THE REGIONAL MUNICIPALITY OF PEEL
COMMUNITY WATER FLUORIDATION COMMITTEE

AGENDA CWFC - 4/2016

DATE: Thursday, June 9, 2016
TIME: 8:30 AM – 9:30 AM
LOCATION: Regional Council Chamber, 5th Floor Regional Administrative Headquarters 10 Peel Centre Drive, Suite A Brampton, Ontario

MEMBERS: F. Dale; J. Downey; A. Groves; M. Palleschi; C. Parrish; K. Ras; J. Sprovieri; J. Tovey

1. DECLARATIONS OF CONFLICTS OF INTEREST

2. APPROVAL OF AGENDA

3. DELEGATIONS

4. REPORTS

4.1. Report on the Legislative Framework for the Authorization and Regulation of Community Water Fluoridation (For information)
     Presentation by Patrick O’Connor, Regional Solicitor and Director of Legal Services and Jeff Hennings, Acting Director, Water Division

4.2. Fluoridation of Drinking Water in the Region of Peel (For information)

5. COMMUNICATIONS

5.1. The Lancet Neurology, Two Articles Published in 2014, Regarding Water Fluoridation (Receipt recommended)

5.2. Dr. G. Richard Dundas, Bennington Oral Health Coalition (BOHC), Letter to the Editor of the Bennington Banner posted April 4, 2016, Regarding the Accomplishments of the BOHC (Receipt recommended)
5.3. **Maurice W. Smith, Resident**, Letter forwarded by the Town of Caledon, Regarding Water Fluoridation (Receipt recommendation)

5.4. **Kallie Miller, Registered Nurse**, Letter dated May 18, 2016, Regarding the Safety of Artificial Water Fluoridation Chemical (Receipt recommended)

5.5. **Richard L. Shames, Doctor of Medicine (MD)**, Email dated May 25, 2016, Regarding Artificial Water Fluoridation (Receipt recommended)

5.6. **Frank Dale, Regional Chair**, Letter dated May 28, 2016, to Eric Hoskins, Minister of Health and Long-Term Care, Regarding Provincial Policy on Water Fluoridation (Receipt recommended)

6. **IN CAMERA MATTERS**

7. **OTHER BUSINESS**

8. **NEXT MEETING**
   
   To be determined.

9. **ADJOURNMENT**
OBJECTIVE

The objective of this report is to provide an overview of the legislative framework that regulates the practice of community water fluoridation in the Province of Ontario.

REPORT HIGHLIGHTS

- The responsibility of providing safe drinking water and community water fluoridation is a shared responsibility between all levels of government.
- The Province of Ontario provides the principal legislation for community water fluoridation that both delegates responsibility to municipalities as well as regulates safe drinking water.
- The Fluoridation Act, R.S.O. 1990, c. F.22 provides the authority for municipalities to fluoridate the water supply, and delegates all decision making regarding community water fluoridation to municipalities.
- The Safe Drinking Water Act, 2002, S.O. 2002, c. 32 regulates municipal drinking water systems by ensuring water quality meets prescribed standards, operating systems comply with the Act and are kept in good state of repair, and regular water sampling, testing and monitoring requirements are completed and reported.
- Specific guidelines for the operation of drinking water systems are detailed for municipalities in several technical documents to ensure municipalities meet safety and quality drinking water standards.
- Health Canada provides the scientific rationale and technical expertise to establish the guideline for fluoride in drinking water.
- The Region of Peel is working under statutory authority as well as the provisions and guidelines of quality and technical standards to support oral health benefits while maintaining a safe municipal drinking water supply.

DISCUSSION

1. Background

In general, the responsibility of providing safe drinking water and community water fluoridation is shared between the federal, provincial/territorial, and municipal governments. However, the primary enabling legislation for community water fluoridation is provided at the Provincial level.
a) Provincial Level: Authority to Fluoridate and Ensuring Safety of the Water

The Province of Ontario has delegated the power to make a decision regarding community water fluoridation to municipalities through the Fluoridation Act, R.S.O. 1990, c. F.22.

The Act provides municipalities with the necessary authority to establish, continue, maintain and operate or discontinue a fluoridation system.

The Fluoridation Act and its predecessor legislation to provide the authority for municipalities to fluoridate the water supply dates back to 1957. Community water fluoridation had occurred before that time in some municipalities, however the express authority was granted by the Province following a 1957 court challenge brought to the Supreme Court of Canada (Toronto v. Forest Hill, [1957] S.C.R. 569) in which the City of Toronto was found to be lacking in legal authority to fluoridate the water system. The Fluoridation Act now provides the lack necessary authority.

Further regulation of fluoride in the water system is provided by the Safe Drinking Water Act, 2002, S.O. 2002. c. 32 (“SDWA”). One of the purposes of the SDWA is “to provide for the protection of human health and the prevention of drinking water health hazards through the control and regulation of drinking water systems and drinking water testing”. Accordingly, the Ministry of the Environment and Climate Change (“the Ministry”) is responsible for overseeing the regulation of safe drinking water in Ontario. Among other things, the SDWA requires that:

- All water provided by a municipal drinking water system must meet prescribed drinking water quality standards;
- The drinking water system be operated in accordance with the Act and regulations and be kept in a good state of repair;
- All sampling, testing and monitoring requirements be complied with; and
- All reporting requirements be complied with.

In accordance with the statutory scheme, Ontario Drinking Water Quality Standards have been established within Ontario Regulation 169/03. Schedule 2 of the Ontario Regulation 169/03 establishes a drinking water quality standard for fluoride expressed as a maximum concentration of 1.5 mg/L. Drinking water that contains fluoride at, or below, this maximum acceptable concentration does not pose a risk to human health according to Health Canada. Where fluoride is added to drinking water the Ministry, in its publication entitled “Technical Support Document for Ontario Drinking Water Standards, Objectives and Guidelines”, recommends that the concentration be adjusted to 0.5 to 0.8 mg/L. This is the range of concentrations that the Ministry has adopted as the optimum level for the prevention and control of tooth decay. Accordingly, the level of naturally-occurring fluoride in Peel’s lake based municipal water supply is adjusted to the optimal concentration range to protect against tooth decay. This process is dealt with in detail in the accompanying report of the Commissioner of Public Works dated May 17, 2016 titled “Fluoridation of Drinking Water in the Region of Peel”. Tests for fluoride concentration are performed regularly and in accordance with the regulations for drinking water systems provided for in Ontario Regulation 170/03. The Region’s drinking water system is also operated in a manner consistent with Ministry guidelines as articulated in the
Ministry’s publication entitled “Design Guidelines for Drinking-Water Systems 2008”. In particular, these guidelines specifically identify hydrofluosilicic acid as an acceptable source of fluoride for community water fluoridation.

b) Federal Level: Providing Scientific and Technical Expertise to Determine Safe Levels of Fluoride in Drinking Water

There is no direct federal regulation of community water fluoridation. However, Health Canada plays a major role in providing the scientific and technical basis for the drinking water standards that are implemented by the provinces and territories. Health Canada’s Federal-Provincial-Territorial Committee on Drinking Water (the “Committee”) has undertaken responsibility for the development of scientifically informed Guidelines for Canadian Drinking Water Quality and the protection of drinking water quality in collaboration with the Canadian Council of Ministers of the Environment. The Province of Ontario is represented on the Committee by a representative from the Ontario Ministry of the Environment and Climate Change.

With input from the Committee, Health Canada produces the publication entitled “Guidelines for Canadian Drinking Water Quality” and the related Guideline Technical Documents. Among these Guideline Technical Documents is a document concerning fluoride, the most recent version of which was published in 2010. The guidelines are based on current, published scientific research related to health effects, aesthetics (taste and odour), and operational considerations associated with drinking water supplies.

The process of formulating these guidelines includes internal reviews, external peer reviews, and public consultation through the Health Canada website. Following the period of public consultation, Health Canada convenes a panel of experts to provide advice and recommendations on the scientific studies and approaches that should be incorporated into the guidelines and technical documents.

These Health Canada publications provide the scientific rationale for the Ontario Drinking Water Quality Standards, and in particular the identification of the maximum acceptable concentration of fluoride as 1.5 mg/L. Drinking water that contains fluoride at, or below, this maximum acceptable concentration does not pose a risk to human health according to Health Canada. This maximum acceptable concentration has been established based on the segment of the population most at risk of developing dental fluorosis (i.e. children from 1-4 years of age).

The optimal concentration of fluoride in drinking water to promote dental health has been determined by Health Canada to be 0.7 mg/L. This concentration provides optimal dental health benefits and is well below the maximum acceptable concentration to ensure protection against potential adverse effects.

This recommended level also takes into account the other sources of fluoride to which Canadian consumers are commonly exposed such as food and drinks prepared or processed with fluoridated water, and toothpaste.
REPORT ON THE LEGISLATIVE FRAMEWORK FOR THE AUTHORIZATION AND REGULATION OF COMMUNITY WATER FLUORIDATION

CONCLUSION

Within the legislative framework there is statutory authority for fluoridation of the municipal water supply by the Region. The Region monitors water quality to ensure drinking water is safe for consumption based on all regulatory standards under the SDWA, while providing optimal dental health benefits.

Patrick O’Connor, Regional Solicitor

Approved for Submission:

D. Szwarc, Chief Administrative Officer

For further information regarding this report, please contact Patrick O’Connor, Regional Solicitor and Director, Legal Services, extension 4319, patrick.o’connor@peelregion.ca.
Fluoridation of Drinking Water in Peel

Community Water Fluoridation Committee
June 9, 2016

Patrick O’Connor, Regional Solicitor & Director, Legal Services
Jeff Hennings, Acting Director, Water Division
Justyna Burkiewicz, Supervisor, Water Quality & Compliance
Provincial Role

• Fluoridation Act

**Fluoridation systems**

2.1 (1) The council of a regional municipality may by by-law establish, maintain and operate or discontinue fluoridation systems. 2001, c. 25, s. 476 (3).
Provincial Role

• Safe Drinking Water Act, 2002

**Purposes**

1. The purposes of this Act are as follows:

   1. To recognize that the people of Ontario are entitled to expect their drinking water to be safe.

   2. To provide for the protection of human health and the prevention of drinking water health hazards through the control and regulation of drinking water systems and drinking water testing. 2002, c. 32, s. 1.
Provincial Role

• O. Reg. 169/03: Ontario Drinking Water Quality Standards

• O. Reg. 170/03: Drinking Water Systems
Federal Role

- *Guidelines for Canadian Drinking Water Quality: Guideline Technical Document – Fluoride*
Certification of Water Additives

- NSF/ ANSI 60
- Third party certification process
- Plant audit
Fluoride

• Meets MOECC regulatory standards
• Continuously monitored to maintain the optimal range as recommended within MOECC guidelines
• MOECC regulates and enforces drinking water products
• Legislative and operationally fluoride administered same as chlorine
Drinking Water Fluoride Additives

- Hydrofluorosilicic Acid (HFSA)
  - Phosphorite Rock
  - Calcium Fluoride
- Powder Forms
  - Sodium Fluorosilicate
  - Sodium Fluoride
- Pharmaceutical Grade Fluoride
Hydrofluorosilicic Acid

• Produced from Phosphorite Rock
• Most commonly used additive in North America
• Alternatively produced from Calcium Fluoride
• Others using Calcium Fluoride additive
## Residual Components

<table>
<thead>
<tr>
<th></th>
<th>Concentration in HFSA (from Phosphorite Rock)</th>
<th>Concentration in HFSA (from Calcium Fluoride)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arsenic</td>
<td>12.10</td>
<td>3.00</td>
</tr>
<tr>
<td>Lead</td>
<td>1.70</td>
<td>0.18</td>
</tr>
</tbody>
</table>

- Concentration in treated water is below detection limit.
Dry Powders

• Sodium Fluorosilicate and Sodium Fluoride
• NSF 60 approved
• Produced by further reacting HFSA, likely from phosphorite rock
# Cost of Fluoride Additives

<table>
<thead>
<tr>
<th></th>
<th>Annual Additive Costs</th>
<th>Annual Operating &amp; Maintenance Costs</th>
<th>Retrofit Capital Costs</th>
</tr>
</thead>
<tbody>
<tr>
<td>HFSA (Phosphorite Rock)</td>
<td>$321,000</td>
<td>$130,000</td>
<td>N/A</td>
</tr>
<tr>
<td>HFSA (Calcium Fluoride)</td>
<td>$364,000</td>
<td>$130,000</td>
<td>N/A</td>
</tr>
<tr>
<td>Sodium Fluorosilicate or Sodium Fluoride</td>
<td>$200,000</td>
<td>$250,000</td>
<td>$2,000,000</td>
</tr>
</tbody>
</table>
Pharmaceutical Grade Fluoride

- Not subject to NSF 60 standards; therefore would not be approved by MOECC for use in drinking water
- Difficult to obtain cost and quality data
Lakeview WTP Delivery
Lakeview Tanks and Pumps
Storage and Supply
Safe Work Environment
Monitor and Control
Thank you
DATE: May 26, 2016

REPORT TITLE: FLUORIDATION OF DRINKING WATER IN THE REGION OF PEEL

FROM: Dan Labrecque, Commissioner of Public Works

OBJECTIVE

To provide information on the Region of Peel's current fluoridation practices of the lake based drinking water system and to highlight alternative additives for use in the municipal water supply.

REPORT HIGHLIGHTS

- The level of naturally-occurring fluoride in Peel’s lake based municipal water supply is adjusted to an optimal concentration range to protect against tooth decay: 0.5 to 0.8 mg/L, as recommended by the Ministry of the Environment and Climate Change (MOECC)'s Technical Support Document for Ontario Drinking Water Standards, Objectives and Guidelines.
- For the 2015 reporting year, average fluoride levels at the Lakeview and Lorne Park Water Treatment Plants were 0.64 mg/L and 0.65 mg/L, respectively.
- The Region of Peel’s current fluoride additive, hydrofluorosilicic acid (HFSA), is a co-product collected during the refinement process following phosphate extraction from rocks and is subject to stringent standards, testing and certification for use in drinking water. Hydrofluorosilicic acid can also be manufactured from calcium fluoride mineral in the production of hydrogen fluoride.
- HFSA from both sources (calcium fluoride and calcium phosphate rock) is NSF certified and contains residual component (arsenic and lead) levels well below established guidelines. Fluoride derived from calcium fluoride, however, contains a lower level of residual components when in concentrated form than our current product.
- Dry product forms of fluoride additive are also available but would require a four to six month fluoridation disruption and an expensive retrofit to both water treatment plants.
- Pharmaceutical grade additives are available but are not NSF certified for drinking water, and therefore not permitted by the MOECC.

DISCUSSION

1. Background

The Region of Peel’s (the Region) lake based drinking water supply has been fluoridated for over 40 years, as part of the Region’s comprehensive strategy to protect oral health of Peel residents. Over the years, the process of fluoridation in the Region has been diligently
monitored and carried out in accordance with the regulatory framework for water fluoridation in Canada and Ontario.

Fluoride is an abundant naturally occurring ion of the element fluorine. Fluoride can be found in drinking water sources throughout the world. When naturally occurring fluoride in the source water is below the optimal level of 0.7 mg/L, as recommended by Health Canada, the fluoride level can be adjusted in drinking water supplies to provide optimal oral health benefits. Based on long-term routine tests conducted at the Region, the naturally occurring levels of fluoride in Lake Ontario range from 0.07 to 0.22 mg/L and to attain the optimal level, the fluoride level is adjusted in the lake based drinking water supply.

To date, the fluoridation additive used at the Region has been hydrofluorosilicic acid (HFSA). This product is also known by other names, including hydrofluosilicic acid, fluorosilicic acid and fluosilicic acid. HFSA is the most commonly used additive in communal water fluoridation. The fluoridation process consists of using HFSA to adjust the water’s fluoride content to the recommended level for the promotion of oral health. The concentration of fluoride in the Region’s drinking water is targeted at the operational range between 0.5 – 0.8 mg/L as recommended by the Ministry of the Environment and Climate Change (MOECC) Technical Support Document for Ontario Drinking Water Standards, Objectives and Guidelines. The optimal concentration of fluoride in drinking water, recommended by Health Canada, is 0.7 mg/L. The average level of fluoride in the South Peel drinking water system tested over the past few years has ranged from 0.53 to 0.68 mg/L.

HFSA, formula H₂SiF₆, achieves complete dissolution and ionic disassociation when added to water. Upon contact with water, it immediately breaks up into silicon (Si), hydrogen (H) and fluoride (F) ions, with no HFSA remaining at the end of the treatment process in the water leaving the treatment plant:

\[
H₂SiF₆ + 4H₂O \rightarrow 6H^+ + 6F^- + Si(OH)₄
\]

Fluoride ions that are the product of the HFSA dissolution are comparable to the ions found in the natural source water.

The operation and testing of water treatment systems in Ontario and fluoride levels in water are regulated by the MOECC under the Safe Drinking Water Act, 2002 (SDWA) and its regulations. The current guideline by Health Canada and the Ontario Drinking Water Quality Standards (Ontario Regulation 169/03) sets the maximum acceptable concentration for fluoride in drinking water at 1.5 mg/L. Through continuous monitoring of fluoride levels at the Region’s Lakeview and Lorne Park Water Treatment Plants to maintain the optimal target range, the fluoride concentration is found at levels within the operational target range, well below the maximum limit. For the 2015 reporting year, average fluoride levels were 0.64 mg/L and 0.65 mg/L at the Lakeview and Lorne Park Water Treatment Plants respectively. The Region’s fluoridation process complies with the federal and provincial standards for safe drinking water.

2. Findings

The Region currently fluoridates its lake based water supply using HFSA, which is an aqueous (water based) liquid additive obtained from Minchem, a reputable North American distributor, and manufactured by Solvay. Procurement of HFSA through our contracted
operator, the Ontario Clean Water Agency (OCWA) is under a three year contract that is scheduled to expire in 2018.

Fluoride is often found in the same rocks and minerals as phosphate and during the phosphate production process, separate collection of fluoride takes place. HFSA, our current fluoride additive, is a co-product collected during the refinement process following phosphate extraction from rocks and is subject to stringent standards, testing and certification for use in drinking water. The same rigorous standards apply to all drinking water additives, regardless of their source.

It is not uncommon for co-products or by-products of one industry to be used in products manufactured by a different industry. An example of this is the wide use of by-products of the oil industry in products such as personal care and beauty products, capsules for health supplements, toothpaste or food preservatives.

Regulatory Regimen

Through the SDWA, the MOECC regulates and enforces how additives used in drinking water systems in Ontario are handled, stored, added, monitored, controlled and reported. Through annual inspections, the MOECC ensures that drinking water systems that fluoridate do so in accordance with the Technical Support Document for Ontario Drinking Water Standards, Objectives and Guidelines, that the systems are designed in conformance to the Design Guidelines for Drinking Water Systems and that all additives that come in contact with drinking water meet the applicable standards set by the American Water Works Association (AWWA) and safety criteria standard NSF 60. The same standards apply for mandatory chemicals such as chlorine as for discretionary additives such as fluorides.

AWWA, a well-respected water supply industry association, sets standards for all additives used in the water treatment process. NSF International, an independent accredited global organization, tests, certifies products, and develops public health standards that help protect food, drinking water, consumer products and the environment. The NSF certification program includes unannounced audits of production and distribution facilities to certify that the products are properly formulated, packaged, and transported with appropriate safe guards in place to protect against potential contamination and to confirm they meet the requirements of the standards. It also includes testing and evaluation of each NSF certified product to confirm the absence of residual components at concentrations of concern; not to exceed 10 per cent of the allowable maximum contaminant level for the substance. For example, if the Maximum Acceptable Concentration of a residual component in drinking water is 10 mg/L, then the additive can’t increase the level by more than 1 mg/L.

The Region is mandated through its Municipal Drinking Water License, issued by the MOECC under the SDWA, to have all drinking water additives used at the Region’s water treatment plants, including fluoride, chlorine and coagulants meet the industry accepted quality standards: the AWWA standards, and the American National Standards Institute safety criteria standard NSF 60. In situations where the NSF approval of additives is difficult to obtain, food grade products, approved by the MOECC for use in the municipal drinking water systems may be used.

Subject to NSF 60 certification, HFSA supplied to the Region is tested for regulated metal compounds and other substances with an established maximum contaminant level. Each shipment of HFSA requires a NSF certification assuring compliance with the standard. With every HFSA delivery, a Certificate of Analysis is provided, which presents the breakdown of...
the residual components found within, reflective of the pure product. The lead and arsenic residuals sometimes found in HFSA are routinely tested for in the Region’s treated drinking water supply and found to consistently measure at concentrations below the legislated maximum acceptable concentrations, 0.010mg/L for lead and 0.025 mg/L for arsenic, in drinking water (Ontario Regulation 169/03).

<table>
<thead>
<tr>
<th>Residual Component</th>
<th>Concentration in HFSA (delivered) (mg/L)</th>
<th>Concentration added to Drinking Water when HFSA is 0.7 mg/L (mg/L)</th>
<th>Concentration in Treated Water (mg/L)</th>
<th>SDWA Limit (mg/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arsenic</td>
<td>12.10</td>
<td>&lt; 0.0010</td>
<td>0.025</td>
<td></td>
</tr>
<tr>
<td>Lead</td>
<td>1.70</td>
<td>0.000006</td>
<td>0.0059</td>
<td>0.010</td>
</tr>
</tbody>
</table>

Table 1. Comparing concentrations of residuals in HFSA and in drinking water in Peel.

All types of fluoride additives, regardless of their source or production process, contain trace levels of residual components in varying degrees. Fluoride addition to drinking water to achieve the target range of 0.5-0.8 mg/L, does not result in any significant increase in residual components in drinking water, which testing demonstrates remain at levels below method detection limit and well under the maximum acceptable concentration.

Health and Safety

Procedures are established specific to HFSA delivery to ensure product acceptability and to minimize possible hazards. HFSA deliveries are always supervised by qualified, MOECC certified water operators, who verify that the product is NSF 60 certified before accepting the shipment. Procedures on safe handling and storage of the product are also in place to ensure employee safety. The management of potential hazards includes review of HFSA Material Safety Data Sheets, familiarity with the Liquid Spill Contingency Plan, the Region’s Sewer Use By-law and other procedures for the safe handling of fluoride and other substances, as well as use of appropriate personal protective equipment. Standard health and safety protocols are carefully followed by trained operators throughout the entire water treatment process.

Appropriate equipment is in place to aid the operators in the safe handling of each product used in the Region’s water treatment facilities. The chemical storage tanks are lined with a corrosion-resistant material, and fluoride feed pumps and piping made of appropriate materials for the safe storage, containment and application of fluoride comply with the MOECC’s Design Guidelines for Drinking Water Systems. The design and maintenance of our drinking water systems, well documented procedures and highly skilled and trained staff help us maintain a good safety record with no lost time incidents in the last ten years related to fluoride handling.

Process Automation

To achieve the optimal fluoride levels with the use of HFSA, the Region utilizes a liquid fluoride feed system that is controlled through a computerized system overseen by water operators. The system is designed with several safeguards for the accurate control of...
fluoride addition and is monitored around the clock, 365 days per year. It automatically adjusts HFSA flow to achieve the correct fluoride concentration and maintain the desired levels. Fluoride concentration is monitored and measured by continuous analyzers and also sampled and tested manually twice daily. Testing equipment is maintained and calibrated regularly by qualified technicians.

In addition to fluoridation compounds, many other chemicals are routinely added to water throughout various stages of the treatment process. From the operational and legislative standpoint, fluoride is administered and monitored in the same way as chlorine, which is used at the treatment plants for primary and secondary disinfection of our water supplies. These two additives are governed by the same drinking water legislation and treated with the same due diligence.

3. Alternate Fluoride Additives

As mentioned above, our current source of HFSA is manufactured during the extraction of phosphate from calcium phosphate rock. Alternatives to our current source of HFSA and other additives are listed below.

**Hydrofluorosilicic Acid (from Calcium Fluoride)**

HFSA can also be manufactured from calcium fluoride mineral in the production of hydrogen fluoride. Like the HFSA made from phosphorite rock it is a commonly used additive to fluoridate water supplies, especially in large water plants, due to the ease of its use, transportation, the minimum need for different equipment and its cost effectiveness. Similar to the current product, this alternate product is liquid and hence easy to handle and to measure accurately when it is added to water. In addition, as noted in Table 2, HFSA derived from calcium fluoride presents a concentration of residual components at a lower level than the HFSA additive currently used in Peel.

<table>
<thead>
<tr>
<th>Residual Component</th>
<th>Concentration in HFSA (from Phosphorite Rock) (mg/L)</th>
<th>Concentration in HFSA (from Calcium Fluoride) (mg/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arsenic</td>
<td>12.10</td>
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</tr>
<tr>
<td>Lead</td>
<td>1.70</td>
<td>0.18</td>
</tr>
</tbody>
</table>

Table 2. Concentrations of residuals in the two different sources of HFSA.

A number of local municipalities have recently started using HFSA manufactured from calcium fluoride, including the cities of Toronto and Hamilton, as well as the Region of Durham.

**Sodium Fluorosilicate and Sodium Fluoride**

Two less frequently used additives are sodium fluorosilicate (NaSF) and sodium fluoride (NaF), both produced by neutralizing HFSA using sodium chloride (table salt) or caustic soda. Smaller water treatment plants tend to use these fluorides, which are transported and stored as a solid, dry product.

The quality standards for these fluorides, as with all products used in drinking water treatment processes, are set by NSF 60, which, as mentioned earlier, addresses the health
effects of treatment additives and sets a criterion that determines that fluorides are safe at their maximum use level with respect to potential residual components.

The use of powder additives introduces the need for specific health and safety protocols to protect water operators from exposure to fine powders of the dry product during its delivery, storage and handling. Since both the Lakeview and Lorne Park Water Treatment Plants currently use liquid HFSA, switching to a dry powder would require the retrofit of both plants to replace the large liquid storage tanks with storage silos, dust collectors, feed hoppers, mixing tanks, and a different fire protection system. Further investigation would be required to determine if a switch to a dry feed system could be achieved without a four to six month disruption to the fluoridation program. The estimated cost to retrofit the two plants is approximately $2,000,000, including engineering fees.

<table>
<thead>
<tr>
<th>Fluoride Additive</th>
<th>Annual Chemical Costs</th>
<th>Annual Operating &amp; Maintenance Costs</th>
<th>Retrofit Capital Costs</th>
</tr>
</thead>
<tbody>
<tr>
<td>HFSA (current source)</td>
<td>$321,000</td>
<td>$130,000</td>
<td>N/A</td>
</tr>
<tr>
<td>HFSA (alternate source)</td>
<td>$364,000</td>
<td>$130,000</td>
<td>N/A</td>
</tr>
<tr>
<td>Either Dry Powder</td>
<td>$200,000</td>
<td>$250,000</td>
<td>$2,000,000</td>
</tr>
</tbody>
</table>

Table 3. Cost comparison of the different fluoride additives.

Pharmaceutical Grade Fluoride

Pharmaceutical grade fluoride compounds fall under the Food and Drug Administration and are used in the formulation of prescription medications, not for use in water treatment. Unlike the fluoride additives described above, they are not subjected to the stringent NSF and AWWA standards and diligent testing for lead and arsenic. It is therefore not certain that pharmaceutical grade fluoride is a higher quality product with fewer residual components.

The NSF certification has been relied on by the drinking water industry and recognized by the MOECC for additives that are deemed safe for use in drinking water applications. The MOECC’s position is fixed regarding non-NSF certified fluoride additive being unsuitable for use in the drinking water systems in Ontario, which is supported by the regulatory approvals under the SDWA.

A comparison of the three types of industrial grade fluoride additives as well as information on the pharmaceutical grade of fluoride are included in Appendix I.
CONCLUSION

The Region’s current fluoridation practice meets all the legislated requirements mandated by the MOECC regulations and standards, and is managed in keeping with Health Canada’s guidelines. The additive used is tested and approved for use under this same regulatory framework.

Alternatives do exist, which range from the use of liquid form calcium fluoride-based HFSA to powder form Sodium based fluoride product. At the scale of the Region’s water treatment facilities, which are some of the largest in Canada, liquid additive is typically used for health and safety purposes, ease of handling and shipment, and cost.

Dan Labrecque, Commissioner of Public Works

Approved for Submission:

D. Szwarc, Chief Administrative Officer

APPENDICES:
Appendix I – Fluoride Additives

For further information regarding this report, please contact Andrew Farr, Executive Director Water and Wastewater Division at ext. 4761 or via email at andrew.farr@peelregion.ca.

Authored By: Jeff Hennings

Reviewed in workflow by:
Public Health
Legal Services
## Fluoride Additives

<table>
<thead>
<tr>
<th>Additive</th>
<th>Source Mineral/Rock</th>
<th>Description</th>
<th>NSF 60 Certification</th>
<th>Annual Cost</th>
<th>Jurisdictions using the Product</th>
</tr>
</thead>
</table>
| Hydrofluorosilicic Acid (HFSA) – water based  | Phosphorite Rock    | • Most fluoride additives used in North America are produced from phosphorite rock.  
• Phosphorite contains calcium phosphate mixed with limestone (calcium carbonates) minerals and apatite - a mineral with high phosphate and fluoride content. It is refluxed (heated) with sulfuric acid to produce a phosphoric acid-gypsum slurry (calcium sulfate-CaSO₄).  
• The heating process releases hydrogen fluoride (HF) and silicon tetrafluoride (SiF₄) gases, which are captured by vacuum evaporators. These gases are then condensed to a water-based solution of approximately 23% FSA. | Yes                  | $321,000                         | Peel Cornwall London Ottawa          |
| Hydrofluorosilicic Acid (HFSA) - water based  | Calcium Fluoride    | • Hydrogen fluoride is produced from fluorspar, the commercial name for the mineral fluorite (CaF₂).  
• The gases, mainly hydrogen fluoride, emerge from the end of a horizontal kiln, and are fractionally distilled in a column, termed the pre-scrubber. Solids and sulfuric acid are removed and the hydrogen fluoride vapour is purified to >99.9% purity by distillation in copper or steel vessels, condensed and stored in steel containers. | Yes                  | $364,000                         | Durham Hamilton Toronto              |
# Fluoridation of Drinking Water in the Region of Peel

## Appendix I

<table>
<thead>
<tr>
<th>Additive</th>
<th>Source Mineral/Rock</th>
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<th>NSF 60 Certification</th>
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<th>Jurisdictions using the Product</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium Fluorosilicate - dry additive</td>
<td>Most likely from Phosphorite Rock HFSA</td>
<td>• Made by neutralizing HFSA with sodium chloride or sodium sulfate.</td>
<td>Yes</td>
<td>$200,000</td>
<td>Denver Water Foothills WTP - a 300 million gallons / day facility.</td>
</tr>
<tr>
<td>Chemical Formula: Na2SiF6</td>
<td></td>
<td>• Similar in application to sodium fluoride.</td>
<td></td>
<td>$130,000 at Lakeview $70,000 at Lorne Park</td>
<td></td>
</tr>
<tr>
<td>Sodium Fluoride - dry additive</td>
<td>Most likely from Phosphorite Rock HFSA</td>
<td>• Sodium fluoride is either produced by neutralizing hydrofluoric acid with</td>
<td>Yes</td>
<td>$200,000</td>
<td>City of Merced, California. Population 80,000. 22 groundwater wells.</td>
</tr>
<tr>
<td>Chemical Formula: NaF</td>
<td></td>
<td>soda ash, or reacting sodium fluoro silicate with caustic soda or soda ash.</td>
<td></td>
<td>$130,000 at Lakeview $70,000 at Lorne Park</td>
<td></td>
</tr>
<tr>
<td>Pharmaceutical Grade Fluoride (usually comes in a powder form)</td>
<td>Most likely from Calcium Fluoride</td>
<td>• Generally, this type of fluoride containing powder is produced by the</td>
<td>NA</td>
<td>Unknown</td>
<td>Could not locate municipalities that use it, likely due to lack of NSF 60 certification.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>same method as exposing calcium fluoride to sulphuric acid, making hydrogen fluoride.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Subject to U.S. Pharmacopeia-National Formulary (USP 29 NF–24) acceptance criteria which do not include specific protection levels for individual contaminants such as arsenic and lead.</td>
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</table>
Neurobehavioural effects of developmental toxicity

Philippe Grandjean, Philip J Landrigan

Neurodevelopmental disabilities, including autism, attention-deficit hyperactivity disorder, dyslexia, and other cognitive impairments, affect millions of children worldwide, and some diagnoses seem to be increasing in frequency. Industrial chemicals that injure the developing brain are among the known causes for this rise in prevalence. In 2006, we did a systematic review and identified five industrial chemicals as developmental neurotoxics: lead, methylmercury, polychlorinated biphenyls, arsenic, and toluene. Since 2006, epidemiological studies have documented six additional developmental neurotoxins—manganese, fluoride, chlorpyrifos, dichlorodiphenyltrichloroethylene, and the polybrominated diphenyl ethers. We postulate that even more neurotoxicants remain undiscovered. To control the pandemic of developmental neurotoxicity, we propose a global prevention strategy. Untested chemicals should not be presumed to be safe to brain development, and chemicals in existing use and all new chemicals must therefore be tested for developmental neurotoxicity. To coordinate these efforts and to accelerate translation of science into prevention, we propose the urgent formation of a new international clearinghouse.

Introduction

Disorders of neurobehavioural development affect 10–15% of all births, and prevalence rates of autism spectrum disorder and attention-deficit hyperactivity disorder seem to be increasing worldwide. Subclinical decrements in brain function are even more common than these neurobehavioural developmental disorders. All these disabilities can have severe consequences—they diminish quality of life, reduce academic achievement, and disturb behaviour, with profound consequences for the welfare and productivity of entire societies.

The root causes of the present global pandemic of neurodevelopmental disorders are only partly understood. Although genetic factors have a role, they cannot explain recent increases in reported prevalence, and none of the genes discovered so far seem to be responsible for more than a small proportion of cases. Overall, genetic factors seem to account for no more than perhaps 30–40% of all cases of neurodevelopmental disorders. Thus, non-genetic, environmental exposures are involved in causation, in some cases probably by interacting with genetically inherited predispositions.

Strong evidence exists that industrial chemicals widely disseminated in the environment are important contributors to what we have called the global, silent pandemic of neurodevelopmental toxicity. The developing human brain is uniquely vulnerable to toxic chemical exposures, and major windows of developmental vulnerability occur in utero and during infancy and early childhood. During these sensitive life stages, chemicals can cause permanent brain injury at low levels of exposure that would have little or no adverse effect in an adult.

In 2006, we did a systematic review of the published clinical and epidemiological studies into the neurotoxicity of industrial chemicals, with a focus on developmental neurotoxicity. We identified five industrial chemicals that could be reliably classified as developmental neurotoxicants: lead, methylmercury, arsenic, polychlorinated biphenyls, and toluene. We also noted 201 chemicals that had been reported to cause injury to the nervous system in adults, mostly in connection with occupational exposures, poisoning incidents, or suicide attempts. Additionally, more than 1000 chemicals have been reported to be neurotoxic in animals in laboratory studies.

We noted that recognition of the risks of industrial chemicals to brain development has historically needed decades of research and scrutiny, as shown in the cases of lead and methylmercury. In most cases, discovery began with clinical diagnosis of poisoning in workers and episodes of high-dose exposure. More sophisticated epidemiological studies typically began only much later. Results from such studies documented developmental neurotoxicity at much lower exposure levels than had previously been thought to be safe. Thus, recognition of widespread subclinical toxicity often did not occur until decades after the initial evidence of neurotoxicity. A recurring theme was that early warnings of subclinical neurotoxicity were often ignored or even dismissed.

David P Rall, former Director of the US National Institute of Environmental Health Sciences, once noted that “if thalidomide had caused a ten-point loss of intelligence quotient (IQ) instead of obvious birth defects of the limbs, it would probably still be on the market”. Many industrial chemicals marketed at present probably cause IQ deficits of far fewer than ten points and have therefore eluded detection so far, but their combined effects could have enormous consequences.

In our 2006 review, we expressed concern that additional developmental neurotoxicants might lurk undiscovered among the 201 chemicals then known to be neurotoxic to adult human beings and among the many thousands of pesticides, solvents, and other industrial chemicals in widespread use that had never been tested for neurodevelopmental toxicity. Since our previous review, new data have emerged about the vulnerability of the developing brain and the neurotoxicity of industrial chemicals. Particularly important new evidence derives from prospective epidemiological birth cohort studies.

In this Review, we consider recent information about the developmental neurotoxicity of industrial chemicals
to update our previous report. Additionally, we propose strategies to counter this pandemic and to prevent the spread of neurological disease and disability in children worldwide.

Unique vulnerability of the developing brain
The fetus is not well protected against industrial chemicals. The placenta does not block the passage of many environmental toxicants from the maternal to the fetal circulation, and more than 200 foreign chemicals have been detected in umbilical cord blood. Additionally, many environmental chemicals are transferred to the infant through human breastmilk. During fetal life and early infancy, the blood–brain barrier provides only partial protection against the entry of chemicals into the CNS.

Moreover, the developing human brain is exceptionally sensitive to injury caused by toxic chemicals, and several developmental processes have been shown to be highly vulnerable to chemical toxicity. For example, in-vitro studies suggest that neural stem cells are very sensitive to neurotoxic substances such as methylmercury. Some pesticides inhibit cholinesterase function in the developing brain, thereby affecting the crucial regulatory role of acetylcholine before synapse formation. Early-life epigenetic changes are also known to affect subsequent gene expression in the brain. In summary, industrial chemicals known or suspected to be neurotoxic to adults are also likely to present risks to the developing brain.

Figure 1 shows the unique vulnerability of the brain during early life and indicates how developmental exposures to toxic chemicals are particularly likely to lead to functional deficits and disease later in life.

New findings about known hazards
Recent research on well-documented neurotoxicants has generated important new insights into the neuro-developmental consequences of early exposures to these industrial chemicals.

Joint analyses that gathered data for lead-associated IQ deficits from seven international studies support the conclusion that no safe level of exposure to lead exists. Cognitive deficits in adults who had previously shown lead-associated developmental delays at school age suggest that the effects of lead neurotoxicity are probably permanent. Brain imaging of young adults who had raised lead concentrations in their blood during childhood showed exposure-related decreases in brain volume. Lead exposure in early childhood is associated with reduced school performance and with delinquent behaviour later in life.

Developmental neurotoxicity due to methylmercury occurs at much lower exposures than the concentrations that affect adult brain function. Deficits at 7 years of age that were linked to low-level prenatal exposures to methylmercury were still detectable at the age of 14 years. Some common genetic polymorphisms seem to increase the vulnerability of the developing brain to methylmercury toxicity. Functional MRI scans of people exposed prenatally to excess amounts of methylmercury showed abnormally expanded activation of brain regions in response to sensory stimulation and motor tasks (figure 2). Because some adverse effects might be counterbalanced by essential fatty acids from seafood, statistical adjustment for maternal diet during pregnancy results in stronger methylmercury effects.

Prenatal and early postnatal exposures to inorganic arsenic from drinking water are associated with cognitive deficits that are apparent at school age. Infants who survived the Morinaga milk arsenic poisoning incident had highly raised risks of neurological disease during adult life.

The developmental neurotoxicity of polychlorinated biphenyls has been consolidated and strengthened by recent findings. Although little new information has been published about the developmental neurotoxicity of toluene, much has been learned about the developmental neurotoxicity of another common solvent, ethanol, through research on fetal alcohol exposure. Maternal consumption of alcohol during pregnancy, even in very small quantities, has been linked to a range of neurobehavioural adverse effects in offspring, including reduced IQ, impaired executive function and social judgment, delinquent behaviour, seizures, other neurological signs, and sensory problems.

Newly recognised developmental neurotoxicants
Prospective epidemiological birth cohort studies make it possible to measure maternal or fetal exposures in real time during pregnancy as these exposures actually occur, thus generating unbiased information about the degree and timing of prenatal exposures. Children in these prospective studies are followed longitudinally and assessed with age-appropriate tests to show delayed or deranged neurobehavioural development. These powerful epidemiological methods have enabled the discovery of additional developmental neurotoxicants.

Figure 1: Effect of neurotoxicants during early brain development
Exposures in early life to neurotoxic chemicals can cause a wide range of adverse effects on brain development and maturation that can manifest as functional impairments or disease at any point in the human lifespan, from early infancy to very old age.
Cross-sectional data from Bangladesh show that exposure to manganese from drinking water is associated with reduced mathematics achievement scores in school children. A study in Quebec, Canada, showed a strong correlation between manganese concentrations in hair and hyperactivity. School-aged children living near manganese mining and processing facilities have shown associations between airborne manganese concentrations and diminished intellectual function and with impaired motor skills and reduced olfactory function. These results are supported by experimental findings in mice.

A meta-analysis of 27 cross-sectional studies of children exposed to fluoride in drinking water, mainly from China, suggests an average IQ decrement of about seven points in children exposed to raised fluoride concentrations. Confounding from other substances seemed unlikely in most of these studies. Further characterisation of the dose–response association would be desirable.

The occupational health literature suggests that solvents can act as neurotoxins, but the identification of individual responsible compounds is hampered by the complexity of exposures. In a French cohort study of 3000 children, investigators linked maternal occupational solvent exposure during pregnancy to deficits in behavioural assessment at 2 years of age. The data showed dose-related increased risks for hyperactivity and aggressive behaviour. One in every five mothers in this cohort reported solvent exposures in common jobs, such as nurse or other hospital employee, chemist, cleaner, hairdresser, and beautician. In Massachusetts, USA, follow-up of a well-defined population with prenatal and early childhood exposure to the solvent tetrachloroethylene (also called perchlorethylene) in drinking water showed a tendency towards deficient neurological function and increased risk of psychiatric diagnoses.

Acute pesticide poisoning occurs frequently in children worldwide, and subclinical pesticide toxicity is also widespread. Clinical data suggest that acute pesticide poisoning during childhood might lead to lasting neurobehavioural deficits. Highly toxic and bio-accumulative pesticides are now banned in high-income nations, but are still used in many low-income and middle-income countries. In particular, the organochlorine compounds dichlorodiphenyltrichloroethane (DDT), its metabolite dichlorodiphenyldichloroethylene (DDE), and chlordecone (Kepone), tend to be highly persistent and remain widespread in the environment and in people's bodies in high-use regions. Recent studies have shown inverse correlations between serum concentrations of DDT or DDE (which indicate accumulated exposures), and neurodevelopmental performance.

Organophosphate pesticides are eliminated from the human body much more rapidly than are organochlorines, and exposure assessment is therefore inherently less precise. Nonetheless, three prospective epidemiological birth cohort studies provide new evidence that prenatal exposure to organophosphate pesticides can cause developmental neurotoxicity. In these studies, prenatal organophosphate exposure was assessed by measurement of maternal urinary excretion of pesticide metabolites during pregnancy. Dose-related correlations were recorded between maternal exposures to chlorpyrifos or other organophosphates and small head circumference at birth—which is an indication of slowed brain growth in utero—and with neurobehavioural deficits that have persisted to at least 7 years of age. In a subgroup study, MRI of the brain showed that prenatal chlorpyrifos exposure was associated with structural abnormalities that included thinning of the cerebral cortex.

Herbicides and fungicides might also have neurotoxic potential. Propoxur, a carbamate pesticide, and permethrin, a member of the pyrethroid class of pesticides, have recently been linked to neurodevelopmental deficits in children.

The group of compounds known as polychlorinated diphenyl ethers (PBDEs) are widely used as flame retardants and are structurally very similar to the polychlorinated biphenyls. Experimental evidence now suggests that the PBDEs might also be neurotoxic. Epidemiological studies in Europe and the USA have shown neurodevelopmental deficits in children with increased prenatal exposures to these compounds. Thus, the PBDEs should be regarded as hazards to human neurobehavioural development, although attribution of relative toxic potentials to individual PBDE congeners is not yet possible.

**Other suspected developmental neurotoxicants**

A serious difficulty that complicates many epidemiological studies of neurodevelopmental toxicity in children is the problem of mixed exposures. Most populations are exposed to more than one neurotoxicant at a time, and yet
most studies have only a finite amount of power and precision in exposure assessment to discern the possible effects of even single neurotoxicants. A further problem in many epidemiological studies of non-persistent toxicants is that imprecise assessment of exposure tends to obscure associations that might actually be present. Guidance from experimental neurotoxicity studies is therefore crucial. In the assessment of potential developmental neurotoxicants, we have used a strength of evidence approach similar to that used by the International Agency for Research on Cancer for assessing epidemiological and experimental studies.

Phthalates and bisphenol A are added to many different types of plastics, cosmetics, and other consumer products. Since they are eliminated rapidly in urine, exposure assessment is complicated, and such imprecision might lead to underestimation of the true risk of neurotoxicity. The best-documented effects of early-life exposure to phthalates are the consequence of disruption of endocrine signalling. Thus, prenatal exposures to phthalates have been linked to both neurodevelopmental deficits and to behavioural abnormalities characterised by shortened attention span and impaired social interactions. The neurobehavioural toxicity of these compounds seems to affect mainly boys and could therefore relate to endocrine disruption in the developing brain. In regard to bisphenol A, a prospective study showed that point estimates of exposure during gestation were linked to abnormalities in behaviour and executive function in children at 3 years of age.

Exposure to air pollution can cause neurodevelopmental delays and disorders of behavioural functions. Of the individual components of air pollution, carbon monoxide is a well-documented neurotoxicant, and indoor exposure to this substance has now been linked to deficient neurobehavioural performance in children. Less clear is the reported contribution of nitrogen oxides to neurodevelopmental deficits, since these compounds often co-occur with carbon monoxide as part of complex emissions. Tobacco smoke is a complex mixture of hundreds of chemical compounds and is now a well-documented cause of developmental neurotoxicity. Infants exposed prenatally to polycyclic aromatic hydrocarbons from traffic exhausts at 5 years of age showed greater cognitive impairment and lower IQ than those exposed to lower levels of these compounds.

Perfluorinated compounds, such as perfluorooctanoic acid and perfluorooctane sulphonate, are highly persistent in the environment and in the human body, and seem to be neurotoxic. Emerging epidemiological evidence suggests that these compounds might indeed impede neurobehavioural development.

### Developmental neurotoxicity and clinical neurology

Exposures in early life to developmental neurotoxicants are now being linked to specific clinical syndromes in children. For example, an increased risk of attention-deficit hyperactivity disorder has been linked to prenatal exposures to manganese, organophosphates, and phthalates. Phthalates have also been linked to behaviours that resemble components of autism spectrum disorder. Prenatal exposure to automotive air pollution in California, USA, has been linked to an increased risk for autism spectrum disorder.

The persistent decrements in intelligence documented in children, adolescents, and young adults exposed in early life to neurotoxicants could presage the development of neurodegenerative disease later in life. Thus, accumulated exposure to lead is associated with cognitive decline in the elderly. Manganese exposure may lead to parkinsonism, and experimental studies have reported Parkinson’s disease as a result of developmental exposures to the insecticide rotenone, the herbicides paraquat and maneb, and the solvent trichloroethylene. Any environmental exposure that increases the risk of neurodegenerative disorders in later life (figure I) requires urgent investigation as the world’s population continues to age.

### The expanding complement of neurotoxicants

In our 2006 review, we expressed concern that additional developmental neurotoxicants might lie undiscovered in the 201 chemicals that were then known to be neurotoxic to human adults, in the roughly 1000 chemicals known to be neurotoxic in animal species, and in the many thousands of industrial chemicals and pesticides that have never been tested for neurotoxicity. Exposure to neurotoxic chemicals is not rare, since almost half of the 201 known human neurotoxicants are regarded as high production volume chemicals.

Our updated literature review shows that since 2006 the list of recognised human neurotoxicants has expanded by 12 chemicals, from 202 (including ethanol) to 214 (table 1 and appendix)—that is, by about two substances per year. Many of these chemicals are widely used and disseminated extensively in the global environment. Of the newly identified neurodevelopmental toxicants, pesticides constitute the largest group, as was already the case in

<table>
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<th><strong>Number known since 2006</strong></th>
<th><strong>Number known before 2006</strong></th>
<th><strong>Number identified</strong></th>
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<tr>
<td>Metals and inorganic compounds</td>
<td>25</td>
<td>26</td>
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<tr>
<td>Organic solvents</td>
<td>30</td>
<td>40</td>
</tr>
<tr>
<td>Pesticides</td>
<td>92</td>
<td>101</td>
</tr>
<tr>
<td>Other organic compounds</td>
<td>46</td>
<td>47</td>
</tr>
<tr>
<td>Total</td>
<td>202*</td>
<td>214</td>
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</table>

*Including ethanol.

**Table 1: Industrial chemicals known to be toxic to the human nervous system in 2006 and 2013, according to chemical group**
2006. In the same 7-year period, the number of known developmental neurotoxicants has doubled from six to 12 (table 2). Although the pace of scientific discovery of new neurodevelopmental hazards is more rapid today than in the past, it is still slower than the identification of adult neurotoxicants.

The gap that exists between the number of substances known to be toxic to the adult brain and the smaller number known to be toxic to the much more vulnerable developing brain is unlikely to close in the near future. This discrepancy is attributable to the fact that toxicity to the adult brain is usually discovered as a result of acute poisoning incidents, typically with a clear and immediate association between causative exposure and adverse effects, as occurs for workplace exposures or suicide attempts. By contrast, the recognition of developmental neurotoxicity relies on two sets of evidence collected at two different points in time: exposure data (often obtained from the mother during pregnancy), and data for the child’s postnatal neurobehavioural development (often obtained 3–10 years later). Because brain functions develop sequentially, the full effects of early neurotoxic damage might not become apparent until school age or beyond. The most reliable evidence of developmental neurotoxicity is obtained through prospective studies that include real-time recording of information about exposure in early life followed by serial clinical assessments of the child. Such research is inherently slow and is hampered by the difficulty of reliable assessment of exposures to individual toxicants in complex mixtures.

### Consequences of developmental neurotoxicity

Developmental neurotoxicity causes brain damage that is too often untreatable and frequently permanent. The consequence of such brain damage is impaired CNS function that lasts a lifetime and might result in reduced intelligence, as expressed in terms of lost IQ points, or disruption in behaviour. A recent study compared the estimated total IQ losses from major paediatric causes and showed that the magnitude of losses attributable to lead, pesticides, and other neurotoxicants was in the same range as, or even greater than, the losses associated with medical events such as preterm birth, traumatic brain injury, brain tumours, and congenital heart disease (table 3). 98

Loss of cognitive skills reduces children’s academic and economic attainments and has substantial long-term economic effects on societies. 11 Thus, each loss of one IQ point has been estimated to decrease average lifetime earnings capacity by about €12 000 or US$18 000 in 2008 currencies. 4 Thus, each loss of one IQ point has been estimated to decrease average lifetime earnings capacity by about €12 000 or US$18 000 in 2008 currencies. 98 The most recent estimates from the USA indicate that the annual costs of childhood lead poisoning are about US$50 billion and that the annual costs of methylmercury toxicity are roughly US$5 billion. 98 In the European Union, methylmercury exposure is estimated to cause a loss of about 600 000 IQ points every year, corresponding to an annual economic loss of close to €10 billion. In France alone, lead exposure is associated with IQ losses that correspond to annual costs that might exceed €20 billion. 98 Since IQ losses represent only one aspect of developmental neurotoxicity, the total costs are surely even higher.

Evidence from worldwide sources indicates that average national IQ scores are associated with gross domestic product (GDP)—a correlation that might be causal in both directions. 10 Thus, poverty can cause low IQ, but the opposite is also true. In view of the widespread exposures to lead, pesticides, and other neurotoxicants in developing countries, where chemical controls might be ineffective compared with those in more developed countries, 109–110 developmental exposures to industrial chemicals could contribute substantially to the recorded correlation between IQ and GDP. If this theory is true, developing countries could take decades to emerge from poverty. Consequently, pollution abatement might then be delayed, and a vicious circle can result.

The antisocial behaviour, criminal behaviour, violence, and substance abuse that seem to result from early-life exposures to some neurotoxic chemicals result in increased needs for special educational services, institutionalisation, and even incarceration. In the USA, the murder rate fell sharply 20 years after the removal of lead from petrol, 110 a finding consistent with the idea that
exposure to lead in early life is a powerful determinant of behaviour decades later. Although poorly quantified, such behavioural and social consequences of neurodevelopmental toxicity are potentially very costly. 6

Prevention of developmental neurotoxicity caused by industrial chemicals is highly cost effective. A study that quantified the gains resulting from the phase-out of lead additives from petrol reported that in the USA alone, the introduction of lead-free petrol has generated an economic benefit of $200 billion in each annual birth cohort since 1980,103 an aggregate benefit in the past 30 years of over $3 trillion. This success has since been repeated in more than 150 countries, resulting in vast additional savings. Every US$1 spent to reduce lead hazards is estimated to produce a benefit of US$17–220, which represents a cost-benefit ratio that is even better than that for vaccines. 10 First, however, the costs associated with the late-life consequences of developmental neurotoxicity are enormous, and the benefits from prevention of degenerative brain disorders could be very substantial.

New methods to identify developmental neurotoxicants
New toxicological methods now allow a rational strategy for the identification of developmental neurotoxicants based on a multidisciplinary approach. 104 A new guideline has been approved as a standardised approach for the identification of developmental neurotoxicants. 105 However, completion of such tests is expensive and requires the use of many laboratory animals, and reliance on mammals for chemicals testing purposes needs to be reduced. 106 US governmental agencies have established the National Center for Computational Toxicology and an initiative—the Tox21 Program—to promote the evolution of toxicology from a mainly observational science to a predominantly predictive science. 107

In-vitro methods have now reached a level of predictive validity that means they can be applied to neurotoxicity testing. 108 Some of these tests are based on neural stem cells. Although these cell systems do not have a blood–brain barrier and particular metabolising enzymes, these approaches are highly promising. As a further option, data for protein links and protein–protein interactions can now be used to explore potential neurotoxicity in silico, 109 thus showing that existing computational methods might predict potential toxic effects. 110

In summary, use of the whole range of approaches along with clinical and epidemiological evidence, when available, should enable the integration of information for use in at least a tentative risk assessment. With these methods, we anticipate that the pace of scientific discovery in developmental neurotoxicology will accelerate further in the years ahead.

Conclusions and recommendations
The updated findings presented in this Review confirm and extend our 2006 conclusions. 1 During the 7 years since our previous report, the number of industrial chemicals recognised to be developmental neurotoxicants has doubled. Exposures to these industrial chemicals in the environment contribute to the pandemic of developmental neurotoxicity.

Two major obstacles impede efforts to control the global pandemic of developmental neurotoxicity. These barriers, which we noted in our previous review 6 and were recently underlined by the US National Research Council, 112 are: large gaps in the testing of chemicals for developmental neurotoxicity, which results in a paucity of systematic data to guide prevention; and the huge amount of proof needed for regulation. Thus, very few chemicals have been regulated as a result of developmental neurotoxicity.

The presumption that new chemicals and technologies are safe until proven otherwise is a fundamental problem. 113 Classic examples of new chemicals that were introduced because they conveyed certain benefits, but were later shown to cause great harm, include several neurotoxicants, asbestos, thalidomide, diethylstilboestrol, and the chlorofluorocarbons. 114 A recurring theme in each of these cases was that commercial introduction and wide dissemination of the chemicals preceded any systematic effort to assess potential toxicity. Particularly absent were advance efforts to study possible effects on children’s health or the potential of exposures in early life to disrupt early development. Similar challenges have been confronted in other public health disasters, such as those caused by tobacco smoking, alcohol use, and refined foods. These problems have been recently termed industrial epidemics. 115

To control the pandemic of developmental neurotoxicity, we propose a coordinated international strategy (panel). Mandatory and transparent assessment of evidence for neurotoxicity is the foundation of this strategy. Assessment of toxicity must be followed by governmental regulation and market intervention. Voluntary controls seem to be of little value. 116

Panel: Recommendations for an international clearinghouse on neurotoxicity
The main purpose of this agency would be to promote optimum brain health, not just avoidance of neurological disease, by inspiring, facilitating, and coordinating research and public policies that aim to protect brain development during the most sensitive life stages. The main efforts would aim to:

• Screen industrial chemicals present in human exposures for neurotoxic effects so that hazardous substances can be identified for tighter control
• Stimulate and coordinate new research to understand how toxic chemicals interfere with brain development and how best to prevent long-term dysfunctions and deficits
• Function as a clearinghouse for research data and strategies by gathering and assessing documentation about brain toxicity and stimulating international collaboration on research and prevention
• Promote policy development aimed at protecting vulnerable populations against chemicals that are toxic to the brain without needing unrealistic amounts of scientific proof
The three pillars of our proposed strategy are: legally mandated testing of existing industrial chemicals and pesticides already in commerce, with prioritisation of those with the most widespread use, and incorporation of new assessment technologies; legally mandated premarket evaluation of new chemicals before they enter markets, with use of precautionary approaches for chemical testing that recognise the unique vulnerability of the developing brain; and the formation of a new clearinghouse for neurotoxicity as a parallel to the International Agency for Research on Cancer. This new agency will assess industrial chemicals for developmental neurotoxicity with a precautionary approach that emphasises prevention and does not require absolute proof of toxicity. It will facilitate and coordinate epidemiological and toxicological studies and will lead the urgently needed global programmes for prevention.

These new approaches must reverse the dangerous presumption that new chemicals and technologies are safe until proven otherwise. They must also overcome the existing requirement to produce absolute proof of toxicity before action can be started to protect children against neurotoxic substances. Precautionary interpretation of data about developmental neurotoxicity should take into account the very large individual and societal costs that result from failure to act on available documentation to prevent disease in children.106 Academic research has often favoured scepticism and required extensive replication before acceptance of a hypothesis,107 thereby adding to the inertia in toxicology and environmental health research and the consequent disregard of many other potential neurotoxicants.108 Additionally, the strength of evidence that is needed to constitute “proof” should be analysed in a societal perspective, so that the implications of ignoring a developmental neurotoxicant and of failing to act on the basis of available data are also taken into account.

Finally, we emphasise that the total number of neurotoxic substances now recognised almost certainly represents an underestimate of the true number of developmental neurotoxicants that have been released into the global environment. Our very great concern is that children worldwide are being exposed to unrecognised toxic chemicals that are silently eroding intelligence, disrupting behaviours, truncating future achievements, and damaging societies, perhaps most seriously in developing countries. A new framework of action is needed.

Contributors
Both authors did the literature review, wrote and revised the report, and approved the final version.

Conflicts of interest
PG has provided paid expert testimony about mercury toxicology for the US Department of Justice. PJL has provided paid expert testimony in cases of childhood lead poisoning. We declare that we have no other conflicts of interest.

Acknowledgments
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Neurodevelopmental toxicity: still more questions than answers

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Julianna Gelinas, Myron Allukian Jr

The American Association for Community Dental Programs’ primary goal is to support the efforts of those serving the oral health needs of vulnerable populations at the community level. In view of our commitment to preventing oral diseases and improving access to services for the public, we read with interest Grandjean and Landrigan’s Review on neurobehavioral effects of developmental toxicity.1

In their Review, Grandjean and Landrigan claim that fluoride might cause neurodevelopmental harm, a claim based on only one paper,2 of which Grandjean is a coauthor. The study methodology contains several flaws that undermine its credibility and calls into question its applicability to the community water fluoridation programme in the USA.

The study2 is a meta-analysis of 27 cross-sectional studies done in poor, rural communities in China, Mongolia, and Iran, countries where the drinking water contains high levels of naturally occurring fluoride. The 27 original studies did not adequately control for a variety of intervening and confounding variables that could have affected intelligence quotient (IQ) scores, such as parents’ education and socioeconomic status and air and water pollution. It is unfortunate that Grandjean and Landrigan did not mention these limitations.

Additionally, they did not clearly state that the reference groups in their article2 use water fluoridated at about the recommended level. Thus, another interpretation of their analysis could be that communities fluoridated at the recommended level have a higher IQ.

No credible scientific studies show a relation between fluoride consumption and IQ levels; however, several have shown that fluoride ingested at recommended levels is not harmful. Grandjean and Landrigan did not acknowledge the animal study3 that showed no evidence of a neurotoxic effect of fluoride, even at levels up to 230 times the recommended concentration; an earlier study showing that fluoride causes no harm to children;4 two formal reviews that delineate weaknesses in the Chinese fluoride and IQ studies;5, 6 and the conclusion by one of these sets of investigators6 that biological plausibility for a link between fluoridated water and IQ has not been established.
Unfortunately, Grandjean and Landrigan's Review has been aggressively and improperly used by antifluoridationists to frighten the public about the effects of fluoridation, a well-established public health measure that has been shown to be cost-effective and safe. As a result, the public's oral health, especially that of the most vulnerable people, is put in jeopardy.

As advocates for better oral health and for serving the public's best interest, we are pleased that The Lancet Neurology is providing a forum for credible experts and organisations to reaffirm the safety and cost-effectiveness of fluoridation—a proven public health measure.

A statement from Grandjean and Landrigan clearly stating that their addition of fluoride to their list of neurotoxins does not apply to fluoridation at the recommended levels of 0.7–1.2 ppm would clarify our concerns on the misuse and misinterpretation of their paper.

We declare no competing interests.

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   - View in Article
   - | Summary
   - | Full Text
   - | Full Text PDF
   - | PubMed
   - | Scopus (163)

   - View in Article
   - | CrossRef
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Help sought to do more for teeth

I enjoyed reading Makayla McGeeney's fine article about primary care doctors and nurses promoting oral health by applying Fluoride Varnish. This is yet another program initiated and sponsored by the Bennington Oral Health Coalition, an organization of volunteers working to improve public health.

The situation is as follows. Fluoride is good for teeth. Bennington's community water is not fluoridated. Bennington's citizens have some of the worst dental problems in the entire state. Children's teeth need attention when they first erupt. Most local dentists don't see children until the child reaches age 3. Our local primary care physicians and pediatricians could fill the gap between the first tooth and age 3. If they apply Fluoride Varnish to these younger kids then the amount of childhood tooth decay might be reduced. We hope that the doctors can establish a Fluoride Varnish program in their offices.

This is not the only accomplishment of the BOHC. Over the last year we have held three community-wide forums to consider tactics to improve oral health. We worked with doctors and nurses to provide better education to pregnant women. We developed educational programs for schools and so far have presented in over 50 classrooms. We helped to start classroom toothbrushing programs at Bennington Elementary School. We sponsored classroom essay contests about the importance of teeth, with winners choosing a book from the Bennington Bookstore. We have given out thousands of Dental Goody Bags (toothbrush, floss, and toothpaste). We screened 520 kids at MAUMS for tooth decay (we found a lot of it).

We reestablished the long-unused dental chair at MAUMS and arranged to staff it with a dental hygienist and a supervising dentist. We have worked with the Select Board to establish the Oral Health Commission and with the SVSU Administration to look for a Tooth Tutor.

We are proud of our accomplishments but we are stretched thin. If you can help us financially or can volunteer, please call 447-3700. You could get more information about us on our Facebook page Bennington Oral Health.

— G. Richard Dundas, MD Bennington
PEEL REGION ORAL HEALTH COMMITTEE TO PUT CHILDREN’S DENTAL HEALTH AT RISK

“it is estimated that over 20% of Peel’s children under 15 live in poverty and 167,000 residents are living below the poverty line”. Ref. Knights Table website. Residents of Region of Peel elected Councillors with the expectation and understanding that they would address poverty, economic development, crime prevention and affordable housing as priorities. Instead, almost five years after Peel Region Councillors closed a lengthy debate on the merits of municipal water fluorination by unanimously voting to continue the longstanding practice, council members (Oral Health in Peel Committee) have taken it upon themselves to reopen the issue. Chair Carolyn Parrish emphasized gathering “scientific information” should be their focus before reaching any conclusions. Ref. Caledon Enterprise, February 18, 2016. Do we want Councillors addressing priorities or gathering “scientific information”? The question of who is qualified to make medical decisions that affect our children comes to mind.

There are only two possible outcomes that could come from the committee. One is the recommendation for no change to fluorination practices-highly unlikely from this committee. The other possibility (highly likely from this committee) is cessation of fluorination. “Water fluorination is the cheapest, most economical way to protect dental health in children—especially for families who can’t afford dental care” Ref. Denise Kokaram, Alex Dental Heath Bus, as reported by CBC on February 17, 2016. Why should unnecessary hardship be dumped on the children of Peel Region by a decision made by Oral Health in Peel Committee?

Interestingly, at the time members of the Region’s newly established Oral Health in Peel Committee formally met for the first time, National News carried the headline “Lack of fluoride in Calgary drinking water leads to rise in kids tooth decay, study finds”. The Region of Peel was the laughing stock of Canada over the formation of this committee. The study, published on February 17, 2016 in the journal Community Dentistry and Oral Epidemiology reported tooth decay for children in Grade 2 in Calgary climbed by an average of 3.8 tooth surfaces in the 2013-14 school year compared with the 2004-05 year, while increasing by 2.1 in Edmonton with fluorinated water.

To protect the children of Peel from unnecessary risk of increased tooth decay, The Oral Health in Peel Committee must be disbanded immediately because:

- The Committee circumvents the role of the Medical Officer of Health. It is an insult to that office and residents of Peel for Councillors to be wasting taxpayer’s money on a medical subject i.e. “gathering scientific information “when qualified people on the subject work for Region of Peel. How many of the Councillors are dentists?

- The Oral Health in Peel Committee is biased and in “Conflict of Interest” from the beginning! Brampton Councillor John Sproveiri, ardent fluorination critic is Vice Chairperson of the committee! "Brampton councillor John Sproveiri has been relentless in trying to force the Region to stop fluorination to the drinking water supply” Ref. Fluoride Alert.Org News. Is there any doubt of the forgone conclusion of this committee? Citizens of Peel Region do not deserve a “Kangaroo Court”!

- Accountability and Governance. Who made motion to form committee? Second? Terms of Reference for Committee? When will committee know its work is completed?
Process? Budget?

All the issues facing the Region of Peel, it is in the best interest of Residents that Councillors perform political process. For a committee to question the existing fluorination of drinking water puts children, especially those most vulnerable in society at unnecessary risk of increased tooth decay.

Maurice W. Smith
Maurice W. Smith, DVM PhD

Inglewood, Ontario
May 18, 2016

Re: Inquiry regarding safety of artificial water fluoridation chemical

Dear Dr. de Villa Medical Officer of Health,

I draw your attention to a recent article in the Brampton Guardian where you made the following statement.

"Peel health staff will not ‘cherry-pick’ studies to support water fluoridation: Medical Officer of Health" and then I heard about this tweet.

"From Dr. de Villa's twitter feed:

"Eileen de Villa Retweeted
David Juurlink @DavidJuurlink Feb 19

@picardonhealth I recently reviewed safety of fluoridation for a city council. 0.7 ppm is safe, full stop. Same for fluoridation agent HFSA"

I'm a retired Registered Nurse and have been researching the practice of artificial water fluoridation for a number of years and am shocked to learn that the practice continues without toxicology studies showing that the chemical in question, Hydrofluorosilicic Acid is safe for human consumption for short and long term use particularly for the sub groups who are at greater risk of harm such as unborn children, infants, renal and cancer patients, people with autoimmune disorders, hypothyroidism, diabetics, etc.

My neighbor is a Registered Nurse with a university degree and she works at a local Health Unit. She told me that it's the Health Unit's policy to tell new mothers to mix baby formula with fluoridated tap water as long as it's boiled for five minutes prior to use. I'm afraid she is terribly ill informed, because by boiling the water, it increases the concentration of fluoride and is extremely harmful to the infants developing brain according to a recent Harvard study which shows that fluoride lowers the IQ of infants up to 7 points. [http://fluoridealert.org/studies/brain01](http://fluoridealert.org/studies/brain01) Why are citizens not being warned of this?

Unfortunately, I've only been able to locate many scientific studies showing harmful effects regarding the consumption of Hydrofluorosilicic Acid on human health.

Dr. de Villa, you said that you recently researched the issue of 'safety' yourself. For that, I'm very grateful as it will relieve my worry somewhat.

Please refer me to the toxicology/scientific peer-reviewed studies showing that the toxic waste by-product, Hydrofluorosilicic Acid which is a known neurotoxin used to mass medicate the public is safe, for human consumption.

I've been searching this issue for a long time and haven't been able to locate the studies and when I asked my City Councillor for them - they are nowhere to be found. I draw your attention to a letter from Health Canada stating they do not have any such studies. I understand the

REFERRAL TO ______________________________
RECOMMENDED
DIRECTION REQUIRED _______________________
RECEIPT RECOMMENDED ✓
Concerned Citizens of Peel to End Fluoridation have provided the letter to the Region and to refresh your memory, I have attached a copy for you.

Please find attached, an extensive bibliography from a retired Professor, Dr. Roger Masters. Dr. Masters many studies on the chemical in question, shows beyond a doubt that there are many adverse harmful effects on human health when exposed to this toxic chemical.

I understand that Consumer Advocate, Erin Brockovich's signed letter to the Institute of Medicine, Environmentalist Lois Gibbs, a Nobel Prize Nominee and Civil Rights Leader Henry Rodrigues have all sent important information in order to inform the Water Fluoridation Committee, Regional Councillors and all staff which would include you; of the many dangers in using Hydrofluorosilicic Acid to fluoridate the municipal drinking water in Peel Region.

Most recently, Dr. Mark Hyman, a prestigious well known specialist in Functional Medicine has publicly announced his opposition to fluoridation and has asked the US government to undergo an investigation on this issue.

Many community safe water advocates like myself are supporting the pending lawsuit against Peel Region and the Province of Ontario as we trust the judicial system will call upon the required experts in the fields necessary to determine the legality, safety and efficacy of the chemical in question.

Thank you in advance for your consideration and I look forward to receiving the links to the toxicological studies showing that Hydrofluorosilic Acid used in the municipal drinking water is safe for human consumption.

Regards,

Kallie Miller, RN
May 26, 2014

Joanne David
<address snipped>
EDMONTON AB T6R 0B4

Dear Ms. David:

This is in response to your request under the Access to Information Act (the Act) for: **Clarified Request Text:**
Reports, studies, toxicology and clinical tests relating to hydrofluosilicic acid in Canadian tap water

**Original Request Text:**
Documents pertaining specifically to hydrofluosilicic acid in Alberta and Canadian tap water:
- Studies from 1940 showing dental efficacy and human safety.
- Studies from 1950s showing dental efficacy and human safety.
- Any double blind study done by Canada or any province showing dental efficacy and human safety, of any date.
- Any double blind study done by anywhere in the world that was considered.
- Any toxicity study, of any date, done by Canada or the world that was considered.
- Evidence of any kind (not opinion) that shows statistical viability of water fluoridation in terms of efficacy, and margin of error calculations.
- Evidence of any kind (not opinion) that shows statistical viability of water fluoridation in terms of human safety over a life-time, and margin of error calculations.
- Evidence of any kind (not opinion) that shows statistical viability of water fluoridation in terms of human safety, and margin of error calculations, for infants, young children, elderly, or any adult with disability, diabetes, bone disease, autism, thyroid ailments, kidney disease, etc.
- Evidence of any kind of consideration of human rights and medical ethics, namely our human right to opt out of the forced water fluoridation program, and if that consideration exists, why the overriding of these well-established medical standards are breached.

After a thorough search for the requested information, no records were located which respond to your request.

If you have any questions or concerns about the processing of your request, please do not hesitate to contact Nancy Armstrong, the analyst responsible for this request, either by phone at (613) 960-4457, or by fax at (613) 941-4541, or by e-mail at nancy.armstrong@hc-sc.gc.ca with reference to the file number cited above.
Please be advised that you are entitled to complain to the Office of the Information Commissioner of Canada concerning the processing of your request within 60 days of the receipt of this notice. In the event you decide to avail yourself of this right, your notice of complaint should be addressed to:

Office of the Information Commissioner of Canada  
30 Victoria Street  
Gatineau, Québec K1A 1H3

Yours sincerely,

[Signature]

Amanda Wilson  
Coordinator, Access to Information and Privacy Division
May 15, 2014

Joanne David  
<address snipped>  
EDMONTON AB T6R 0B4

Dear Ms. David:

This is to acknowledge receipt of your request made under the Access to Information Act (the Act) for the following information:

Clarified Request Text:
Reports, studies, toxicology and clinical tests relating to hydrofluosilicic acid in Canadian tap water

Original Request Text:
Documents pertaining specifically to hydrofluosilicic acid in Alberta and Canadian tap water:  
- Studies from 1940 showing dental efficacy and human safety.  
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- Any toxicity study, of any date, done by Canada or the world that was considered.  
- Evidence of any kind (not opinion) that shows statistical viability of water fluoridation in terms of efficacy, and margin of error calculations.  
- Evidence of any kind (not opinion) that shows statistical viability of water fluoridation in terms of human safety over a life-time, and margin of error calculations.  
- Evidence of any kind (not opinion) that shows statistical viability of water fluoridation in terms of human safety, and margin of error calculations, for infants, young children, elderly, or any adult with disability, diabetes, bone disease, autism, thyroid ailments, kidney disease, etc.  
- Evidence of any kind of consideration of human rights and medical ethics, namely our human right to opt out of the forced water fluoridation program, and if that consideration exists, why the overriding of these well-established medical standards are breached.

Should you require any information or wish to clarify any aspect of your request, please do not hesitate to contact Nancy Armstrong, the Request Coordinator responsible for this request, either by phone at (613) 960-4457, or by fax at (613) 941-4541, or by e-mail at nancy.armstrong@hc-sc.gc.ca with reference to the file number cited above. In the event that our program officials require clarification of your request in order to properly identify the relevant records, you will be contacted by us.
Please note that you may be required to pay additional fees in accordance with section 11 of the Act. The charges may cover search time in excess of five hours at a cost of $10.00 per hour and/or the cost of records reproduction calculated at $0.20 per page. You will be notified of any fees required.

Please be advised that you are entitled to complain to the Office of the Information Commissioner of Canada concerning the processing of your request within 60 days of the receipt of this notice. In the event you decide to avail yourself of this right, your notice of complaint should be addressed to:

Office of the Information Commissioner of Canada
30 Victoria Street
Gatineau, Québec K1A 1H3

Yours sincerely,

[Signature]

Amanda Wilson
Coordinator, Access to Information and Privacy Division
Publications on Silicofluorides, Neurotoxicity, and Behavior
Roger D. Masters & Myron J. Coplan

This bibliography was the collective work of the late Myron J. Coplan and Roger D. Masters, and is distributed in memory of Mike Coplan's pioneering work in the study of the harmful effects of water treated with either fluorosilicic acid or sodium silicofluoride.

I. Early Work by Other Authors


“In dilute aqueous solutions the hydrolysis of these two fluorine salts yielding fluoride ions is comparatively simple in the case of sodium fluoride, which is practically completely ionized, but quite complex and somewhat obscure in the case of sodium fluosilicate.” Following the specific chemical reactions “postulated” or suggested by chemists, McClure considers “the rate of retention and paths of excretion of fluorine” when ingested from these compounds, beginning by summarizing data in a 1935 study by Kick et al., who found that “there was no difference between sodium fluosilicate and sodium fluoride as regards the ultimate percent of fluorine retained in the rat’s body, i.e., the percent fluorine balance in the above data. There were some differences, however, in the paths of excretion, i.e., in urine or feces.” McClure’s replication confirms data on percentage of fluorine retained but does not challenge Kick’s finding of a difference in pathways of excretion (which is consistent with hypothesis of different biochemical side-effects from “residual species of silicate found by Westendorf).


The first sentence of this article confirms that, at the time of their approval in 1950, the extent of dissociation of silicofluorides injected in a water supply was unknown: "The widespread use of sodium silicofluoride in fluoridating drinking water has made it important to determine the state of the fluoride in such water, specifically, how much is fluoride ion, how much, if any, is unchanged silicofluoride, how much is fluoride bound to other ions. If all or nearly all of the fluoride is the ion F\textsuperscript{-}, the great body of information about the biologic effects of fluorides can be brought forward as a guarantee of safety. If considerable amounts of silicofluoride remain, a question can legitimately be raised since comparatively little work has been done on the biologic effects of silicofluorides." (p.192). Despite the authors’ claim to present (in 1957) “experimental results,” their analysis is essentially a theoretical extrapolation which does not provide a direct test of chemical and biochemical effects under conditions approximating actual usage. Moreover, the claim of safety is limited to the extent of dissociation of fluoride, ignoring issues of biological effects of “residual species” of silicates such as those found by Westendorf.


Silicofluorides are unlikely to dissociate completely under water plant conditions, producing only free fluoride and silicic acid.
without side reactions because the silicofluoride moiety \([\text{SiF}_6]^{2-}\) can react with \(\text{Al(OH)}_3\) to produce a number of derivative compounds. Moreover, silicofluoride residues may reassociate either within the stomach or in food preparation.


First publication on research more fully reported in Westendorf, 1975.


Experimental evidence showing that the extent of SiF dissociation into its component elements is at odds with the assumption that SiF and NaF are equivalent sources of free fluoride when used for water fluoridation. While the “residual species” of silicate remaining in water is not precise identified, the thesis confirms potentially harmful biological effects (acetylcholinesterase inhibition). These demonstrations of biochemical differences between silicofluorides and sodium fluoride have never been challenged with experimental data.


At a meeting in January 2001: “Several fluoride chemistry related research needs were identified including; (1) accurate and precise values for the stability constants of mixed fluorohydroxo complexes with aluminum (III), iron (III) and other metal cations likely to be found under drinking water conditions and (2) a kinetic model for the dissociation and hydrolysis (sic) of fluosilicates and stepwise equilibrium constants for the partial hydrolysis products. As a result of these discussions, ORD is exploring options to initiate research in the identified research areas.” (OFFICIAL CONFIRMATION THAT, WHEN APPROVED IN 1950, PRECISE CHEMISTRY AND BIOLOGICAL EFFECTS OF SILICOFLUORIDES WERE NOT FULLY KNOWN.)


“Substances Nominated to the NTP for Toxicological Studies and Recommendations Made by the ICCEC on April 17, 2002. **Table 1. -- Substances Recommended for Study**

<table>
<thead>
<tr>
<th>Substance [CAS No.]</th>
<th>Nominated by:</th>
<th>Nominated for:</th>
</tr>
</thead>
</table>

**Rationale for Nomination:** Primary agents used to fluoridate public drinking water systems; lack of toxicity information; assumed complete dissociation to free fluoride under normal conditions of use not supported by experimental evidence. **ICCEC Recommendations:** - Chemical characterization studies to assess chemical fate under aqueous conditions. -Toxicological studies may be considered when results of chemical characterization studies are available for review.”

Source: Above “information about substances nominated to the NTP for toxicology and carcinogenesis studies and the ICCEC's recommendations was published in This notice is available on the web (http://ntp-server.niehs.nih.gov/htdocs/Liason/ICCECFinal02JuneFR.html) along with
supporting documents for each nomination:(http://ntp-server.niehs.nih.gov/htdocs/liason/BkgrSum02June.html)...” TO OUR KNOWLEDGE, NO RESULTS FROM A STUDY IMPLEMENTING THIS NOMINATION HAVE BEEN PUBLISHED. NOTE ALSO: THE POSSIBILITY THAT SILICIC ACID RESIDUES MIGHT BOND TO ALUMINUM COULD RELATE TO CONDITIONS LIKE AUTISM AND ALZHEIMER’S DISEASE (WHOSE APPARENT INCREASE IN FREQUENCY MIGHT BE DUE TO ALUMINUM NEUROTOXICITY).


Cites work of Roger Masters and Myron Coplan, 1999a & 1999b.

Experimental demonstration that when rats are simultaneously exposed to both fluoride (from 150mg/L sodium fluoride in drinking water) and lead (from lead acetate), expression of Calcium/calmodulin-dependent protein kinase II (CaMKII) in hippocampus is significantly decreased. Since CaMKII is "a leading candidate in the search for the molecular basis of learning and memory," this effect is a plausible mechanism for harmful effects on educational performance associated with the combination of environmental exposures to lead and fluoride. Moreover, the hypothesized effects should be at least as great in communities with sources of exposure to lead (whether from industrial lead pollution, old housing with lead paint, or other sources of high lead in tapwater) and water treatment with silicofluorides (from which quantities of free fluoride are released). As a result, this study can be viewed as a model of a laboratory study of the effects of silicofluoride water treatment in American communities. While all behavioral findings are reinforced by this study, it is especially striking as an explanation for the Massachusetts data showing behavioral effects congruent with prediction of authors both in community frequencies of learning disabled students ["Neurotoxins, Disease, and Behavior," Fig. 12] and for scores on standardized MCAS tests in
seven different subjects and grades ["Lead, Brain Chemistry, and Educational Faillure," Fig. 1]).


Manganese (Mn) is essential for a variety of physiological processes, but at elevated levels, can be neurotoxic. While cognitive dysfunction has been recently appreciated to occur as a result of chronic Mn exposures, it is still unclear as to which cognitive domains are most susceptible to disruption by Mn exposure. We previously decried early appearing Mn-induced changes in performance on a paired associate learning task in monkeys chronically exposed to Mn and suggested that performance of this task might be a sensitive too for detecting cognitive dysfunction resulting from Mn exposure. As chronic Mn exposure has been suggested to be associated with attention, working memory and executive function deficits, the present study was conducted to assess the extent to which detrimental effects of chronic Mn exposure could be detected using tasks specifically designed to preferentially assess attention, working memory and executive function ..... These data suggest that in addition to the paired associate learning task, cognitive processing speed (as measured by the 5-CSRT) may be a sensitive measure of Mn toxicity and the brain circuits involved in performance of the SOSS task ["perform a self-ordered spatial search...task"] may be somewhat less sensitive to disruption by chronic Mn exposure.

Ellingsen, Dag G; Chashchin, Maxim; Bast-Pettersen, Rita; Zibarev, Evgenij; Thomassen, Yngvar; Chashhin, Valery (2015), "A follow-up study of neurobehavioral functions in welders exposed to manganese,," *NeuroToxicology* 47: 8-15.

Welders may be exposed to high amounts of manganese (Mn). In this study 63 welders and 65 referents were followed up with neurobehavioral tests approximately 6 years after the initial examination at baseline.... When subjects with sCDT above the upper reference limit of the laboratory (> /=1/7%) were excluded from the analyses, no difference in the decline in performance was observed between welders and referents for any of the applied neurobehavioral tests... Three welders had developed bradykinesia at follow-up, as assessed by a substantial decline in their Finger Tapping Test performance. They had also experienced a severe decline in Foot Tapping,
Grooved Pegboard and Postural Sway Test Scores (while blindfolded), while postural tremor as assessed with the CATSYS Tremor 7.0 was normal.

II. Publications co-authored by Roger Masters & Myron Coplan et al.

Legal implications of the evidence linking neurotoxicity and crime (including data from Toxic Release Inventory and crime for partial sample of US counties)

Survey of evidence linking lead and manganese neurotoxicity to aggressive behavior and crime, presenting multivariate analysis correlating Toxic Release Inventory for lead and manganese with crime data for 1991 from all 3141 US counties. Emphasizes effects of heavy metals on neurotransmitter function and behavior.

First published analysis of data linking silicofluoride treatment of public water supplies with higher uptake of lead, using survey of children’s blood lead in Massachusetts (by town).

Articulation of the linkages between neurotoxicity, brain chemistry, environmental pollution, and behavior (with focus on substance abuse and crime), using data from National Institute of Justice study of drug use in over 30,000 criminal offenders at time of arrest). Data show that where silicofluorides are in use, criminals are more likely to consume alcohol, more likely to have used cocaine at time of arrest – and that communities have significantly higher crime rates.

Analysis and rejoinder to letter dated 12 June 1999 from J. Charles Fox, Assistant Administrator, EPA, to Hon. Kenneth Calvert, U. S. House of Representative, commenting on errors and omissions in a "Question and Answer" statement and "Fluorosilicate Fact Sheet" enclosed by Mr. Fox. This document contains a preliminary review of scientific data on the differences between sodium fluoride (NaF) and the silicofluorides (H$_2$SiF$_6$ and Na$_2$SiF$_6$), with an emphasis on the complex production process and chemical interactions of the latter compounds.


Review of the evidence linking neurotoxicity and crime, using data from both county-level study (correlating EPA Toxic Release Inventory with FBI crime reports) and Massachusetts data on silicofluorides and lead uptake.


Summary of findings from our project


Follow-up epidemiological study of the association between silicofluoride treated community water and enhanced child blood lead parameters. This statistical study of 151,225 venous blood lead (VBL) tests taken from children ages 0-6 inclusive, living in 105 communities with populations from 15,000 to 75,000 in New York state, shows for every age and racial group a significant association between silicofluoride treated community water and elevated blood lead.

A survey of the scope of the emerging subfield called “biopolitics,” reflecting the activities of the membership of the Association for Politics and the Life Sciences. Four areas are discussed in some detail: 1). genetics and health; 2), toxins and behavior (including hyperactivity, depression, and violent crime), 3) the specific case of silicofluorides in water treatment and their effect in enhancing lead uptake; and 4) biopolitics and political theory.

Note: one-time e-print available at following URL:
http://polisci.annualreviews.org/cgi/content/full/4/1/345?ljkey=0K1GnNcUKf2Gq&keytype=ref&siteid=arjournals


Given data showing harmful side-effects of water treated with hydrofluorosilicic acid or sodium silicofluoride, a moratorium is desireable on continued use of silicofluorides until such time as they are shown to be safe (and contrary findings explained).


Survey of research on neurotoxicity, brain chemistry and behavior, including evidence of the role of lead and other heavy metal pollution and crime (as demonstrated by individual data, neurochemistry, and both geographic and longitudinal data} as well as survey of data linking silicofluorides to enhanced lead uptake. First presentation of findings on the extremely high correlation (r = .90) between gallons of leaded gasoline sold and the crime rates sixteen years later, confirming special vulnerability of pregnant mothers and newborns to lead toxicity.


Analysis of evidence of neurotransmitter dysfunction due to toxins associated with increased rates of violent crime, with extensive discussion of silicofluoride neurotoxicity as an important instance.
Masters Roger D. (2005). “A Moritorium on Silicofluoride Usage will Save
Estimation of rates of harmful effects of water treated with silicofluorides, based on national county-level data for violent crime and other statistics, and corresponding costs to taxpayers. Total financial benefits to taxpayers are in the Millions – and probably Billions – of dollars, with virtually no costs to the public.


Disinfection agents (chloramines as well as chlorine) have the effect of leaching lead from leaded-brass water fixtures, and this effect is significantly enhanced where fluoride compounds are also used to treat the water supply (with higher effects from long term combinations including fluorosilicic acid).

Confirmation of association between silicofluoride use in local water supplies and significant increase in absorption of lead from environmental sources, using new children's blood lead data from National Health and Nutrition Evaluation Survey III, counties of over 150,000 population. Review of important new findings, including lead leaching from brass water fixtures where systems combine use of silicofluorides with chloramine for disinfection; evidence confirming incomplete dissociation of silicofluorides (contradicting original "assumption" when silicofluorides were approved without testing by the Public Health Service in 1950); Westendorf's finding of acetylcholinesterase inhibition, interference with cholinergic function; and other evidence confirming research hypotheses and data explanations in our previous published research.

Analysis of high cost and disappointing outcomes of American health care system when compared to other industrialized countries. The U.S. has average per capita health costs over twice the OECD average, but a year less of life expectancy and over 1 additional infant death per 1,000 live births. This paradox is explained by the great expense of health care for the uninsured, which is a hidden cost associated with the low percent of American health costs that's supported by public funding (only 45.8%), compared to the average of 73% public health financing for all OECD countries. Preventive care (epitomized by reducing toxic exposure and uptake) has the potential to improve outcomes at low cost.


A study testing the hypothesis that a neuroscientific analysis of brain and behavior can explain hitherto unexplained local differences in rates of violent crime, to explore whether these behavior are significantly influenced both by local exposure to either fluorosilicic acid or sodium silicofluoride (together "SiF") -- toxic compounds used in place of sodium fluoride (NaF) in over 90% of fluoridated water in the U.S. Multivariate statistical analysis confirms the predicted association between SiF and violent crime as well as the hypothesis that "children in communities using SiF should have increased uptake of lead from environmental sources and higher rates of behavioral dysfunctions known to be caused by lead neurotoxicity."


Contrary to the prevailing gulf separating the social sciences from contemporary biology, this essay explores parallels between the Darwinian theoretical approach to human social behavior as a product of evolutionary biology and the tradition of "classical liberalism" from Locke to the present, based on "the core concept" of "freedom from the restraints of political, social and theological practices and power structures that block innovation in social behavior."
Kennedy, David; Seneff, Stephenie; Davidson, Robert M.; Haley, Boyd E.; & Masters, Roger D., "Environmental Toxicants and Infant Mortality," accepted pending acceptance of revision by ENTROPY (ISSN 2099-4300).

This article presents a highly innovative exploration of the relationship between "environmental toxicants" (i.e., toxins intentionally added to the environment) and the public policies of "water fluoridation" as factors contributing the relatively high rates of infant mortality in the U.S. My contribution explained the role of silicofluorides in the chemical process by which the aluminum adjuvants added to vaccines to increase their effectiveness bonds to fluoride (from water treatment) to form highly toxic aluminum fluoride ($\text{Al}_2\text{F}_3$). Given the relationship between aluminum fluoride and higher rates of many diseases (explored elsewhere in this volume), the ability to end the use of silicofluorides by listing these compounds under the Toxic Substances Control Act, §5-6 might make it possible to end this source of disease without a highly controversial attempt to end the use of aluminum adjuvants in vaccines.

**Articles in Preparation**

Masters, Roger D., "Neurotoxins, Disease, and Behavior," (in manuscript), Poster proposal for First Annual Neuroscience, Behavior and Health Research Forum, University of Vermont Medical School (Jan. 21-22, 2011).

Survey of wide variety of outcomes for which data provide evidence of harmful effects of toxins and their exacerbation by presence of water treated with silicofluorides.

Masters, Roger D. & Coplan, Myron J., (in manuscript), "Behavioral Effects of Water Toxicity: An Unexpected Problem in Experimental Methodology" (UNPUBLISHED)

John Crabbe, Jr.'s ambitious attempt to demonstrate the importance of the principle of replication in experimental behavioral neuroscience had an unintended consequence, which was viewed in as evidence that such scientific studies are in principle impossible to replicate. Crabbe conducted a series of behavioral experiments with 16 different strains of rats, taking great pains to insure that handling of animals and all experimental protocols were as identical as possible in three different Canadian cities. To his disappointment, rats in one of the three cities did not exhibit an aversion to entry in the dark arm of a "Y maze" -- which seemingly meant that the experiment failed to have reproducible effects. In fact, however, one of the "controls" was
the use of "tap water" for all animals. Of the three cities, only one -- Edmunton, Alberta -- treats its public water supplies with silicofluoride, and it was in that city that the rats failed to learn reliably and impulsively entered the dark arm of the maze more often than expected. Far from being a demonstration of non-reproducibility, therefore, Crabbe and his colleagues provided experimental evidence confirming our hypotheses on the neurotoxic effects of silicofluoride treated water. As a result, this study not only strengthens our findings; it also suggests a possible danger in reproducibility for laboratory studies if water treatment systems differ from one test to another.

Masters, Roger D.  (UNPUBLISHED). "Lead, Brain Chemistry, and Educational Failure"

Multivariate statistical analyses of outcome variables related to learning and educational systems are considered in the light of the established hypotheses in cognitive neuroscience and neurotoxicology. As a result, many negative outcomes attributed to poor teaching staff or racial inferiority illustrate the gene-environment interactions associated with impulse control and learning.

Roger D. Masters* & Myron J. Coplan**

Presentations to Scientific Conferences:

Report on findings of elevated blood lead associated with communities using silicofluoride, based on sample of over 250,000 children in Massachusetts (see Masters and Coplan, 1999a)


Review of evidence linking heavy metal pollution with substance abuse and crime, including presentation of data linking ban on sales of leaded gasoline with decline in crime 16 years later.
Summary of geographical data analyses contradicting the "null hypothesis" that there is no difference in the effects of sodium fluoride and the silicofluorides.


Preliminary report on data from analysis of national sample of over 4,000 children in NHANES III, showing that while water fluoridation is associated with a significant increase in children's blood lead (with especially strong effects among minority children), data on tooth decay from the same survey show limited benefits that are no longer evident among those aged 15-17.


Preliminary report on data from analysis of sample of blood lead testing of over 150,000 children in New York State communities of 15,000 to 75,000 population. Once again, average blood lead levels were significantly higher (p < .0001) in communities using silicofluorides in water treatment than in those with unfluoridated water. The effect was found independently in every age group for three ethnic subsamples


Analysis of bureaucratic opposition to reconsideration of public policy decisions challenged by new data on silicofluoride chemistry and its effects on human biology and behavior.


Design manual, removal of fluoride from drinking water supplies by activated alumina / by Frederick Rubel, Frederick, 1931-2010.

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The equilibrium fluoride capacity of activated alumina / Gurinderjit Singh and Dennis A. Clifford, Singh, Gurinderjit.

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Removal of fluorides from industrial wastewaters using activated alumina / by Irwin Frankel and Eric Frankel, I. F.

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Recovery of plutonium from sodium fluoride and alumina traps loaded with plutonium tetrafluoride by Gray, L. W.
Masters, Roger D
Dept. of Government
Dartmouth College
23311001226764  53 Lyme Rd, Hanover, NH 03755

Roger.D.Masters@Dartmouth.EDU
RETIRED PROFESSOR
Dear Regional Councilors, Water Fluoridation Committee Members, City Solicitors and City Staff - -

I am a Harvard graduate, former researcher at the U.S. National Institutes of Health (NIH), and now a practicing physician specializing in thyroid. Together with my wife, Karilee Halo Shames, R.N., Ph.D., I have authored several well-received books on thyroid health. Most recently, I helped put together a well-documented letter to the American Thyroid Association (ATA), urging them to oppose artificial community water fluoridation.

This letter briefly summarizes the extensive science of low-level fluoride's harmful impact on thyroid hormones, its interference with glucose and calcium metabolism in susceptible populations, and its general capacity for endocrine disruption. (One of the stated reasons why the entire country of Israel recently suspended all water fluoridation.)

Given your region's current conflict regarding fluoridation, I would like to submit that ATA letter for your review. I trust after reading it, you will agree with me that regardless of what little dental benefit fluoridation may or may not provide, the damage caused by this outdated practice can no longer be ignored. See: [http://www.ehcd.com/wp-content/uploads/2016/02/2016_02_11_ATALtrCWF.pdf](http://www.ehcd.com/wp-content/uploads/2016/02/2016_02_11_ATALtrCWF.pdf)

Sincerely,

Richard L. Shames, MD

[www.ThyroidPower.com](http://www.ThyroidPower.com)
To: American Thyroid Association
6066 Leesburg Pike
Suite 550
Falls Church, VA 22041

From:
February 11, 2016
Dear Vivian Cody and Warren Oliveri as Registered Agents and ATA Leadership,

We recognize the American Thyroid Association (ATA) as the world’s leading organization ‘devoted to thyroid biology and to the prevention and treatment of thyroid disease through excellence in research, clinical care, education, and public health.’ We appreciate the ATA values that include, ‘scientific inquiry, public service patient advocacy, education, and ethical conduct.’ We are writing because it is time for the ATA, in keeping with its mission and values, to openly advise the American public of what many physicians have been privately advising patients behind closed doors for decades, that drinking fluoridated water is harmful to thyroid health.

We suggest that February 2016 is the time to step forward for several reasons:

1. We believe it is unconscionable for this professional association to stand by silently while fluoridation advocates in the American Dental Association, American Medical Association and the American Academy of Pediatrics continue to aggressively market fluoridation as a dental panacea by distorting medical facts and denying recent scientific studies regarding endocrine disruption.

2. We believe that the political lobbying of fluoridationists who are increasingly attempting to mandate fluoridation at the state level, as they have done successfully in over a dozen states already, including California, Connecticut and Arkansas, will invariably increase the burden on those already ill with thyroid and other endocrine disorders, as well as increase their numbers substantially.

Antonio C. Bianco, President
Victor J. Bernet, COO
David H. Sarne, Treasurer
John C. Morris, III, President Elect
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James V. Hennessey
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Rebecca E. Schwegge
Susan A. Sherman
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February 11, 2016

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Communication to American Thyroid Association re fluoridation science, 11 Feb 2016
3. We believe that the examination by the National Toxicology Program (NTP) into the science indicating fluoridated waters are neurotoxic to fetuses and young children, although commendable, may be too limited. We know that exposure to fluoride lowers thyroid function, and that even subclinical hypothyroidism during pregnancy and childhood can and does result in lowered IQ, learning disabilities, and other psychomotor deficits, whether or not fluoride is characterized as “neurotoxic.” We suggest the ATA go on record with this medical fact sooner rather than later. Although the public comment period to the NTP on this topic closed January 8, 2016, we suspect the ATA will be able to provide a comment past that date.

4. We believe that the science published in 2014 and 2015 is sufficient on its own to recast artificial fluoridation as a public harm rather than as a public good. Consistent with science dating back a hundred years, recent science confirms that community water fluoridation is medically inadvisable for thyroid patients and most, if not all, of the population. We are attaching supporting resources to the end of this letter for your convenience.

We suggest that the cornerstone for an ATA public stand can be found in the 2015 report out of England that documents a significant increase in diagnosed cases of hypothyroidism in artificially fluoridated communities with a .7 ppm water concentration as compared to communities with .3 ppm naturally occurring fluoride (Peckham 2015) This finding is no surprise to us. Our medical community has known since the early 20th century that fluoride lowers thyroid function, and even prescribed fluoride tablets and baths as an effective treatment for hyperthyroidism. We also labeled a malaise seen in the 1950s during the early years of community fluoridation as “fluoride fatigue.” That term was subsequently replaced with the diagnosis of fibromyalgia or ME/CFS, conditions with no known etiology often accompanied by thyroid disorders as well as autoimmune and inflammatory diseases that are also linked to fluoride poisoning. (Galetti 1958; Laylander 1999a, 1999b; PFPC 1996; Waldbott 1978, 1998)

We find it very telling that, in stark contrast to our colleagues from the 1930s and 1940s, instead of regularly prescribing fluoride to lower thyroid function, modern endocrinologists regularly prescribe Synthroid to increase thyroid hormone levels. As you know, Synthroid is now one of the most common prescriptions in the United States. American fluoridation began in 1945.

The 2006 National Research Council (NRC) panelists who evaluated EPA fluoride contaminant levels (MCL/MCLG) in drinking water reviewed dozens of endocrine studies. They found the evidence of adverse thyroid impact to be among the most compelling (NRC, Chapter 8 and Appendix E). NRC panelist Dr. Kathleen Thiessen, who authored much of the endocrine disruption section of NRC report, filed a comment with the US EPA in 2011 and submitted an affidavit in 2014 for a pending lawsuit in Ontario, Canada that details the risk posed by artificial community water fluoridation to susceptible populations such as diabetics, cancer patients and those with low iodine levels.

Dr. Thiessen also has reminded the EPA and the Ontario courts that the 2006 NRC wrote, “Fluoride appears to have the potential to initiate or promote cancers, particularly of the bone.” (NRC, p 336) Additionally, the NRC reported that fluoride’s interference with calcium metabolism and in the presence of calcium deficiency has direct and indirect impacts on the functioning of the parathyroid, which in turn has an impact on the bones (NRC, 236-251). This should not be surprising, since Table 9 of a ten years later study of the first fluoridation trial revealed a number of notable differences on X-rays, such as children growing up in fluoridated Newburgh, NY had the twice cortical bone defects and twice the

Communication to American Thyroid Association re fluoridation science, 11 Feb 2016
exostoses compared to those found in the control population of un-fluoridated Kingston. Since we know that dental fluorosis is a poisoning of ameloblast mitochondria that results in a structural change to the composition of the tooth, it is not unreasonable to suspect a similar fluoride mediated etiology is at play in bones. We also know that the increased fluoride levels in tooth and bone may increase hardness and density, but reduce elasticity resulting in more brittleness. Some studies, as well as clinical reports, demonstrate that the 41% of children with dental fluorosis have more non-traumatic bone fractures than children without dental fluorosis correlated with the severity of their visible dental fluorosis. (Schlesinger 1956, Thiessen 2011, Beltran 2010, Alarcón-Herrera 2001)

Most disconcerting, the 2006 study by Bassin et al. published in Cancer Causes & Control identified an age-specific increased risk of osteosarcoma in boys drinking artificially fluoridated water in the US. This also should not be surprising, given our increasing awareness of the impact of environmental toxins on the genesis of cancers. In addition to being an endocrine disruptor, fluoride is a poison, an adjuvant and an inflammatory drug with an affinity for bone. What is surprising is that none of our professional organizations have clamored for follow-up on these alarming anomalies that suggest fluoride contributes to pediatric cancer, or called for a fluoridation moratorium as evidence of harm continues to mount.

The one other attempt to seriously investigate the connection between cancer and fluoride resulted in allegations of data tampering, harassment, and intimidation. In that instance, the accidental whistle blower, Senior Science Advisor Wm. Marcus of the EPA, was vindicated, reinstated and awarded back pay and legal fees. However, the falsified report that downgraded multiple cancerous tumors in thyroid, liver, kidney and bone to benign was allowed to stand. The matter of fluoridated water and cancer was dropped. Incredibly, the ADA partnering with the Centers for Disease Control (CDC) and American Academy of Pediatrics (AAP) returned to their mantra of “safe and effective” in their promotion of artificial water fluoridation that casts fluoride as an optimization of community water supplies with a naturally occurring mineral necessary to dental health. Municipal water fluoridation products are the contaminated waste products of industry, harvested from toxic slurry. (Marcus 1990, Dearen 2015, Mullenix 2014)

The ATA would not be the first medical association to break ranks with the ADA’s, AAP’s and AMA’s unequivocal support of fluoridation as a safe practice implemented for the public good. The National Kidney Foundation (NKF) removed their name from the list of fluoridation endorsers in 2008. As you know, inadequate kidney function results in a higher percentage of ingested fluoride being sequestered in the body where it can build up in soft tissues as well as bone, even causing calcification (Martin 2014, Waldbott 1978). The NKF took a neutral stand on the topic of fluoridation rather than opposing fluoridation, while officially recommending that those with Stage 4 kidney disease be advised to avoid fluoridated water and foods. It hasn’t been made clear whose responsibility it is to do the advising.

The NKF also advised that it would be “prudent” for children, those with renal impairment, and those with prolonged health conditions to “monitor” their fluoride intake, while acknowledging that they knew no way to do so since once fluoride is in the water it becomes ubiquitous in our diets and individual dosage is dependent on a myriad of factors. The NKF also implied that those who drink a lot of water should be concerned about their fluoride intake and its impact on their health. We can only assume that this weak stand was made in an effort to avoid angering the dental lobby who loudly insisted, then as now, that fluoridation prevents cavities and is perfectly safe for the general population. We suggest that the science since 2008 makes it easier for the ATA to take a stronger stand with firm footing in 2016.

Communication to American Thyroid Association re fluoridation science, 11 Feb 2016
In 2015, a US study found that even after adjusting for confounding factors such as socioeconomic status, fluoridated regions have between 67,000 and 131,000 more diagnosed cases of hyperactivity among school children than non fluoridated regions (Malin et al.) This is consistent with twenty years of science that include animal studies, ecologic studies, and studies that evaluated individuals that found exposure to fluoridated water as a fetus or during youth results in cognitive and emotional deficits correlated with severity of dental fluorosis in those individuals. Put more simply, in fluoridated communities, there are more tired moms and hyperactive special needs children, both explainable by fluoride’s impact on thyroid hormones. (See Resources)

Consistent with these findings is the presentation at 27th Conference of the International Society for Environmental Epidemiology (Aug 30- Sept 3, 2015) entitled “Evaluation of thyroid hormones (TSH and T4) in pregnant women exposed to fluoride (F-) in drinking water” by Rocha Amador D, et al. Using ATA guidelines, the team demonstrated F- toxicity on CNS during human pregnancy.

Several other 2015 studies, although not as specific to thyroid function, should also be of interest to the ATA. One found that even the low concentration of fluoride in “optimally” fluoridated drinking water causes inflammation of the immune system. Another found that the central nervous system has lymphatic/immune structures vulnerable to inflammation. A third identified a gene that predicts who will have a lower tolerance to fluoride and therefore exhibit both dental fluorosis and measurable neurodevelopmental deficits if exposed to fluoridated water in utero or during early childhood. (Resources)

Also in 2015, the Cochrane Review panel agreed with the 2000 York Review panel that the dental proclamations regarding fluoridation were overstated. Both international reviews of fluoridation literature found the low quality studies to be of high risk of bias with limited evidence of reduction in childhood cavities amounting to a lifetime benefit of perhaps one or two fewer cavities. The expert panels could not confirm that fluoridation reduced socioeconomic inequities among children or provided any benefit to adults in their reviews of the evidence. Moreover, both reviews confirmed that 12% of the general population living in artificially fluoridated communities would consider their fluoridation caused dental fluorosis ‘aesthetically displeasing.’ Finally, both reviews found there was neither any serious attempt to prove whole health safety nor evidence of safety. (Iheozor-Ejiofor et al. 2015, McDonagh et al. 2000)

For your convenience, we have included references to the York and Cochrane dental reviews together with a selection of relevant studies and reports in the Resources section of this communication. We suggest the ATA also carefully consider the three recent aggregate documents we’ve attached which include significant scientific citations:

i. The 2014 analysis by Prof. Rita Barnett-Rose JD on the legal and ethical implications of the current municipal water fluoridation practice

ii. The 2014 legal memo with attached scientific affidavit of NAS/NRC panelist Dr. Kathleen Thiessen prepared by Nader R. Hasan, Esq. in the Peel, Ontario lawsuit based on disproportionate harm

iii. The 2015 letter to the Institute of Medicine (IOM) signed by safe water consumer advocate Erin Brockovich, Dr. Wm. Ingram as president of the American Academy of Environmental Medicine on its behalf, “super lawyer” David P. Matthews of Matthews & Associates, and others regarding the failure of the IOM to update age specific fluoride dietary intake references published by the IOM in 1997 in light of 21st century science.

Communication to American Thyroid Association re fluoridation science, 11 Feb 2016
Moreover, corrosive fluoridation chemicals increase blood lead levels. (Coplan 2007, Maas 2007, Masters 2000)

The weight of the evidence is undeniable. We know fluoridation is unsafe. We’ve hundreds of studies and reports attesting to that fact. We know that thyroid and parathyroid diseases cause misery. We see that in our clinical practices. We know that fluoridation is mass medication without medical consent, and consequently an immoral act. We owe it to our patients, to the general public, and to ATA membership to exemplify medical integrity and scientific courage. The American Thyroid Association should be the spokesmen on thyroid health, not dentists or marketeers funded by the fluoride industry.

We are petitioning the American Thyroid Association to:

1. Publish a position statement opposing the practice of community water fluoridation (CWF) based on its impact on thyroid hormones, interference with glucose and calcium metabolism in susceptible populations, and general capacity for endocrine disruption.

2. Send a copy of that position statement to the National Toxicology Program in North Carolina with a cover letter that reminds them that the impact of hypothyroidism on the developing brain might not be scientifically categorized as neurotoxic but that medical fact is a distinction without a difference.

In closing, given the fluoridation lawsuit pending in Peel, Ontario based on the principle of disproportionate harm, i.e. an action that may have small benefit to some is not justified when that action poses a risk of great harm to others, and other anticipated American lawsuits yet to be filed, we suggest that the ATA leadership and directors should be prepared to demonstrate their scientific integrity and professional ethics. We suggest the ATA speak for themselves, as physicians and endocrinologists with specialities that range from nutrition to cancer, as to the interpretation of relevant scientific studies and testify on behalf of their patients as to the impact of fluoridation and endocrine disruption on thyroid health.

Respectfully,

Richard L. Shames, MD
Thyroid Physician, Health Author

William J. Rea, MD
Surgeon, Founder EHC-D, Author

Nathan Becker, MD, FACE, FACP
Endocrinologist, member AACE

Heather Dawn Gingerich, MSc.
Medical Geologist, AAAS, UNESCO

Eleanor V. Phillips, DDS
former ADA & AAPD, Health Author

Marcus L. Scott, Esq.
Attorney at Law

“Long-continued ingestion of minute quantities of fluorine causes disease of the thyroid gland.” - Douglas D. Styne, MD

“There’s no doubt that the intake of fluoridated water is going to interrupt basic functions of nerve cells in the brain, and this is certainly not going to be [for] the benefit of anybody.” Dr. Robert Isaacson, 2006 NRC

“‘It is reckless to assume that when fluoride is damaging the baby’s growing tooth cells, it is not damaging other delicate tissues like the bone, brain and endocrine system.’” – Dr. Paul Connett, Professor Emeritus of Chemistry (2015)

CC: Dr. Sanjay Gupta at CNN, Dr. Stephen Peckham, Moms Against Fluoridation, Fluoride Action Network
Prepared by: KS Spencer
Communication to American Thyroid Association re fluoridation science, 11 Feb 2016
RESOURCES
2014-2015 Aggregations:

   • Excerpt: The cessation of all compulsory water fluoridation schemes should be the goal of all public health agencies, ethical lawmakers, and informed citizens.

   • Excerpt: Marginal benefit in exchange for significant risk is the sine qua non of gross disproportionality… the stronger the scientific evidence of risk of harm, the greater the gross disproportionality.


Summary:
- Fluoride is an enzyme poison and an endocrine disruptor
- Fluoride is a potent adjuvant… causing or worsening allergies
- Fluoride is a proliferative agent… causing or worsening inflammation
- Fluoride accumulates in bones and tissue… causing or worsening arthritis and other ailments
- Fluoride impacts thyroid hormones… resulting in both hypo and hyper disorders
- Fluoride interferes with glucose metabolism… a concern for diabetics
- Fluoride causes dental fluorosis… disproportionately by race and social economic status
- Fluoride is neurotoxic to fetuses, infants and young children… resulting in permanent deficits
- Fluoride is a burden to kidneys… resulting in increased fluoride retention and possible renal damage in those with kidney disease.

Selected 2014-2015 Studies and Reports:


Earlier References & Resources:


19. Hsien-Wen Kuo, Chuan-Juan Lin, Li-Li Chen. Factors Affecting Urinary Fluoride Concentrations Among Patients With Renal Dysfunction. Institute of Environmental Health, and Department of Nursing, China Medical College; Department of Nursing, Hung-Kuang Technology College, Taichung, Taiwan, R.O.C. 2001. http://ir.cmu.edu.tw/ir/bitstream/310903500/1332/1/2001067481.pdf


Office of the Chair

REFERRAL TO ______________________________
RECOMMENDED
DIRECTION REQUIRED _______________________
RECEIPT RECOMMENDED ✓

May 28, 2016

Honourable Dr. Eric Hoskins
Minister of Health and Long-Term Care
Hepburn Block, Queen’s Park
10th Floor
Grosvenor Street
Toronto, Ontario
M7A 2C4

May 28, 2016

Dear Minister:

Re: Provincial Policy Regarding Water Fluoridation

You will be aware that the issue regarding the merits of retaining fluoride in municipal drinking water systems has come under renewed debate. You may also be aware that Peel Region Council has established a committee, and undertaken a process, to study and debate this issue. To date, the committee members have heard from various subject matter experts and continue with the process of information gathering.

It is not my intention to request that you be interjected into our deliberations. The position of the Ministry of Health and Long-Term Care (MHLTC) is well known to us. A recent review of your ministry’s website informs us that the efforts of the Chief Medical Officer of Health to promote the continued use of fluoride in water, and discourage any fears related to negative health effects, continue.

However, what may be the case, based on some information we are hearing, is that the province may be considering going beyond the exclusive use of persuasion, regarding the continued use of fluoridation, and is considering some type of legislation that could make it difficult for municipalities to decide to no longer fluoridate their drinking water.

The Regional Municipality of Peel
10 Peel Centre Dr, Suite A, Brampton, ON L6T 4B9 905-791-7800 Fax 905-791-2567
Website: www.peelregion.ca
This by no means is an attempt by me to influence your decision making. At Peel, we have the variation of views, with committee members acknowledging that effective arguments are made by experts expressing alternate perspectives.

However, you will appreciate that should Ontario be considering implementing a province-wide directive mandating its preferred policy, that would have an obvious impact on our current deliberative process.

Therefore, I would be most appreciative if we could be made aware of the most up to date position of the MHLTC regarding water fluoridation and be advised with respect to any steps that are being considered which could impact municipal decision-making in this regard.

In the event, you have any questions, or require additional information, please feel free to contact my office directly. I look forward to hearing from you.

Sincerely,

Frank Dale
Chair, Region of Peel