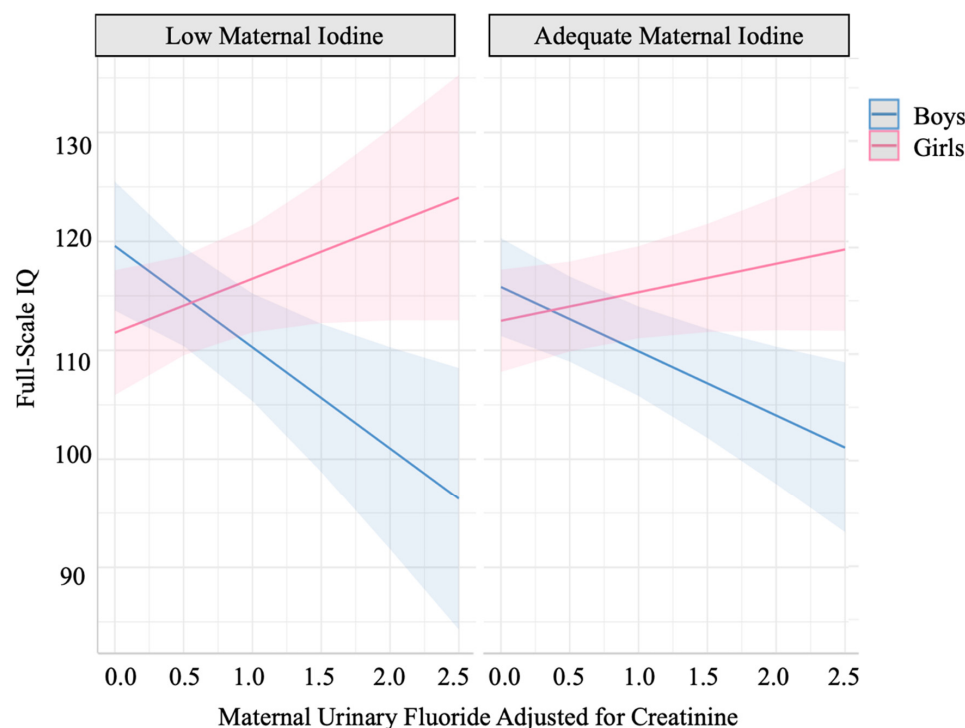
**Table 2.** MUF<sub>CRE</sub>, MUIC<sub>CRE</sub>, and Full-Scale IQ by sex.

Urinary Measurement	All		Boys		Girls		<i>p</i> <sup>1</sup>
	<i>n</i>	Median (IQR)	<i>n</i>	Median (IQR)	<i>n</i>	Median (IQR)	
MUF <sub>CRE</sub> (mg/g)	366	0.61 (0.49)	186	0.63 (0.52)	180	0.61 (0.48)	0.538
MUIC <sub>CRE</sub> (µg/g)	366	294 (181)	186	309 (181)	180	287 (203)	0.059
Low	86	148 (47)	31	131 (73)	55	152 (37)	0.083
Adequate	280	341 (165)	155	348 (187)	125	336 (146)	0.893
Outcome	<i>n</i>	Mean ± SD	<i>n</i>	Mean ± SD	<i>n</i>	Mean ± SD	<i>p</i> <sup>1</sup>
FSIQ	366	107.46 ± 13.75	186	105.25 ± 14.90	180	109.75 ± 12.09	0.002

Low MUIC<sub>CRE</sub> < 200 µg/g, Adequate MUIC<sub>CRE</sub> ≥ 200 & < 600 µg/g; Abbreviations: MUF<sub>CRE</sub> = Maternal urinary fluoride corrected for creatinine; MUIC<sub>CRE</sub> = maternal urinary iodine concentration corrected for creatinine; FSIQ = Full-Scale IQ. <sup>1</sup> Comparing boys with girls.

*Three-Way Interaction Model*

We found a significant three-way interaction between MUF<sub>CRE</sub>, MUIC<sub>CRE</sub>, and sex while controlling for relevant covariates (*p* = 0.019; see Table 3 and Figure 2). The two-way MUIC<sub>CRE</sub> by MUF<sub>CRE</sub> interaction was significant for boys (*p* = 0.042), but not girls (*p* = 0.190). For boys whose mothers had a low MUIC<sub>CRE</sub>, every 0.5 mg/g increase in MUF<sub>CRE</sub> was associated with a 4.65-point lower FSIQ score (95% CI: −7.67, −1.62; *p* = 0.003). For boys whose mothers had adequate MUIC<sub>CRE</sub>, every 0.5 mg/g increase in MUF<sub>CRE</sub> was associated with a 2.95-point lower FSIQ score (95% CI: −4.77, −1.13; *p* = 0.002). In contrast, MUF<sub>CRE</sub> was marginally associated with FSIQ for girls whose mothers had low MUIC<sub>CRE</sub> (B = 2.48; 95% CI: −0.31, 5.26; *p* = 0.081) and was not significantly associated with FSIQ for girls whose mothers had adequate MUIC<sub>CRE</sub> (B = 1.31, 95% CI: −0.41, 3.03; *p* = 0.135).



**Figure 2.** Model—implied three-way interaction between maternal urinary fluoride (MUF<sub>CRE</sub>), maternal urinary iodine concentration (MUIC<sub>CRE</sub>) and child sex. Every 0.5 mg/g increase in MUF<sub>CRE</sub> was significantly associated with a 4.65- and −2.95-point lower FSIQ score for boys whose mothers had low MUIC<sub>CRE</sub> or adequate MUIC<sub>CRE</sub>, respectively. MUF<sub>CRE</sub> was marginally associated with FSIQ for girls whose mothers had low MUIC<sub>CRE</sub> and not significantly associated with FSIQ for girls whose mothers had adequate MUIC<sub>CRE</sub>.

**Table 3.** Results of the three-way interaction model.

Variable	B	SE(B)	p
MUF <sub>CRE</sub> (mg/g)	−5.89	1.85	0.002
MUIC <sub>CRE</sub> (μg/g)	−0.03	0.01	0.023
Sex	−3.09	2.17	0.155
MUF <sub>CRE</sub> × MUIC <sub>CRE</sub>	0.02	0.01	0.042
MUF <sub>CRE</sub> × Sex	8.51	2.40	<0.001
MUIC <sub>CRE</sub> × Sex	0.03	0.02	0.042
MUF <sub>CRE</sub> × MUIC <sub>CRE</sub> × Sex	−0.04	0.02	0.019

Note. SE: Standard Error,  $R^2 = 0.28$ ,  $F(15, 350) = 8.97$ ,  $p < 0.001$ ; Abbreviations: MUF<sub>CRE</sub> = maternal urinary fluoride corrected for creatinine; MUIC<sub>CRE</sub> = maternal urinary iodine concentration corrected for creatinine. Model adjusted for maternal level of education, maternal ethnicity, HOME score, and study site. MUIC<sub>CRE</sub> is centered around the “adequate” level of iodine, and boys are coded as the reference. The coefficient for MUF<sub>CRE</sub> represents the association between MUF<sub>CRE</sub> and FSIQ for a boy whose mother had an adequate level of MUIC<sub>CRE</sub> during pregnancy.

#### 4. Discussion

We examined whether gestational iodine status modifies the association between prenatal fluoride exposure and preschool boys’ and girls’ intelligence in the Maternal Infant Research on Environmental Chemicals (MIREC) Study. To do so, we estimated the three-way interaction between prenatal fluoride exposure, gestational iodine status, and sex on children’s FSIQ. We found that the association between prenatal fluoride exposure and FSIQ was stronger among boys whose mothers had low urinary iodine concentrations in pregnancy compared to boys whose mothers had adequate iodine concentrations in pregnancy. These findings are consistent with previous experimental and human epidemiological studies [27–30,45] and indicate that even mildly reduced iodine levels may have biological significance when interacting with fluoride. Importantly, our findings were observed in a Canadian pregnancy sample with, on average, sufficient iodine intake (median iodine = 294 μg/g) and with 88% of women taking prenatal multi-vitamins.

Regarding potential mechanisms, experimental evidence demonstrates that prenatal exposure to both high fluoride and low iodine can induce neurochemical changes in offspring. For example, Ge et al., (2011) found that brains of rat offspring exposed to both high fluoride and low iodine in utero had different protein profiles compared with controls; proteins involved in cellular signaling and metabolism were most affected [46]. Other studies with similar experimental designs found higher levels of superoxide dismutase and malondialdehyde (biomarkers of oxidative stress), apoptosis, and histopathological changes (e.g., elongation of neural dendrites and missing nuclei) in the brains of rat offspring exposed to high fluoride and low iodine compared with those exposed to either alone [29,47,48].

The combination of low iodine and high fluoride may also adversely impact thyroid function. A prior study conducted in Canada showed that higher urinary fluoride levels in adults were associated with higher thyroid stimulating hormone levels, but only among adults who had low urinary iodine concentrations ( $\leq 0.38$  μmol/L) [49]. One potential mechanism by which fluoride may interact with iodine to affect thyroid function is by inhibiting one or more enzymes involved in normal thyroid function, such as deiodinases [50]. This would increase the iodine requirement, such that the effect would be more severe in the presence of iodine deficiency. Another common hypothesis is that fluoride displaces thyroidal iodine uptake since it is more electronegative and has a lighter atomic weight than iodine [51].

Experimental studies have also shown that the effects of fluoride may be exacerbated by deficient or excess iodine [52]. For instance, Guan et al. (1988) observed decreases in T3 and T4 among adult Wistar rats with sufficient iodine intake who were exposed to fluoride at a concentration of 30 mg/L [53]. These same changes were observed among iodine-deficient rats who were exposed to fluoride at a lower concentration of only 10 mg/L. Another study with adult mice observed lower levels of triiodothyronine (T3) and thyroxine

(T4) among mice with a deficient iodine intake coupled with low fluoride intake when compared with mice with a moderate iodine intake [52]. Thus, the relationship between fluoride exposure and thyroid function may differ as a function of iodine intake.

The impact of fluoride and iodine on thyroid hormones during pregnancy is especially relevant to cognitive abilities in offspring. The fetus is entirely dependent on maternal thyroid hormones until mid-gestation and continues to be partially dependent until birth [54]. Even subtle changes in maternal thyroid hormone levels in pregnancy can have adverse effects on brain structure [55–57] and neurodevelopment [55,56,58]. Low iodine and exposure to higher levels of fluoride in drinking water during pregnancy are both independently associated with a greater risk of developing hypothyroidism [59,60]. Our results are consistent with the combination of low iodine and high fluoride compounding thyroid disruption during fetal development, the most vulnerable period of brain development.

In our study, the interaction between fluoride and iodine was only evident in boys. This finding is consistent with a recent cross-sectional study conducted in China showing that iodine modified the susceptibility of the thyroid gland to fluoride exposure in school-aged boys, but not girls [45]. For boys with lower urinary levels of iodine, higher urinary fluoride was associated with larger thyroid volumes, whereas higher levels of iodine reduced the effects of fluoride on the thyroid. To our knowledge, no study has examined sex-specific effects of the interaction between prenatal fluoride and iodine on neurodevelopmental outcomes, but some epidemiological and animal studies of fluoride neurotoxicity found that boys are more vulnerable to prenatal fluoride exposure than girls [33]. Sex-specific effects in the interaction between fluoride and iodine, particularly among mothers with insufficient iodine intake, may disrupt in utero thyroid hormones. Given that the thyroid gland expresses estrogen and androgen receptors, boys and girls may respond differently to thyroid hormone insufficiency [61–63]. One study, for example, found that maternal trajectories of thyroid hormones were associated with preschool boys' behavioural development but not girls' [64]. Taken together, these findings indicate that future investigations of fluoride's neurotoxicity should examine the roles of iodine intake and child sex, and whether thyroid hormones mediate the pathway for fluoride and iodine's effects on boys' IQ.

#### *Strengths and Limitations*

Our study has several strengths, including a modest-sized pregnancy cohort, prospective design, state-of-the-art biomarkers of fluoride exposure and iodine status, available information on a wide array of potential maternal and child confounders, and use of standardized and valid measures of child intelligence. Our study also has limitations. Compared with the general Canadian population, women in the MIREC cohort were more educated, older, predominantly Caucasian, and more likely to be married or in common law relationships [34], which may limit the generalizability of our findings. The high use of prenatal multivitamins in our sample likely reduced the risk of moderate-to-severe iodine deficiency which may be observed in other populations. Even though we used state-of-the-art biomarkers, fluoride and iodine both have short half-lives and could therefore be impacted by recent fluoride or iodine ingestion. Further, we measured iodine and fluoride in urine spot samples instead of 24 h urine samples. We attempted to minimize this limitation by averaging urine fluoride across all three trimesters of pregnancy, and urine iodine across two trimesters of pregnancy. Nonetheless, we acknowledge that up to ten repeat spot urine samples may be needed to accurately reflect individual iodine status [65].

#### **5. Conclusions**

This is the first prospective epidemiological study to estimate the interplay between prenatal fluoride exposure and maternal iodine status in relation to child IQ in boys and girls. Our findings indicate that the association between prenatal fluoride exposure and full-scale intelligence previously identified in this cohort [6] was exacerbated by low maternal iodine in pregnancy among boys. These results, which were found among mother-child



pairs living in fluoridated and non-fluoridated communities in Canada, underscore the importance of sufficient iodine intake in pregnancy to minimize the neurotoxicity of fluoride in boys.

**Author Contributions:** Conceptualization, C.V.G., M.H., R.G. and C.T.; methodology, C.V.G., M.H., D.F., R.H. and C.T.; software, C.V.G.; validation, D.F. and R.H.; formal analysis, C.V.G.; investigation, B.L. and E.A.M.-M.; resources, E.A.M.-M.; data curation, C.V.G.; writing—original draft preparation, C.V.G. and M.H.; writing—review and editing, C.V.G., M.H., R.G., J.C., P.A., E.A.M.-M., T.M., J.K., D.F., R.H., B.L. and C.T.; visualization, C.V.G.; supervision, C.T.; project administration, C.T.; funding acquisition, B.L., P.A. and C.T. All authors have read and agreed to the published version of the manuscript.

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**Institutional Review Board Statement:** The study was conducted in accordance with the Declaration of Helsinki, and approved by the Institutional Review Board at Health Canada (2019-023H) and York University (12 December 2019).

**Informed Consent Statement:** Informed consent was obtained from all subjects involved in the study.

**Data Availability Statement:** Anonymized data described in the manuscript are available to qualified investigators with IRB approval.

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## Fluoride exposure and hypothyroidism in a Canadian pregnancy cohort

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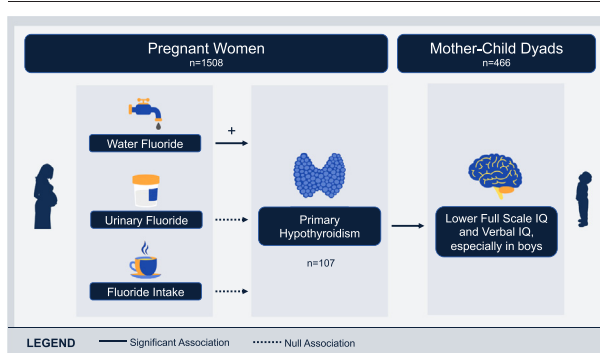
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### HIGHLIGHTS

- Fluoride may disrupt thyroid function.
- Thyroid dysfunction in pregnancy can adversely impact offspring development.
- Fluoride in water increased risk of hypothyroidism in pregnant women.
- Boys had lower IQ scores if their mothers were hypothyroid in pregnancy.
- Thyroid disruption may contribute to developmental neurotoxicity of fluoride.

### GRAPHICAL ABSTRACT



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### ABSTRACT

**Background:** While fluoride can have thyroid-disrupting effects, associations between low-level fluoride exposure and thyroid conditions remain unclear, especially during pregnancy when insufficient thyroid hormones can adversely impact offspring development.

**Objectives:** We evaluated associations between fluoride exposure and hypothyroidism in a Canadian pregnancy cohort.

**Methods:** We measured fluoride concentrations in drinking water and three dilution-corrected urine samples and estimated fluoride intake based on self-reported beverage consumption. We classified women enrolled in the Maternal-Infant Research on Environmental Chemicals Study as euthyroid ( $n = 1301$ ), subclinical hypothyroid ( $n = 100$ ) or primary hypothyroid ( $n = 107$ ) based on their thyroid hormone levels in trimester one. We used multinomial logistic regression to estimate the association between fluoride exposure and classification of either subclinical or primary hypothyroidism and considered maternal thyroid peroxidase antibody (TPOAb) status, a marker of autoimmune hypothyroidism, as an effect modifier. In a subsample of 466 mother-child pairs, we used linear regression to explore the association between maternal hypothyroidism and child Full-Scale IQ (FSIQ) at ages 3-to-4 years and tested for effect modification by child sex.

**Results:** A 0.5 mg/L increase in drinking water fluoride concentration was associated with a 1.65 (95% confidence interval [CI]: 1.04, 2.60) increased odds of primary hypothyroidism. In contrast, we did not find a significant association between urinary fluoride (adjusted odds ratio [aOR]: 1.00; 95%CI: 0.73, 1.39) or fluoride intake (aOR: 1.25; 95%CI: 0.99, 1.57) and hypothyroidism. Among women with normal TPOAb levels, the risk of primary hypothyroidism increased with both increasing water fluoride and fluoride intake (aOR water fluoride concentration: 2.85; 95%CI:

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1.25, 6.50; aOR fluoride intake: 1.75; 95%CI: 1.27, 2.41). Children born to women with primary hypothyroidism had lower FSIQ scores compared to children of euthyroid women, especially among boys (B coefficient:  $-8.42$ ; 95 % CI:  $-15.33, -1.50$ ).

**Discussion:** Fluoride in drinking water was associated with increased risk of hypothyroidism in pregnant women. Thyroid disruption may contribute to developmental neurotoxicity of fluoride.

## 1. Introduction

Fluoride is added to some public water supplies and dental products to prevent dental caries (Community Water Fluoridation, 2019). Drinking water is the largest source of fluoride for children and adults living in fluoridated communities, accounting for 40 %–70 % of daily intake (United States Environmental Protection Agency, n.d.). Black tea is another dietary source of fluoride (Krishnankutty et al., 2021). Questions about systemic fluoride exposure during pregnancy have increased following findings from studies linking gestational exposure to fluoride with neurodevelopmental deficits, including lower intelligence quotient (IQ) (Bashash et al., 2017; Cantoral et al., 2021; Green et al., 2019).

Disruption of thyroid hormone is a potential mechanism underlying developmental fluoride neurotoxicity (National Toxicology Program, n.d.; National Research Council, 2006), especially during the first 10–12 weeks of gestation when the fetus is exclusively reliant on maternal thyroid hormones (Andersen et al., 2013; de Escobar et al., 2004; Chevrier et al., 2011). Maternal thyroid hormone insufficiency (i.e., hypothyroidism) early in gestation is associated with adverse effects on offspring development, including preterm birth (Andersen et al., 2013) and diminished IQ (Andersen et al., 2018; Haddow et al., 1999). A meta-analysis of three prospective birth cohorts found that maternal hypothyroxinemia (i.e., normal thyroid stimulating hormone [TSH] and low [ $<2.5$ th percentile] free thyroxine [FT4]) was associated with a 3 to 4-point reduction in offspring IQ (Levie et al., 2018).

While thyroid-disrupting effects of fluoride have been known since the 1930s (Day and Powell-Jackson, 1972), evidence of an association between low-level exposure to fluoride and thyroid disruption is mixed with few studies conducted in pregnancy. In experimental studies, Wistar rats whose mothers were exposed in pregnancy to higher levels of fluoride (i.e., 20 mg/kg of body weight (Banji et al., 2013) and  $>100$  ppm (Basha et al., 2011)) showed decreases in serum FT4 and free triiodothyronine (FT3). Similar findings were reported in female and male Wistar rats at lower, prolonged fluoride exposure levels (Jiang et al., 2016), but not in Long-Evans hooded male rats (McPherson et al., 2018), perhaps reflecting strain differences in response to fluoride exposure (Kang et al., 1986).

In children and adults, higher fluoride levels in drinking water and in urine were associated with elevated serum TSH and lower serum free and total T4 and T3 levels, considered characteristic of hypothyroidism (Khandare et al., 2018; Kheradpisheh et al., 2018; Wang et al., 2020). Likewise, in a population-based study conducted in England, hypothyroidism was 1.6 times more likely among adults living in areas with water fluoride levels  $>0.7$  mg/L compared to areas with water-fluoride concentrations  $<0.3$  mg/L (Peckham et al., 2015). In contrast, an observational study (Barberio et al., 2017) conducted in Canada reported no association between fluoride exposure and thyroid hormone levels or self-reported diagnosis of a thyroid condition. However, when iodine status was considered, an association between water fluoride concentration and TSH was found among iodine deficient individuals (Malin et al., 2018). A 2018 systematic review also reported a positive relationship between fluoride exposure and hypothyroidism in children and adults (Chaitanya et al., 2018), though this association was limited to a small number of ecological and cross-sectional studies.

To date, no studies have assessed the association between fluoride exposure and thyroid function in pregnant women living in areas with optimally fluoridated water. If fluoride is associated with a reduction in thyroid hormone, thereby reducing the availability of the hormone to the fetus, then

fluoride could affect offspring cognitive function. We evaluated the potential thyroid-disrupting effects of fluoride exposure in pregnancy and explored whether hypothyroidism in pregnancy was associated with children's IQ among Canadian mother-child dyads.

## 2. Methods

### 2.1. Participants

Pregnant women were enrolled in the Maternal-Infant Research on Environmental Chemicals (MIREC) Study (Arbuckle et al., 2013) between 2008 and 2011 from ten cities across Canada, seven of which add fluoride to drinking water (Toronto, Hamilton, Ottawa, Sudbury, Halifax, Edmonton, Winnipeg) and three of which do not (Vancouver, Montreal, Kingston). Women were eligible to participate if they were  $\geq 18$  years of age, able to communicate in English or French, and  $<14$  weeks' gestation. Participants were considered ineligible if they had known fetal abnormalities, medical complications, or reported drug use. Of 2001 women recruited, 1983 consented to participate; of these, 1885 (95.1 %) provided plasma samples in trimester one.

Mothers of singleton children born  $>28$  weeks' gestation who were between the ages of 3–4 years of age at time of study, with no congenital abnormalities, major neurological disorders, or history of convulsions were contacted to participate with their children in a neurodevelopmental follow-up study (i.e., MIREC-Child Development Plus). Owing to limited resources, in-person IQ testing was offered in six study sites (Toronto, Hamilton, Halifax, Vancouver, Kingston, and Montreal). Of 1207 eligible women, 808 consented to participate in this follow-up study; of these, 610 (76 %) agreed to child neurodevelopmental testing and 601 completed IQ testing in entirety.

The current study received approval from the research ethics boards at Health Canada and York University. All participants provided written informed consent at time of enrollment in MIREC and MIREC-Child Development Plus.

### 2.2. Maternal fluoride exposure

#### 2.2.1. Water fluoride (mg/L)

Municipal drinking water reports from 2008 to 2012 were solicited from municipal water treatment plants (WTPs) in all ten cities included in MIREC. These reports listed water fluoride concentrations that were measured daily if fluoride was added to public drinking water, and weekly or monthly if fluoride was not added to public water (Till et al., 2018). Using the first three letters of their postal code, participants' residences were matched with boundary regions serviced by each WTP. Average water fluoride concentration (i.e., geometric mean; mg/L) was estimated for each woman who reported drinking tap water in pregnancy by averaging water fluoride concentrations across each woman's pregnancy; thus, each woman has a water fluoride concentration that is matched in time to the levels of fluoride found in tap water for the duration of her pregnancy. Further details can be found in Till et al. (Till et al., 2018).

#### 2.2.2. Maternal fluoride intake (mg/day)

We collected information on women's consumption of tap water, tea, and coffee from a self-reported questionnaire completed in the first and third trimesters. We estimated daily maternal fluoride intake (mg/day) in trimester one and three separately by multiplying water fluoride concentration (mg/L) by total volume (L) of water, tea, and coffee consumed. Volume

of water and water-based beverages was only derived for women who reported drinking tap water in pregnancy. We then added the additional fluoride content that would be expected from drinking each 200-mL cup of black tea (0.326 mg) or green tea (0.260 mg) if made with deionized water (Krishnankutty et al., 2021). Estimated maternal dietary fluoride intake (mg/day) was derived by taking the average of fluoride intake for trimesters one and three. See Supplemental Material, Appendix A for the formula used to derive maternal fluoride intake.

### 2.2.3. Maternal urinary fluoride (MUF; mg/L)

MUF concentration was analyzed in spot urine samples collected in each trimester of pregnancy, using a modification of the hexamethyldisiloxane (HMDS; Sigma Chemical Co., USA) microdiffusion method with ion-selective electrode by the Indiana University School of Dentistry (Martínez-Mier et al., 2011). The limit of detection (LoD) was 0.02 mg/L; trimester-specific concentrations below the LoD were replaced with the value of  $\text{LoD}/\sqrt{2}$  ( $n = 23$ ), which is a validated method for estimation of the average concentration from data containing nondetectable values (Hornung and Reed, 1990). Each MUF concentration was standardized for urine specific gravity (SG) to account for variability due to urinary dilution using the following equation:  $\text{MUF}_{\text{SG}} = \text{MUF} \times [(\text{SG}_M - 1) \div (\text{SG}_i - 1)]$ , where  $\text{MUF}_{\text{SG}}$  is the SG-adjusted fluoride concentration (mg/L),  $\text{SG}_i$  is the observed SG concentration for the individual urine sample, and  $\text{SG}_M$  is the median SG for the cohort (Duty et al., 2005). We derived the average dilution-adjusted  $\text{MUF}_{\text{SG}}$  concentration by taking the average across all three trimesters for each woman. We removed one averaged  $\text{MUF}_{\text{SG}}$  value ( $>5$  mg/L) because of uncertainty that it reflected an individual's true exposure; this high  $\text{MUF}_{\text{SG}}$  concentration was driven by one trimester-specific value of 16 mg/L, which was not consistent with the other trimester values that were close to zero. Thus, this extreme value was more likely to represent fluoride ingestion (e.g., swallowing toothpaste prior to urine sample) rather than a reliable exposure measure.

For comparison, we also used urinary creatinine to correct for dilution when measuring MUF. Urinary creatinine was measured using a colorimetric end-point assay (Jaffe) on an Indiko instrument (Indiko Plus, ThermoFisher Scientific). An alkaline sodium picrate solution was used to react with creatinine in urine to form a red Janovski complex using Mircogenics DRI® Creatinine-Detect® Test. The absorbance was read at 510 nm on an Indiko chemistry autoanalyzer with a detection limit of 0.069 mmol/L, reporting limit of 0.23 mmol/L, and reproducibility of 2.2%. While both methods are commonly used to correct for urine dilution with little difference observed between the two methods (Till et al., 2018), we chose to use  $\text{MUF}_{\text{SG}}$  for our primary model because of the larger sample size relative to creatinine-adjusted MUF.

### 2.3. Classification of thyroid status

TSH and FT4 were analyzed in maternal plasma samples from the first trimester (mean [standard deviation (SD)] = 11.6 (1.6) weeks). Plasma FT4 was measured using gold standard equilibrium dialysis isotope dilution mass spectrometry (ED-ID-MS) by the accredited Toxicology Laboratory at the Institut National de Santé Publique du Québec (INSPQ). Plasma FT4 was considered normal if it fell between the 10th and 90th percentiles of FT4 levels for all women in MIREC (i.e., 11–17 pg/mL). Plasma TSH was quantified using a commercial immunoassay by an accredited biochemistry laboratory at the Institut Universitaire de Cardiologie et de Pneumologie de Québec (IUCPQ) and compared against the trimester one reference range as recommended by the American Thyroid Association (Stagnaro-Green et al., 2011) (i.e., 0.1–2.5  $\mu\text{IU/mL}$ ). The LoD was 0.0025  $\mu\text{IU/mL}$ ; concentrations below the LoD ( $n = 7$ ) were given a value of  $\text{LoD}/\sqrt{2}$  (Hornung and Reed, 1990). We also measured thyroid peroxidase antibody (TPOAb) levels, a marker of autoimmune hypothyroidism. Elevated TPOAb levels (considered in this study as  $\geq 5.61$  IU/mL based on lab protocols from the IUCPQ), which have been associated with a reduced capacity to regulate thyroid hormones due to autoimmune destruction of the thyroid gland, have been shown to modify the association between per- and

polyfluoroalkyl substances and thyroid hormones in pregnant women (Webster et al., 2014).

Women were classified as euthyroid if their TSH and FT4 levels fell within the population reference range (i.e., 0.1–2.5  $\mu\text{IU/mL}$  and 11–17 pg/mL, respectively), and subclinical hypothyroid if their TSH levels were above the top limit of the reference range but FT4 levels were within range (i.e., 2.5–10  $\mu\text{IU/mL}$  (Stagnaro-Green and Pearce, 2012) and 11–17 pg/mL, respectively). Notably, women were classified as primary hypothyroid if their TSH levels were high and FT4 levels were low (i.e.,  $>2.5$   $\mu\text{IU/mL}$  and  $< 11$  pg/mL, respectively), or if they had reported a prior clinical diagnosis of hypothyroidism. Women meeting criteria for hyperthyroidism (subclinical or primary) were excluded from all analyses.

### 2.4. Measures of iodine status

Thyroglobulin (Tg) was measured by the accredited Toxicology Laboratory at the INSPQ using an Abbott Architect i2000SR immunoassay analyzer. Tg was used as a biomarker of long-term iodine nutrition (Ma and Skeaff, 2014), which was used as a covariate in sensitivity analyses. The LoD was 0.09  $\mu\text{g/L}$ . In addition, we describe urinary iodine adjusted for creatinine (UIC/Cr) as well as daily iodine intake in our sample. UIC/Cr ( $\mu\text{g/g}$ ) was calculated by dividing UIC ( $\mu\text{g/L}$ ) by creatinine concentration ( $\text{g/L}$ ). Daily iodine intake was estimated using the following formula:  $\text{UIC/Cr} \times \text{predicted 24-hour creatinine excretion}/0.92$ . Predicted 24-hour urine creatinine was estimated using an established equation for adult females as described in Krzeczowski et al. (2022) (Krzeczowski et al., 2022); 0.92 is the urinary iodine excretion rate.

### 2.5. Children's intelligence

Intellectual abilities were assessed at 3-to-4 years of age using the Wechsler Preschool and Primary Scale of Intelligence-III [Canadian norms; mean (SD) = 100 (15)]. Testing was conducted in the child's primary language (English or French) by trained research assistants who were blinded to women's fluoride exposure and thyroid status in pregnancy. Integrity of test administration was ensured by periodic observation of testers and double scoring of all protocols. We used Full-Scale IQ (FSIQ), a measure of global intellectual and cognitive functioning, as the primary outcome. Verbal (VIQ) and Performance IQ (PIQ) were used in supplementary analyses.

### 2.6. Statistical analysis

We examined the distribution and descriptive statistics for all demographics, maternal fluoride exposure and thyroid hormone variables, and child FSIQ. We used Spearman correlations to examine associations between exposure variables. Our primary analyses used multinomial logistic regression to estimate odds of subclinical and primary hypothyroidism associated with each exposure measure (water fluoride concentration, fluoride intake, and  $\text{MUF}_{\text{SG}}$  concentration). We adjusted for maternal age, level of education (dichotomized as bachelor's degree or higher), pre-pregnancy body mass index (BMI), and race (White or Other). Race was self-reported by participants and dichotomized given the majority of women (86%) in MIREC identified as White, with the remaining individuals identifying as other categories, including Asian/Pacific Islander, multi-racial or other. Race was included as a covariate given evidence of disparities in women's exposures to, and metabolism of, environmental chemicals (Nguyen et al., 2020), and evidence of differences in diagnosis of thyroid disorders by race (McLeod et al., 2014). Covariates were included in models based on prior studies suggesting that they are causal determinants of thyroid function and may be associated with fluoride exposure (Till et al., 2018; Buzalaf and Whitford, 2011; Collares et al., 2017). We also adjusted for study site when  $\text{MUF}_{\text{SG}}$  was used as the independent variable because thyroid function problems could vary across the study sites. The same set of covariates was used in the models with fluoride intake and water fluoride concentration, apart from study site because site

is collinear with fluoride levels in municipal drinking water (which was used to derive fluoride intake). Moreover, the models involving fluoride intake or water fluoride concentration only included women who reported drinking tap water in pregnancy. We tested for effect modification by maternal TPOAb status in all models through inclusion of interaction terms. If the interaction term was significant, we probed the predicted slopes by running each model twice, once with normal TPOAb levels (<5.61 IU/mL) set as the reference category, and again with high TPOAb levels ( $\geq 5.61$  IU/mL) as the reference.

For all models, odds ratios and associated confidence intervals (CIs) were reported per 0.5 mg/L or mg/day increase in water fluoride concentration, fluoride intake, and MUF<sub>SG</sub> concentration; 0.5 mg/L corresponds to the approximate difference in water fluoride concentration between a fluoridated and non-fluoridated community and interquartile range (IQR) for MUF<sub>SG</sub>, and 0.5 mg/day represents the approximate difference in dietary fluoride consumption between women at the 25th and 60th percentiles of fluoride intake. Regression diagnostics confirmed no assumption violations, issues with model fit, collinearity, heteroskedasticity, influential cases, or outliers in any of the above models.

As a secondary aim, we investigated the association between maternal hypothyroidism and child IQ. We used multiple linear regression to test whether subclinical or primary hypothyroidism was associated with child FSIQ; this analysis included a total of 466 mother-child dyads with thyroid and FSIQ data, of whom 411 women were classified as euthyroid, 27 women as subclinical hypothyroid, and 28 as primary hypothyroid. Effect modification by child sex was explored through inclusion of interaction terms. Given the smaller sample size, we probed an interaction if the *p* value for the interaction term was <0.15 (Rothman, 2014). We estimated slopes by running each model twice, once with female children set as the reference category, and again with male children as the reference. Models involving IQ were adjusted for maternal age, race, level of education, second-hand smoke exposure (yes/no/I don't know), parity, study site, child sex, and a continuous measure of the quality of the home environment (HOME score). We did not adjust for gestational age in these models given that gestational age may be a causal intermediate between hypothyroidism in pregnancy and child IQ (Consortium on Thyroid and Pregnancy – Study Group on Preterm Birth, 2019; Nasirkandy et al., 2017); however,

gestational age was added to our model in a sensitivity analysis. We further probed the association between primary hypothyroidism and IQ using VIQ and PIQ. Participants who were missing covariates (<7.5 % of total sample; see Fig. 1) were excluded from all models.

We used STATA version 17.0 (STATA corporation) for all statistical analyses. Two-sided *p* values  $\leq .05$  were considered to indicate statistical significance.

## 2.7. Sensitivity analyses

For primary models investigating associations between fluoride exposure and hypothyroidism, we conducted the following sensitivity analyses: First, we entered thyroglobulin (Tg), a biomarker of long-term iodine nutrition, as a covariate in all three multinomial regression models used to estimate odds of subclinical or primary hypothyroidism associated with each exposure measure to evaluate confounding by iodine insufficiency in these relationships. Second, we ran three separate binary logistic regression models to estimate odds of primary hypothyroidism associated with water fluoride concentration, fluoride intake, and MUF<sub>SG</sub> concentration including only women who self-reported clinical diagnoses of primary hypothyroidism at the time of enrollment in MIREC. Third, we ran seven separate multinomial logistic regression models to estimate the association between water fluoride concentration and odds of primary hypothyroidism with adjustment for other environmental toxicants, including arsenic, lead, manganese, mercury, perfluorooctanoic acid (PFOA), perfluorooctanesulfonic acid (PFOS), and perfluorohexanesulfonic acid (PFHxS) measured in trimester one. Lastly, we reran the models used to estimate odds of either subclinical or primary hypothyroidism associated with maternal fluoride intake and MUF<sub>SG</sub> concentration using data from trimester one only given that these values were measured at the same time as the thyroid variables and may better represent pre-pregnancy fluoride exposure.

Given the association between hypothyroidism and child FSIQ, we explored whether primary hypothyroidism in pregnancy would mediate the significant association between water fluoride concentration and children's FSIQ that we previously reported in the MIREC sample (Green et al., 2019). The current analysis was considered exploratory given the limited number of participants (*n* = 358) with data related to water fluoride concentration,

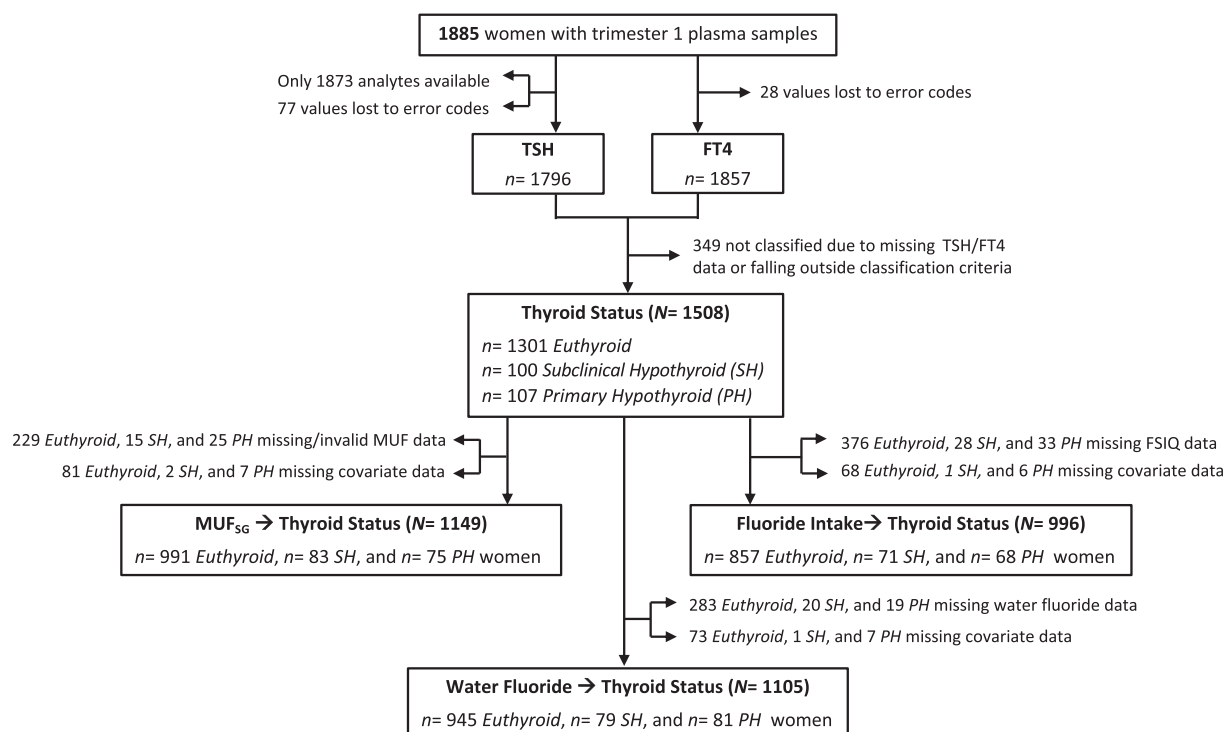


Fig. 1. Study sample flow chart for primary analyses.



**Table 1**  
Demographic characteristics of women classified as euthyroid, subclinical hypothyroid, and primary hypothyroid.

	Overall cohort	Euthyroid	Subclinical hypothyroid	Primary hypothyroid	Missing <sup>a</sup>
<i>N</i>	1508	1301	100	107	377
Maternal age (years; mean; SD)	32.2 (5.0)	32.1 (5.0)	32.1 (4.8)	33.2 (5.1)**	32.2 (5.2)
Ethnicity ( <i>n</i> ; %)					
White	1302 (86.3)	1117 (85.9)	86 (86.0)	99 (92.5)*	317 (84.1)
Other	206 (13.7)	184 (14.1)	14 (14.0)	8 (7.5)*	60 (15.9)
Marital status ( <i>n</i> ; %)					
Married or common law	1436 (95.2)	1238 (95.2)	94 (94.0)	104 (97.2)	357 (94.7)
Single	72 (4.8)	63 (4.8)	6 (6.0)	3 (2.8)	20 (5.3)
Level of education ( <i>n</i> ; %)					
College diploma or less	558 (37)	483 (37.1)	37 (37.0)	38 (35.5)	154 (40.8)
University degree	950 (63)	818 (62.9)	63 (63.0)	69 (64.5)	223 (59.2)
Household income (CAD) ( <i>n</i> ; %)					
<100,000	856 (59.6)	753 (60.7)	52 (53.1)	52 (52.0)*	221 (60.4)
≥ 100,000	580 (40.4)	487 (39.3)	45 (46.9)	48 (48.0)*	145 (39.6)
City ( <i>n</i> ; %)					
Fluoridated <sup>b</sup>	912 (60.5)	774 (59.5)	61 (61.0)	77 (71.9)**	246 (65.3)
Non-fluoridated <sup>c</sup>	596 (39.5)	527 (40.5)	39 (39.0)	30 (28.1)**	131 (34.7)
Second-hand smoke in trimester 1 ( <i>n</i> ; %)					
Yes	93 (6.2)	80 (6.2)	6 (6.0)	7 (6.6)	22 (5.9)
No	1414 (93.8)	1221 (93.8)	94 (94.0)	99 (93.4)	354 (94.1)
Pre-pregnancy BMI (kg/m <sup>2</sup> ; mean; SD)	24.8 (5.4)	24.7 (5.3)	24.3 (5.9)	26.4 (6.5)**	25.5 (5.7)**
Parity ( <i>n</i> ; %)					
0	662 (43.9)	567 (43.6)	52 (52.0)**	43 (40.2)	173 (45.9)
1	622 (41.3)	533 (41.0)	42 (42.0)	47 (43.9)	143 (37.9)
2+	224 (14.8)	201 (15.4)	6 (6.0)**	17 (15.9)	61 (16.2)
Gestational age (weeks; mean; SD) <sup>d</sup>	11.6 (1.6)	11.6 (1.6)	11.7 (1.4)	11.8 (1.4)	11.5 (1.6)
Maternal fluoride exposure					
MUF (mg/L; mean; SD)	0.59 (0.42)	0.59 (0.42)	0.57 (0.40)	0.62 (0.35)	0.61 (0.38)
Water fluoride (mg/L; mean; SD)	0.41 (0.26)	0.41 (0.26)	0.42 (0.25)	0.48 (0.24)**	0.44 (0.25)**
Fluoride intake (mg/day; mean; SD)	0.66 (0.50)	0.65 (0.50)	0.67 (0.48)	0.78 (0.59)**	0.69 (0.48)
Maternal thyroid hormones					
TSH (mean; SD)	1.4 (1.1)	1.2 (0.55)	3.1 (0.66)**	3.2 (2.8)**	1.2 (1.6)**
FT4 (pg/mL; mean; SD)	13.5 (1.9)	13.5 (1.7)	13.1 (1.5)**	14.0 (3.7)*	14.3 (9.3)*
TT4 (ng/mL; mean; SD)	106.1 (20.3)	105.8 (19.8)	107.1 (18.9)	109.3 (26.6)*	108.9 (26.1)**
Tg (ng/mL; mean; SD)	17.4 (16.8)	17.7 (16.6)	16.6 (17.2)	14.9 (19.4)*	18.1 (17.0)
Maternal thyroid antibodies					
Anti-Tg (IU/mL; mean; SD)	12.3 (57.4)	7.5 (39.2)	33.0 (107.8)**	52.2 (124.1)**	9.9 (50.0)
Anti-TPO (IU/mL; mean; SD)	28.5 (107.5)	15.0 (73.1)	51.9 (123.4)**	183.4 (252.7)**	22.6 (100.5)
Anti-TPO + (≥ 5.61 IU/mL; <i>n</i> ; %)	232 (15.6)	137 (10.6)	33 (33.0)**	62 (63.3)**	40 (11.4)**
Iodine intake (µg/day; mean; SD)	444.0 (343.5)	440.9 (356.7)	458.0 (273.8)	470.0 (208.4)	482.4 (442.8)*
UIC/Cr (µg/g; mean; SD)	344.5 (252.7)	342.1 (261.0)	370.6 (218.3)	348.7 (167.6)	362.6 (326.8)
Reported taking a prenatal vitamin ( <i>n</i> ; %)	1323 (87.8)	1134 (87.2)	95 (95.0)**	94 (87.9)	325 (86.2)
Reported taking thyroid medication ( <i>n</i> ; %)	78 (5.2)	0 (0.0)	0 (0.0)	78 (72.9)**	4 (1.1)**
Child sex ( <i>n</i> ; %)					
Male	763 (52.2)	676 (53.5)	49 (50.0)	38 (37.6)**	211 (57.5)*
Female	699 (47.8)	587 (46.5)	49 (50.0)	63 (62.4)**	156 (42.5)*
Child IQ (mean; SD)					
Full scale IQ	107.3 (13.7)	107.6 (13.8)	108.5 (13.0)	101.5 (11.9)**	104.9 (12.9)**
Verbal IQ	109.6 (13.5)	109.8 (13.6)	110.1 (11.5)	106.4 (13.8)	108.1 (12.4)
Performance IQ	103.4 (14.9)	103.9 (14.9)	104.7 (14.4)	96.3 (13.3)**	101.0 (14.7)*
HOME score (mean; SD)	47.3 (4.3)	47.3 (4.2)	47.7 (4.9)	46.6 (4.4)	47.1 (4.5)

Due to missing data, percentage totals for subgroups may not sum to the total sample population; percentages are reported based on total sample in each subgroup with available data.

\*/\*\* Denotes significant differences (\*:  $p < .10$ ; \*\*:  $p < .05$ ) between subclinical or primary hypothyroid women compared with euthyroid women, or between women with missing thyroid data compared with the overall cohort. Calculated using the Chi-square test for categorical variables and *t*-test for continuous variables.

Abbreviations: CAD = Canadian; SD = standard deviation; IQ = intelligence quotient; HOME = home observation measurement of the environment; MUF<sub>SG</sub> = maternal urinary fluoride, adjusted for specific gravity; TSH = thyroid stimulating hormone; FT4 = free thyroxine; TT4 = total thyroxine; Tg = thyroglobulin; TPO = thyroid peroxidase; UIC/Cr = urinary iodine concentration, adjusted for creatinine.

<sup>a</sup> Refers to women in the MIREC cohort who provided a blood plasma sample in trimester one but did not have TSH and/or T4 measured, or did not meet criteria for any thyroid function category (i.e., euthyroid, subclinical hypothyroid, or primary hypothyroid).

<sup>b</sup> Edmonton, Winnipeg, Toronto, Hamilton, Sudbury, Ottawa, Halifax.

<sup>c</sup> Vancouver, Kingston, Montreal.

<sup>d</sup> Gestational age at time of maternal blood collection in trimester one.

child FSIQ, and thyroid status. Notably, only 25 of 107 (23.4 %) women in the primary hypothyroid group were retained in the mediation analysis. We used mediation analysis within the counterfactual framework (Valeri and VanderWeele, 2013) (via *paramed* package in STATA) to explore the potential mediating effect (or indirect effect) of primary hypothyroidism in the association between water fluoride concentration and child FSIQ. We estimated the effect of a 0.5 mg/L increase in water fluoride concentration on child FSIQ. Mediation models were rerun using child VIQ and PIQ.

### 3. Results

Fig. 1 shows the sample flow chart for all primary variables and subgroups of interest, with detailed descriptions of where there was missing exposure, outcome, and covariate data. We studied a total of 1508 women (mean (SD) age = 32.2 (5.02) years) with thyroid data, of whom 1301 women (86.3 %) were classified as euthyroid, 100 met criteria for subclinical hypothyroidism (6.6 %), and 107 (7.1 %) met criteria for primary hypothyroidism; among the primary hypothyroid group, 79 women reported a diagnosis at time of study enrollment and 28 were identified based on thyroid hormone levels measured in the first trimester. As expected, among the women who had TPOAb data, more women in the subclinical (33 of 100; 33.0 %) and primary hypothyroid (62 of 98; 63.3 %) groups had elevated TPOAb levels ( $\geq 5.61$  IU/mL) relative to the euthyroid group (137 of 1295; 10.6 %), suggesting active or incipient autoimmune hypothyroidism (Table 1). As predicted, water fluoride concentration was moderately-to-strongly associated with fluoride intake ( $r = 0.76, p < .01$ ) and urinary fluoride ( $r = 0.49, p < .01$ ) and fluoride intake was associated with urinary fluoride ( $r = 0.59, p < .01$ ).

Note. MUF<sub>SG</sub> = maternal urinary fluoride, standardized for specific gravity; PH = primary hypothyroid; SH = subclinical hypothyroid.

#### 3.1. Maternal fluoride exposure and thyroid status

Among the 1105 women with a water fluoride measurement, 945 (85.5 %) were classified as euthyroid, 79 (7.2 %) as subclinical hypothyroid, and 81 (7.3 %) as primary hypothyroid. Mean water fluoride concentration was 0.42 mg/L (median = 0.52; range: 0.04 to 0.87 mg/L) and 60.5 % lived in fluoridated communities. Covariate-adjusted multinomial logistic regression revealed a statistically significant association between water fluoride concentration and risk of primary hypothyroidism; a 0.5 mg/L increase in water fluoride concentration was associated with 1.65 times greater odds (95 % CI: 1.04, 2.60) of having a diagnosis or meeting criteria for primary hypothyroidism. In contrast, no statistically significant association was

observed between water fluoride concentration and risk of subclinical hypothyroidism (adjusted odds ratio [aOR]: 1.15; 95 % CI: 0.73, 1.82) (Fig. 2).

Of the 996 women with a fluoride intake measurement, 857 (86.1 %) were classified as euthyroid, 71 (7.1 %) as subclinical hypothyroid, and 68 (6.8 %) as primary hypothyroid. Mean fluoride intake was 0.67 mg/day (median = 0.58; range: 0.02 to 2.84 mg/day) and 61 % lived in fluoridated communities. Daily fluoride intake was not significantly associated with risk of subclinical (aOR: 1.03; 95 % CI: 0.81 to 1.32) or primary hypothyroidism (aOR: 1.25; 95 % CI: 0.99, 1.57) (Fig. 2).

Among the 1149 women with a SG-adjusted urinary-fluoride measurement, 991 (86.3 %) were classified as euthyroid, 83 (7.2 %) as subclinical hypothyroid, and 75 (6.5 %) as primary hypothyroid. Mean MUF<sub>SG</sub> was 0.59 mg/L (median = 0.49; range: 0.05 to 3.33 mg/L) and 59.5 % lived in fluoridated communities. Results from multinomial logistic regression analysis indicated that MUF<sub>SG</sub> concentration was not significantly associated with risk of subclinical hypothyroidism (aOR: 0.94; 95 % CI: 0.67, 1.31) or primary hypothyroidism (aOR: 1.00; 95 % CI: 0.73, 1.39) (Fig. 2). Results did not differ when using creatinine-adjusted MUF concentration (Table S3).

#### 3.1.1. Effect modification by TPOAb status

The interaction between fluoride exposure and maternal TPOAb status in predicting risk of primary hypothyroidism was statistically significant for models with water fluoride concentration ( $p$  interaction term = 0.03) and daily fluoride intake ( $p$  interaction term = 0.01). Women with normal TPOAb levels were 2.85 times (95 % CI: 1.25, 6.50) more likely to have or meet criteria for primary hypothyroidism for each 0.5 mg/L increase in water fluoride concentration. Likewise, fluoride intake was significantly associated with risk of primary hypothyroidism among women with normal TPOAb levels (aOR: 1.75; 95 % CI: 1.27, 2.41). In contrast, there was no evidence of effect modification by maternal TPOAb status in the associations between water fluoride or fluoride intake and risk of subclinical hypothyroidism, or between MUF<sub>SG</sub> and risk of subclinical or primary hypothyroidism (Table 2).

#### 3.2. Maternal hypothyroidism and child intelligence

Demographic characteristics comparing the water fluoride cohorts with and without child IQ data are summarized in Table S1. Compared to women without child IQ data, those with child IQ data were more likely to be White, live in non-fluoridated cities, report no second-smoke exposure in the first trimester, and had lower MUF<sub>SG</sub> and water fluoride concentrations, and daily fluoride intake.

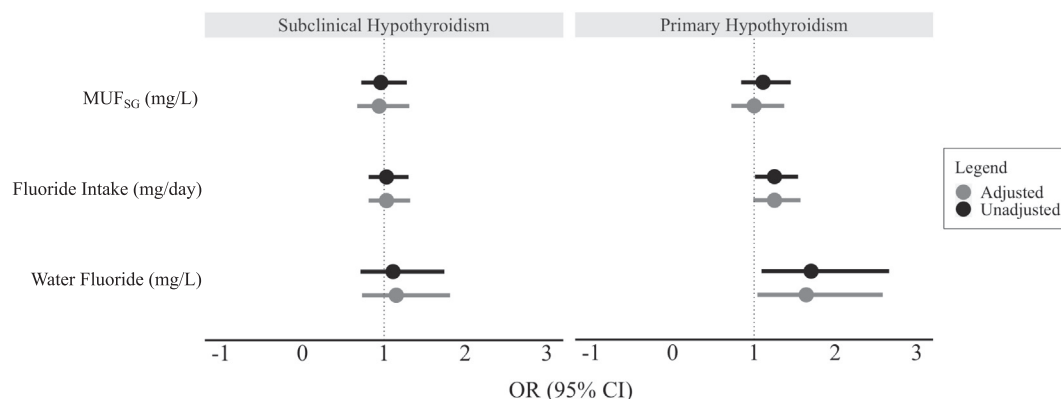


Fig. 2. Covariate-adjusted and unadjusted effect estimates of associations between water fluoride concentration, daily fluoride intake, and urinary fluoride concentration, and subclinical and primary hypothyroidism relative to euthyroid women.

Note. Covariate-unadjusted and adjusted effect estimates from multinomial logistic regression models shown in black and grey, respectively. Results are also summarized in Table 2. OR = odds ratio; reported for every 0.5 mg/L or 0.5 mg/day increase in water fluoride concentration, MUF<sub>SG</sub> concentration, and daily fluoride intake.

Abbreviations: MUF<sub>SG</sub> = maternal urinary fluoride, standardized for specific gravity; CI = confidence interval

**Table 2**

Associations between water fluoride concentration, daily fluoride intake, and urinary fluoride concentration, and subclinical and primary hypothyroidism in pregnant women participating in the MIREC study.

	Model 1 <sup>a</sup> : unadjusted				Model 2 <sup>b</sup> : covariate adjusted				Model 2 + normal TPOAb <sup>c</sup>				Model 2 + high TPOAb <sup>d</sup>			
	n	OR	95 % CI	p	n	aOR	95 % CI	p	n	aOR	95 % CI	p	n	aOR	95 % CI	p
<b>Primary hypothyroidism</b>																
Water fluoride (mg/L)	1185	1.71	1.10,2.67	0.02	1105	1.65	1.04,2.60	0.03	1094	2.85	1.25,6.50	0.01	1094	0.88	0.45,1.72	0.71
Fluoride intake (mg/day)	1071	1.25	1.01,1.54	0.04	996	1.25	0.99,1.57	0.06	988	1.75	1.27,2.41	0.00	988	0.87	0.58,1.31	0.51
MUF <sub>SG</sub> (mg/L)	1239	1.11	0.85,1.45	0.46	1149	1.00	0.73,1.39	0.98	1141	0.94	0.53,1.67	0.82	1141	0.90	0.56,1.44	0.66
<b>Subclinical hypothyroidism</b>																
Water fluoride (mg/L)	1185	1.12	0.71,1.74	0.63	1105	1.15	0.73,1.82	0.54	1094	1.15	0.67,1.99	0.61	1094	1.13	0.48,2.70	0.78
Fluoride intake (mg/day)	1071	1.03	0.81,1.30	0.83	996	1.03	0.81,1.32	0.79	988	0.98	0.73,1.33	0.91	988	1.18	0.73,1.89	0.51
MUF <sub>SG</sub> (mg/L)	1239	0.96	0.72,1.28	0.76	1149	0.94	0.67,1.31	0.71	1141	1.04	0.72,1.51	0.84	1141	0.74	0.37,1.49	0.40

<sup>a</sup> Model 1: multinomial logistic regression models of water fluoride concentration, daily fluoride intake, and MUF<sub>SG</sub> concentration predicting risk of either primary or subclinical hypothyroidism, not adjusted for covariates.

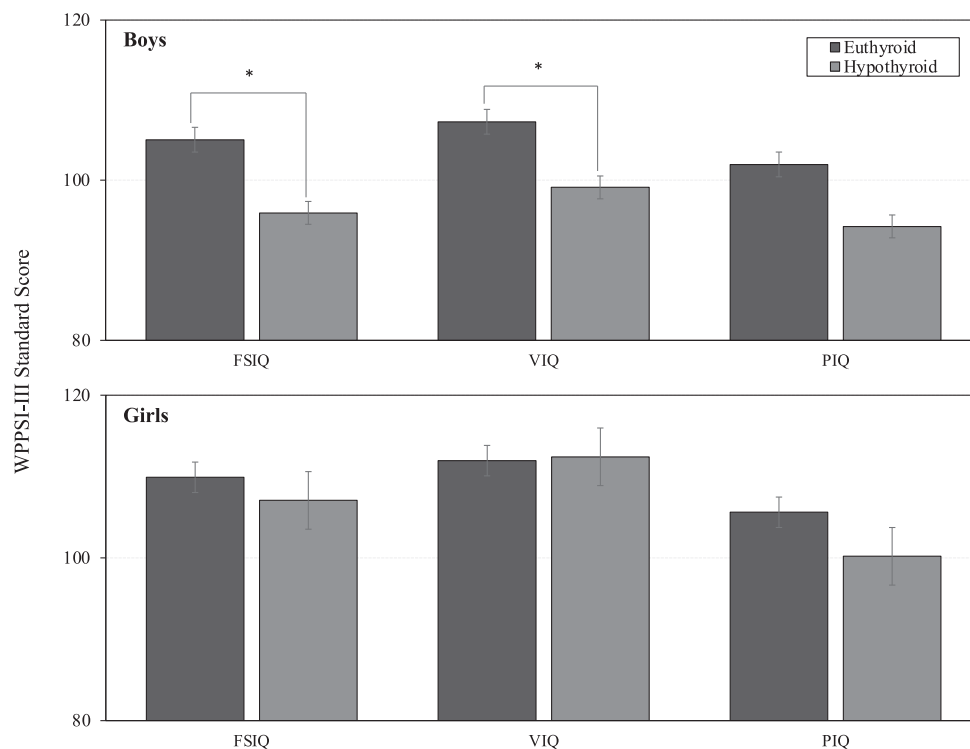
<sup>b</sup> Model 2: primary multinomial logistic regression models of water fluoride concentration, daily fluoride intake, and MUF<sub>SG</sub> concentration predicting risk of either primary or subclinical hypothyroidism, adjusted for relevant covariates.

<sup>c</sup> Model 2 + normal TPOAb: primary multinomial logistic regression models of water fluoride concentration, daily fluoride intake, and MUF<sub>SG</sub> concentration predicting risk of either primary or subclinical hypothyroidism for women with normal levels of TPOAb (i.e., <5.61 IU/mL).

<sup>d</sup> Model 2 + high TPOAb: primary multinomial logistic regression models of water fluoride concentration, daily fluoride intake, and MUF<sub>SG</sub> concentration predicting risk of either primary or subclinical hypothyroidism for women with elevated levels of TPOAb (i.e., ≥ 5.61 IU/mL). OR = Odds ratio, aOR = adjusted odds ratio; reported for every 0.5 mg/L or 0.5 mg/day increase in water fluoride concentration, MUF<sub>SG</sub> concentration, and daily fluoride intake. MUF<sub>SG</sub> = maternal urinary fluoride, standardized for specific gravity.

Median (IQR) FSIQ score was 108 (19) for the sample of 439 children with euthyroid or primary hypothyroid mothers (females: 110 (17); males: 105 (19)). FSIQ scores were 4.45-points (95 % CI: -9.17, 0.26) lower, on average, among children of primary hypothyroid women compared to children of euthyroid women. The interaction between maternal primary hypothyroidism and child sex in predicting child FSIQ scores met our threshold for model-selection purposes ( $p$  interaction term = 0.13).

Males born to women with primary hypothyroidism ( $n = 13$ ) had significantly lower FSIQ scores (B coefficient: -8.42; 95 % CI: -15.33 to -1.50) compared with males born to euthyroid women ( $n = 201$ ) (Fig. 3). In contrast, FSIQ scores did not differ significantly among females born to primary hypothyroid women ( $n = 15$ ) versus euthyroid women ( $n = 210$ ; B coefficient: -1.04; 95 % CI: -7.47, 5.38). Results remained the same with gestational age included as a covariate. Further probing of the



**Fig. 3.** Sex-specific effects in the association between maternal primary hypothyroidism and child FSIQ, VIQ, and PIQ.

Note. Top (left to right): mean FSIQ, VIQ, and PIQ scores of male children born to euthyroid (mean: FSIQ = 105.06, VIQ = 107.3, PIQ = 101.97) and primary hypothyroid (mean: FSIQ = 95.92, VIQ = 99.1, PIQ = 94.23) women.

Bottom (left to right): mean FSIQ, VIQ, and PIQ scores of female children born to euthyroid (mean: FSIQ = 109.91, VIQ = 111.96, PIQ = 105.61) and primary hypothyroid (mean: FSIQ = 107.07, VIQ = 112.43, PIQ = 100.20) women.

Error bars represent the 95 % confidence intervals.

\* Indicates a significant difference between means ( $p < .05$ ).

association between maternal primary hypothyroidism and child IQ indicated that the significant difference in FSIQ among male children born to primary hypothyroid versus euthyroid women was primarily driven by a difference in VIQ (B coefficient:  $-8.76$ ; 95 % CI:  $-15.59$  to  $-1.93$ ) and not PIQ (B coefficient:  $-5.93$ ; 95 % CI:  $-13.64$  to  $1.79$ ; Fig. 3). For complete results, see Table S2.

We did not observe a significant association between maternal subclinical hypothyroidism and child FSIQ ( $n = 438$ ; B coefficient:  $0.05$ ; 95 % CI:  $-4.78$ ,  $4.89$ ). Further, we found no evidence of effect modification by child sex.

### 3.3. Sensitivity analyses

#### 3.3.1. Primary models

Controlling for Tg as a covariate in our models estimating associations between fluoride exposure (water fluoride concentration, fluoride intake, and MUF<sub>SG</sub> concentration) and risk of either subclinical or primary hypothyroidism did not alter any of the results (Table S3).

When the sample was restricted to include only women who reported clinical diagnoses of primary hypothyroidism at the time of enrollment in MIREC ( $n = 59$ ), a  $0.5$  mg/L increase in water fluoride concentration was associated with 1.88 times greater odds (95 % CI:  $1.10$ ,  $3.21$ ) of having a diagnosis after controlling for covariates. Similarly, results revealed a statistically significant association between daily fluoride intake and diagnosed primary hypothyroidism (aOR:  $1.42$ ; 95 % CI:  $1.10$ ,  $1.82$ ). In contrast, no significant association was found between MUF<sub>SG</sub> concentration and diagnosed primary hypothyroidism (Table S3).

We found no evidence of confounding by other environmental toxicants (i.e., arsenic, lead, manganese, mercury, PFOA, PFOS, or PFHxS) in the association between water fluoride concentration and risk of primary hypothyroidism (Table S3).

Finally, MUF<sub>SG</sub> concentration from trimester one was not significantly associated with hypothyroidism (Table S3). In contrast, the association between daily fluoride intake and risk of primary hypothyroidism was statistically significant when using fluoride intake from trimester one in the model (aOR:  $1.42$ ; 95 % CI:  $1.18$ ,  $1.71$ ) (Table S3).

#### 3.3.2. Mediation analysis

Mediation analysis on the subsample of 358 mother-child pairs with water fluoride, thyroid (i.e., euthyroid and primary hypothyroid women), and FSIQ data, as well as complete covariates, indicated a significant direct effect of water fluoride concentration on child FSIQ (natural direct effect estimate =  $-3.55$ ; 95 % CI:  $-5.95$ ,  $-1.15$ ). In contrast, the indirect effect was close to null (natural indirect effect estimate =  $-0.12$ ; 95 % CI:  $-0.41$ ,  $0.16$ ), indicating that maternal primary hypothyroidism did not significantly mediate the relationship between maternal water fluoride concentration and child FSIQ in this subsample of mother-child pairs (Table 3). Likewise, there was no evidence of mediation when the model was rerun using child VIQ and PIQ (Table S4).

## 4. Discussion

In this Canadian pregnancy and birth cohort, fluoride in drinking water was associated with risk of primary hypothyroidism in pregnant women. Specifically, risk of hypothyroidism was 1.65 times higher per  $0.5$  mg/L increase in water fluoride concentration after accounting for potential confounding variables. To contextualize these results, the difference in water fluoride concentration between cities that are fluoridated at the recommended level of  $0.7$  mg/L and those without fluoridation is approximately  $0.5$  mg/L (Till et al., 2018). It is noteworthy that among the 1508 women for whom thyroid status was defined in the current study, 60.5 % lived in fluoridated areas, which is considerably higher than the proportion of the general Canadian population receiving fluoridated municipal drinking water (i.e., 38 %) (Public Health Agency of Canada, 2017). While fluoridated tap water is the main source of fluoride exposure among adults in fluoridated communities (United States Environmental Protection Agency,

**Table 3**

Maternal primary hypothyroidism as a mediator of the association between maternal water fluoride concentration and child FSIQ.

Mediation parameters	B	$p >  z $	95 % Confidence Interval	
Natural direct effect <sup>a</sup>	$-3.55$	$0.00$	$-5.95$	$-1.15$
Natural indirect effect <sup>b</sup>	$-0.12$	$0.40$	$-0.41$	$0.16$
Marginal total effect <sup>c</sup>	$-3.67$	$0.79$	$-30.66$	$23.32$

Output from mediation analysis in the counterfactual framework.

B = effect of a  $0.5$  mg/L increase in water fluoride concentration on child FSIQ.

<sup>a</sup> Effect of maternal water fluoride concentration on child FSIQ not mediated by maternal primary hypothyroidism.

<sup>b</sup> Effect of maternal water fluoride concentration on child FSIQ mediated by maternal primary hypothyroidism.

<sup>c</sup> Total effect of maternal water fluoride concentration on child FSIQ, mediated and not mediated by maternal primary hypothyroidism (i.e., sum of the natural direct<sup>a</sup> and indirect<sup>b</sup> effects).

n.d.), we also estimated fluoride intake by weighting each cup of water and other water-based beverages (i.e., tea, coffee) consumed by fluoride concentration in tap water. Findings revealed the same pattern of results, where higher fluoride intake was associated with risk of primary hypothyroidism, particularly among women with normal TPOAb levels.

In contrast, maternal urinary fluoride concentration was not associated with hypothyroidism. Fluoride concentrations in municipal water supplies are relatively constant and therefore are more likely to be indicative of chronic fluoride exposure, and perhaps body burden, than urinary fluoride. It is possible that day-to-day variations in intake of high-fluoride foods, beverages, or dental products before sample collection could influence urinary fluoride measurement and contribute to exposure misclassification that would bias estimates toward the null. Moreover, thyroid disorders tend to develop over time (Casey et al., 2017; Chatzitomaridis et al., 2017). Thus, it is reasonable that our more stable measure of fluoride exposure (i.e., water fluoride concentration) would be more strongly associated with risk of primary hypothyroidism than maternal urinary fluoride. Among the women with primary hypothyroidism, 73.8 % reported having a diagnosis prior to their pregnancy and enrollment in the MIREC study. In contrast, urinary fluoride was measured throughout pregnancy, and thus, following diagnosis for most women. Therefore, our findings make sense temporally in that we would not expect an exposure variable measured in pregnancy to predict risk of a health condition diagnosed before pregnancy. Similarly, we found a stronger association between daily fluoride intake and risk of primary hypothyroidism when looking at fluoride intake in trimester one only, perhaps indicating that women's self-reported beverage consumption in trimester one was more reflective of their pre-pregnancy beverage consumption habits. Prior research examining associations between fluoride exposure and other health outcomes have also reported stronger associations with water fluoride over urinary fluoride concentration (Green et al., 2019; Malin et al., 2018; Riddell et al., 2019).

We did not observe an association between maternal fluoride exposure and subclinical hypothyroidism. This may be because subclinical hypothyroidism is a milder form of hypothyroidism with more variable presentations that may be exacerbated by pregnancy-associated changes in thyroid hormone levels. Indeed, there is ongoing controversy as to whether subclinical hypothyroidism constitutes a clinical disorder that requires formal treatment, especially in pregnancy, given that abnormalities in thyroid hormone levels are commonly detected in routine blood work during pregnancy (Casey et al., 2017). Notably, when we restricted our analysis to include only women with a reported clinical diagnosis of hypothyroidism, the observed associations between maternal fluoride exposure and primary hypothyroidism were stronger than when we included women identified using trimester one thyroid hormone levels – perhaps reflecting imprecision in our classifications of maternal thyroid function.

Our findings are consistent with prior studies showing a relationship between fluoride exposure and thyroid function. An ecologic study

conducted in England reported a significantly higher prevalence of diagnosed hypothyroidism among adults living in areas with higher fluoride levels in drinking water (Peckham et al., 2015). Considering hypothyroidism is defined by elevated TSH and low FT4 levels, our findings are also in line with epidemiologic studies reporting associations between higher drinking water-fluoride concentrations and higher TSH and lower T4 levels in children and adults (Khandare et al., 2018; Kheradpisheh et al., 2018). In contrast, our results do not align with those of a Canadian study that reported no relationship between fluoride exposure (measured in urine and tap water) and self-reported diagnosis of a thyroid disorder among non-pregnant adults (Barberio et al., 2017). Discrepancy in results could reflect differences in exclusion and inclusion criteria (i.e., the prior study included respondents between the ages of 3–79 years), differences between a pregnant and non-pregnant sample, and differences in how thyroid disorders were classified (i.e., use of different cut-offs for elevated TSH and low FT4).

Fluoride may impact thyroid function by several potential mechanisms. It may inhibit the deiodinase enzymes that are necessary for thyroid hormone production, resulting in decreased blood-T3 and T4 levels and increases in circulating TSH (Malin et al., 2018; Susheela et al., 2005). Fluoride may also induce structural and functional changes to the follicular epithelial cells of the thyroid gland (e.g., decline in the colloidal content and damage to the endoplasmic reticulum) resulting in insufficient secretion of Tg, and thus disruption to thyroid hormone synthesis more broadly (Banji et al., 2013; Basha et al., 2011). Further, fluoride may interfere with iodine to exert its negative effects on thyroid function, perhaps by inhibiting the expression and activity of sodium iodide symporters that are necessary for mediating active iodide transport into the thyroid, resulting in lower iodine availability and the indirect suppression of thyroid hormone production (Greer et al., 2002; Waugh, 2019). Importantly, however, a recent experimental study (Buckalew et al., 2020) refuted this claim by showing that fluoride does not inhibit sodium iodide symporter activity in Fischer rat thyroid follicular cells. This, together with our prior finding of women in MIREC being largely iodine sufficient (Krzeczkowski et al., 2022), and evidence that the effect of water fluoride concentration on maternal primary hypothyroidism remains significant after controlling for a biomarker of chronic iodine nutrition (i.e., Tg), suggest that iodine deficiency is not a confounder in this study. Ultimately, further research in this area is needed to understand fluoride action on thyroid function and whether iodine modifies the neurotoxicity of fluoride as reported in prior studies (Malin et al., 2018; Goodman et al., 2022).

In our study, having an underlying autoimmune condition (i.e., Hashimoto's disease) did not increase vulnerability to fluoride-induced changes in thyroid gland functioning. Rather, our findings showed that pregnant women with normal TPOAb levels (<5.61 IU/mL) were most susceptible to fluoride-associated thyroid disruption, in that the associations between water fluoride concentration and fluoride intake and primary hypothyroidism were stronger among this group of women. It is possible that pregnant women with high TPOAb levels had a pre-existing autoimmune-related decreased capacity to produce thyroid hormones and therefore did not show as strong of a link with fluoride exposure compared to women with normal TPOAb levels. However, the estimates for women with normal TPOAb levels were less precise (i.e., wider confidence intervals) and should thus be interpreted with caution.

Our finding that women with primary hypothyroidism were more likely to have children with lower IQ scores is consistent with previous studies (Andersen et al., 2018; Haddow et al., 1999; Levie et al., 2018), though this association was only significant for FSIQ and VIQ among male children in our study. Few, if any, studies have explored effect modification by child sex when assessing the relationship between maternal hypothyroidism in pregnancy and offspring IQ. However, males born to women with hypothyroidism have been reported to be at increased risk of developing externalizing (e.g., attention deficit and hyperactivity disorder) (Peltier et al., 2021) problems when compared to females. In the context of neurotoxicants, the male brain is known to be more vulnerable to many chemical exposures, including fluoride, when compared with similarly exposed females (Green et al., 2020; Kern et al., 2017). Moreover, in recent studies, women who

were pregnant with males were found to be more likely to have elevated TSH (Sitoris et al., 2022; Wang et al., 2019). Collectively, these findings suggest that sex differences in the association between maternal hypothyroidism in pregnancy and child FSIQ and VIQ are plausible.

Despite the significant direct effect between water fluoride concentration and lower child FSIQ and observed trend toward lower child FSIQ among women with primary hypothyroidism ( $p = .06$ ), results from mediation analysis showed that maternal primary hypothyroidism did not significantly mediate the relationship between water fluoride concentration and child FSIQ. It is important to note that we were unable to account for all relevant covariates in the mediation model; study site was not included due to collinearity, and some of the covariates (e.g., HOME score) that are relevant for the association between hypothyroidism and child IQ are not directly relevant for the association between fluoride exposure and hypothyroidism. Moreover, considering only 25 of 107 (23.4 %) children of mothers with primary hypothyroidism had IQ data, there may have been insufficient statistical power in the mediation model to detect a significant indirect effect.

Among the sample of pregnant women included in this study, 6.1 % met criteria for primary hypothyroidism. Prevalence of primary or overt hypothyroidism has been shown to vary across other Canadian and American pregnant samples, generally falling between 0.7 and 3 % (Stagnaro-Green and Pearce, 2012; Leduc-Robert et al., 2020; Blatt et al., 2012). The relatively higher prevalence rate observed in the current study may be explained, in part, by the fact that women were categorized as primary hypothyroid if they met criteria based on their thyroid hormone levels in trimester one or if they had reported a previous diagnosis. Notably, women with prior diagnoses presented as euthyroid by blood panel in trimester one, and thus, would not normally be accounted for when determining prevalence rates based on thyroid hormone levels alone. This discrepancy may also be attributed to differences in diagnostic criteria used across studies (i.e., use of TSH on its own, with varying cut-offs) and time at thyroid hormone measurement since prevalence rates can vary across trimesters. Moreover, prevalence rates may differ depending on the demographic characteristics (e.g., race, age) of the sample (Stagnaro-Green and Pearce, 2012; Blatt et al., 2012).

#### 4.1. Strengths and limitations

Strengths of our study include the use of multiple measures of maternal fluoride exposure and thyroid hormones measured using gold-standard approaches in a large pregnancy cohort. In addition, our analyses considered an array of potential confounding variables and incorporated several factors that may influence thyroid hormone levels, including Tg, TPOAb levels, and pre-pregnancy BMI. There are some limitations associated with our study as well. Women in the MIREC cohort tend to be older, more educated, more likely to be married or common law, primarily White, and more likely to report prenatal vitamin use (Arbuckle et al., 2013) which may restrict the generalizability of our results to the broader Canadian population. Results pertaining to child IQ may also be restricted in generalizability, given the subsample of mother-child dyads with available IQ data were more likely to reside in non-fluoridated areas (54.0 % of the fluoride-IQ sample lived in a non-fluoridated area compared with 38.1 % in the fluoride-thyroid sample; Table S1). Regarding our measure of maternal race, our prior work conducted in the MIREC pregnancy cohort (Till et al., 2018), as well as in another Canadian population (Riddell et al., 2021), did not find differences in urinary-fluoride levels by race. We therefore do not have evidence of disproportionate exposure to fluoride by race, as reported in a study conducted in U.S. children (Martinez-Mier and Soto-Rojas, 2010). Still, other factors that could differ by race, such as diet (Brooks et al., 2017) and urinary pH (Ekstrand et al., 1982), could contribute to differences in fluoride excretion and therefore control for race is warranted for increasing the accuracy of the estimates. Reporting bias is also possible in that some women may not have self-reported a previous diagnosis of a thyroid disorder or taking thyroid medication. Further, as with any observational study, we cannot exclude the potential for residual confounding, whereby unmeasured or imprecisely measured confounders prevent

causal inferences from begin drawn from associations. Interpretation of findings should also consider the potential for reverse causality. We considered hypothyroidism as a potential mediator of the association between fluoride exposure and child IQ; however, it is conceivable that women with hypothyroidism may drink more water or other beverages given that hypothyroidism is associated with increased thirst. In this case, hypothyroidism would be associated with higher fluoride intake among women drinking fluoridated tap water and/or tea. The plausibility of reverse causality is unlikely, however, given that we would not expect more women with hypothyroidism to drink fluoridated over non-fluoridated water. An additional limitation is that fluoride was measured in spot samples instead of 24-hour urine samples or first morning voids, preventing us from being able to control for behaviours that could contribute to fluctuations in urinary fluoride concentration given the short half-life of fluoride (approximately 5 h). We attempted to mitigate the effects of this limitation by averaging urinary fluoride across all three trimesters. Finally, the questionnaire and methods used to estimate daily fluoride intake have not yet been validated; however, fluoride intake showed the expected associations with urinary fluoride and water fluoride concentrations, suggesting content validity of our derived variable.

#### 4.2. Conclusions

To our knowledge, this is the first study to investigate the relationships between maternal fluoride exposure and thyroid function in a prospective pregnancy cohort receiving optimally fluoridated water. Our findings indicate that higher levels of fluoride exposure in pregnant women were associated with increased risk of hypothyroidism, supporting our hypothesis that fluoride exposure may disrupt thyroid function. Thyroid dysfunction in pregnancy may be one mechanism underlying the previously found association between fluoride exposure in pregnancy and offspring FSIQ in the MIREC cohort (Green et al., 2019), particularly among women with male children, though further research is warranted. Our findings are of public health significance given the large number of people exposed to fluoride in drinking water and the vital role of thyroid hormones in neurodevelopment.

#### CRediT authorship contribution statement

**Meaghan Hall:** Conceptualization, Data curation, Formal analysis, Methodology, Software, Visualization, Writing – original draft, Writing – review & editing. **Bruce Lanphear:** Conceptualization, Funding acquisition, Investigation, Writing – review & editing. **Jonathan Chevrier:** Investigation, Methodology, Validation, Writing – review & editing. **Rick Hornung:** Methodology, Validation, Writing – review & editing. **Rivka Green:** Conceptualization, Methodology, Writing – review & editing. **Carly Goodman:** Methodology, Writing – review & editing. **Pierre Ayotte:** Funding acquisition, Investigation, Writing – review & editing. **Esperanza Angeles Martinez-Mier:** Investigation, Resources, Writing – review & editing. **R. Thomas Zoeller:** Investigation, Writing – review & editing. **Christine Till:** Conceptualization, Funding acquisition, Methodology, Project administration, Supervision, Writing – review & editing.

#### Data availability

The authors do not have permission to share data.

#### Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Rick Hornung reports financial support was provided by National Institute of Environmental Health Sciences. Christine Till reports a relationship with Health Research Board, Dublin that includes: consulting or advisory. Meaghan Hall reports a relationship with NIEHS that includes: travel reimbursement. Christine Till reports a relationship with National Institute of

Environmental Health Sciences that includes: travel reimbursement. Dr. Lanphear served as a non-retained expert witness in the federal fluoride case to describe the results of the fluoride studies using the MIREC cohort (Food & Water Watch, et al. vs. U.S. Environmental Protection Agency, United States District Court for the Northern District of California at San Francisco). He received no payment for his service.

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#### Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.scitotenv.2022.161149>.

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