

111 (B)

## Sprovieri, John Councillor

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**From:** Sprovieri, John Councillor  
**Sent:** 2017/05/08 4:48 PM  
**To:** Sprovieri, John Councillor  
**Subject:** FW:

Hello Councillor,

Please find the article attached.

*"Now, a report from the world's oldest and arguably one of its most prestigious medical journals, The Lancet, has officially classified fluoride as a neurotoxin — in the same category as arsenic, lead and mercury. The news was broken by author Stefan Smyle, who cited a report published in The Lancet Neurology, Volume 13, Issue 3, in the March 2014 edition, by authors Dr. Phillippe Grandjean and Philip J. Landrigan, MD. The report, which was officially released in 2014 and published in the journal."*

Thank-you,

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## Neurobehavioural effects of developmental toxicity

Philippe Grandjean, Philip J Landrigan

*Lancet Neurol* 2014; 13: 330–38

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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 neurotoxicants: lead, methylmercury, polychlorinated biphenyls, arsenic, and toluene. Since 2006, epidemiological studies have documented six additional developmental neurotoxicants—manganese, fluoride, chlorpyrifos, dichlorodiphenyltrichloroethane, tetrachloroethylene, 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,<sup>1</sup> and prevalence rates of autism spectrum disorder and attention-deficit hyperactivity disorder seem to be increasing worldwide.<sup>2</sup> Subclinical decrements in brain function are even more common than these neurobehavioural developmental disorders. All these disabilities can have severe consequences<sup>3</sup>—they diminish quality of life, reduce academic achievement, and disturb behaviour, with profound consequences for the welfare and productivity of entire societies.<sup>4</sup>

The root causes of the present global pandemic of neurodevelopmental disorders are only partly understood. Although genetic factors have a role,<sup>5</sup> 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.<sup>5</sup> 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.<sup>6,7</sup> 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.<sup>8</sup> 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.<sup>6</sup> 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.<sup>9,10</sup> 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.<sup>11</sup> 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”.<sup>12</sup> 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,<sup>6</sup> 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



## Building a Database of Developmental Neurotoxicants: Evidence from Human and Animal Studies

W. Mundy<sup>1</sup>, S. Padilla<sup>1</sup>, T. Shafer<sup>1</sup>, M. Gilbert<sup>1</sup>, J. Breier<sup>1,2</sup>, J. Cowden<sup>1</sup>, K. Crofton<sup>1</sup>, D. Herr<sup>1</sup>, K. Jensen<sup>1</sup>, K. Raffaele<sup>3</sup>, N. Radio<sup>4</sup>, and K. Schumacher<sup>5</sup>.

<sup>1</sup>Neurotoxicology Div. U.S. EPA, RTP, NC 27711; <sup>2</sup>Curriculum in Toxicology, Univ. of N.C. at Chapel Hill, Chapel Hill, NC, 27514; <sup>3</sup>NCEA/ORD, U.S. EPA, Washington, DC, 20460; <sup>4</sup>Cellumen, Inc., Pittsburgh, PA. 15238; <sup>5</sup>U.S. EPA, Region 7, Kansas City, KS, 66101.

### Chemicals with Substantial Evidence of Developmental Neurotoxicity (n≈100)

2-Ethoxyethyl Acetate  
Acibenzolar-S-methyl  
Acrylamide  
Aldicarb  
Allethrin  
Aluminum (cl or lactate)  
Amino-nicotinamide(6-)  
Aminopterin  
Amphetamine(d-)  
Arsenic  
Aspartame  
Azacytidine(5-)  
Benomyl  
Benzene  
Bioallethrin  
Bis(tri-n-butyltin)oxide  
Bisphenol A  
Bromodeoxyuridine(5-)  
Butylated Hydroxy Anisol  
Butylated hydroxytoluene  
Cadmium  
Caffeine  
Carbamazepine  
Carbaryl  
Carbon monoxide  
Chlordecone  
Chlordiazepoxide  
Chlorine dioxide  
Chlorpromazine  
Chlorpyrifos  
Cocaine  
Colcemid  
Colchicine  
Cypermethrin  
Dexamethasone  
Diamorphine hydrochloride

Diazepam  
Cytosine Arabinoside  
DEET  
Deltamethrin  
Diazinon  
Dieldrin  
Diethylstilbestrol  
Diphenylhydantoin  
Epidermal Growth Factor  
Ethanol  
Ethylene thiourea  
Flourouracil(5-)  
Fluazinam  
Fluoride  
Griseofulvin  
Haloperidol  
Halothane  
Heptachlor  
Hexachlorobenzene  
Hexachlorophene  
Hydroxyurea  
Imminodipropionitrile (IDPN)  
Ketamine  
Lead  
Lindane  
LSD  
Maneb  
Medroxyprogesterone  
Mepivacaine  
Methadone  
Methanol  
Methimazole  
Methylparathion  
Monosodium Glutamate  
MPTP  
Naloxone

Naltrexone  
Nicotine  
Methoxyethanol, 2-  
Methylazoxymethanol  
Methylmercury  
Ozone  
Paraquat  
Parathion (ethyl)  
PBDEs  
PCBs (generic)  
Penicillamine  
Permethrin  
Phenylacetate  
Phenylalanine (d,l)  
Phthalate, di-(2-ethylhexyl)  
Propylthiouracil  
Retinoids/vit.A/isotretinoin  
Salicylate  
Tebuconazole  
Tellurium (salts)  
Terbutaline  
Thalidomide  
THC  
Toluene  
Triamcinolone  
Tributyltin chloride  
Trichlorfon  
Trichloroethylene  
Triethyllead  
Triethyltin  
Trimethyltin  
Trypan blue  
Urethane  
Valproate  
Vincristine



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[Justice Laws Website \(http://laws-lois.justice.gc.ca\)](http://laws-lois.justice.gc.ca)

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→ [S.C. \(Statutes of Canada\) 1999, c. 33](#)

**Canadian Environmental Protection Act, 1999 (S.C. (Statutes of Canada) 1999, c. 33)**

Full Document: [HTML \(FullText.html\)](#) | [XML \(/eng/XML/C-15.31.xml\)](#) [984 KB] | [PDF](#)

[\(/PDF/C-15.31.pdf\)](#) [1347 KB]

Act current to 2015-12-22 and last amended on 2015-02-26. [Previous Versions](#)

[\(PITIndex.html\)](#)

<p><a href="#">Previous Page (page-52.html#docCont)</a></p>	<p><a href="#">Table of Contents</a></p>	<p><a href="#">Next Page (page-54.html#docCont)</a></p>
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## SCHEDULE 1

(Sections 56, 68, 71, 77, 79, 90, 91, 93 to 96 and 199)

### List of Toxic Substances #40

For molecular formulae in this schedule, "n" = number of atoms.

1 Chlorobiphenyls that have the molecular formula  $C_{12}H_{(10-n)}Cl_n$  in which "n" is greater than 2

2 Dodecachloropentacyclo [5.3.0.0<sup>2,6</sup>.0<sup>3,9</sup>.0<sup>4,8</sup>] decane (Mirex)

3 Polybrominated Biphenyls that have the molecular formula  $C_{12}H_{(10-n)}Br_n$  in which "n" is greater than 2

4 Chlorofluorocarbon: totally halogenated chlorofluorocarbons that have the molecular formula  $C_nCl_xF_{(2n+2-x)}$

5 Polychlorinated Terphenyls that have a molecular formula  $C_{18}H_{(14-n)}Cl_n$  in which "n" is greater than 2

6 Asbestos

7 Lead

~~8 Mercury and its compounds~~

40 Inorganic fluorides

41 Refractory ceramic fibre

42 Oxidic, sulphidic and soluble inorganic nickel compounds

43 Polycyclic aromatic hydrocarbons

44 Tetrachloroethylene

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October, 1990

HYDROFLUOSILICIC ACID SPECIFICATIONS

H<sub>2</sub>SiF<sub>6</sub>

COMMERCIAL GRADE

	<u>GUARANTEED</u>	<u>TYPICAL</u>
H <sub>2</sub> SiF <sub>6</sub> (MOL. WT. 144.06)	25% +/- 3% by weight	24%
Fluorine	19% +/- 3% by weight	18.5%
Heavy Metals, as lead. Pb.	0.020% MX.	0.0002%
Phosphorus		0.100%
HF		1.0%
Arsenic		0.0035%
Barium		< 0.0002%
Cadmium		0.0004%
Chromium		< 0.00003%
Iron		0.100%
Iodine		0.0015%
Lead		< 0.00005%
Mercury		< 0.000001%
Selenium		< 0.000003%
Silver		0.0004%
Specific Gravity, at 25%, at 60°F		1.224
Boiling Point for 25%		222.5°F
Freezing Point for 25%		-4.0°F
Weight per Gallon for 25%		10.2 lbs/gal
pH 1% Solution		1.2

Material meets AWWA Standard B703 - 89

Water white to straw yellow in color -- transparent aqueous  
 Solution -- acid liquid.

Clean and free of visible suspended matter.

Highly corrosive, pungent odor -- store in structural carbon,  
 Hastelloy C, Durimer 20 and approved rubber or plastic lined containers.

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 FAX 904-241-1220



8813 Hwy 41 South  
 Riverview, Fl.  
 Telephone: (813) 677-9111 - TELX52666  
 Fax: (813) 671-6283 ACCOUNTING

This product was produced at the  
 Production Plant - Riverview facility

CAR NO: GATX002036

Deliveries: 1001288234

Material: Our / Your reference  
 200011 FLUOROSILICIC ACID /

**Quality Certificate**

Date  
 01/29/2013  
 Purchase order item/date  
 45403267 / 01/09/2013  
 Delivery item/date  
 1001288234 000010 / 01/23/2013  
 Order item/date  
 1508377 000010 / 01/09/2013  
 Customer number  
 5002715

Inspection lot 100000056703 from 01/23/2013

Characteristic	Result	Unit
<b>Chemical Analysis</b>		
Net H <sub>2</sub> SiF <sub>6</sub>	23.53	%
P <sub>2</sub> O <sub>5</sub>	0.19	%
Free Acid	0.34	%
Lead	0.00	ppm
Arsenic	68.75	ppm
<b>Physical Analysis</b>		
Density	1.2130	g/cm <sup>3</sup>
APHA	70	CU

"We certify that product analyzed with this  
 Certificate of Analysis meets AWWA 9003-11"

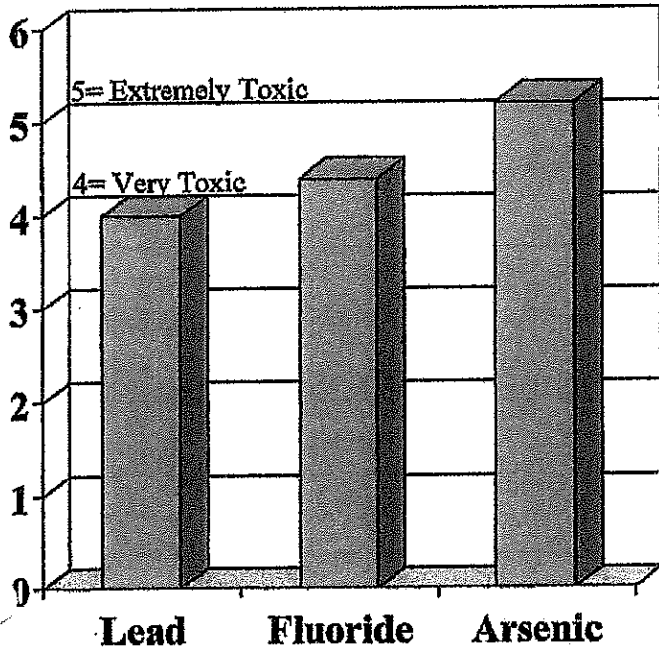


NSF  
 Certified to  
 AWWA 9003-11  
 Max. Use:  
 8 mg/L

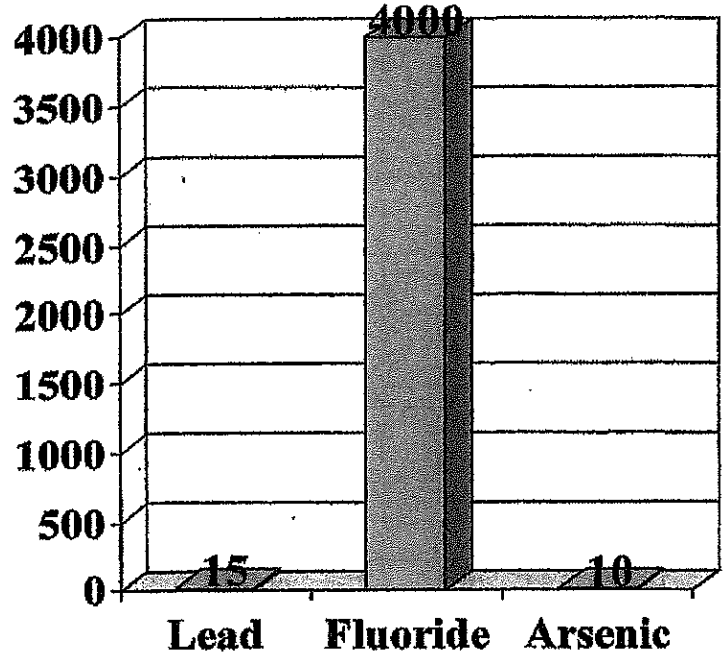
Kwasi Sakyi-Amfo  
 QC Lab Supervisor - Riverview

# How Toxic is Fluoride compared to Lead & Arsenic.

## Relative Toxicity



## EPA Maximum Contaminant Levels



Source: *Clinical Toxicology of Commercial Products* LD50 data - 1984

ppb (Parts per Billion)

## Clinical Toxicology of Commercial Products

**Robert E. Gosselin (Author):** was retired Doctor from Dartmouth Medical School in Hanover, N.H., where he was the founding chairman of the Department of Pharmacology and Toxicology.

**Roger P. Smith (Author):** PhD Emeritus Professor of Pharmacology & Toxicology

**Harold C. Hodge (Author):** was a well-known toxicologist who published close to 300 papers and 5 books with a PhD

**Jeannet Braddock (Author):** PhD

W.H.O. WORLD HEALTH ORGANIZATION

## Media centre

# Lead poisoning and health

Fact sheet

Reviewed September 2016

### Key facts

- Lead is a cumulative toxicant that affects multiple body systems and is particularly harmful to young children.
- Lead in the body is distributed to the brain, liver, kidney and bones. It is stored in the teeth and bones, where it accumulates over time. Human exposure is usually assessed through the measurement of lead in blood.
- Lead in bone is released into blood during pregnancy and becomes a source of exposure to the developing fetus.
- There is no known level of lead exposure that is considered safe.
- Lead poisoning is entirely preventable.

Lead is a naturally occurring toxic metal found in the Earth's crust. Its widespread use has resulted in extensive environmental contamination, human exposure and significant public health problems in many parts of the world.

Important sources of environmental contamination include mining, smelting, manufacturing and recycling activities, and, in some countries, the continued use of leaded paint, leaded gasoline, and leaded aviation fuel. More than three quarters of global lead consumption is for the manufacture of lead-acid batteries for motor vehicles. Lead is, however, also used in many other products, for example pigments, paints, solder, stained glass, lead crystal glassware, ammunition, ceramic glazes, jewellery, toys and in some cosmetics and traditional medicines. Drinking water delivered through lead pipes or pipes joined with lead solder may contain lead. Much of the lead in global commerce is now obtained from recycling.

Young children are particularly vulnerable to the toxic effects of lead and can suffer profound and permanent adverse health effects, particularly affecting the development of the brain and nervous system. Lead also causes long-term harm in adults, including increased risk of high blood pressure and kidney damage. Exposure of pregnant women to high levels of lead can cause miscarriage, stillbirth, premature birth and low birth weight, as well as minor malformations.



## **Sprovieri, John Councillor**

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**From:** Sprovieri, John Councillor  
**Sent:** 2017/01/21 2:19 PM  
**To:** Sprovieri, John Councillor  
**Subject:** FW: Health Canada on health effects of lead

### Guidelines for Canadian Drinking Water Quality - Summary Table

for lead, it says that

***Health basis of MAC [which has not been updated since 1992!]: Biochemical and neurobehavioural effects (intellectual development, behaviour) in infants and young children (under 6 years)***

***Other: Anaemia, central nervous system effects; in pregnant women, can affect the unborn child; in infants and children under 6 years, can affect intellectual development, behaviour, size and hearing; classified as probably carcinogenic to humans.***

and that

***Exposure to lead should nevertheless be kept to a minimum***

1 their Final Human Health State of the Science Report on Lead:

*the following Health Effects are listed with a lot of details and studies on each:*

- Developmental Neurotoxicity
- Neurodegenerative Effects
- Cardiovascular Effects
- Renal Effects
- Reproductive Effects

it also says

*Drinking water is a source of exposure to lead... The amount of lead leaching from the plumbing system is affected by a number of factors, including the age of the plumbing system, the chemistry of the water (e.g., water temperature, pH, buffering capacity/alkalinity)...*

HC'S Lead Information Package says:

***The latest medical and scientific research shows that absorption of even very low levels of lead into the blood may have harmful health effects on the intellectual and behavioural development of infants and young children. Blood lead levels in the range of 10 to 15 micrograms per decilitre in fetuses, infants, and children have been associated with adverse neurobehavioural and cognitive changes. At levels above 40 micrograms per decilitre, there is a decrease in the body's capacity to produce red blood cells.***

**Sprovieri, John Councillor**

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**From:** Sprovieri, John Councillor  
**Sent:** 2014/12/19 7:49 AM  
**To:** Sprovieri, John Councillor  
**Subject:** FW: questions re: Fluoridation and Canada's Food and Drugs Act, etc.

\* "Because arsenic can cause cancer, every effort should be made to keep arsenic levels in drinking water as low as possible."

<http://www.hc-sc.gc.ca/hl-vs/iyh-vsv/environ/arsenic-eng.php>

And regarding lead:

\* "The latest medical and scientific research shows that absorption of even very low levels of lead into the blood may have harmful health effects on the intellectual and behavioural development of infants and young children."

[http://www.hc-sc.gc.ca/ewh-semt/contaminants/lead-plomb/asked\\_questions-questions\\_posees-eng.php](http://www.hc-sc.gc.ca/ewh-semt/contaminants/lead-plomb/asked_questions-questions_posees-eng.php)

"...What can a municipality do to reduce lead levels in water?"

A municipality can evaluate and potentially modify the water's alkalinity and pH levels. In addition, the municipality can use a corrosion inhibitor, which can react with the dissolved lead to form a protective coating on the inside of pipes to reduce the amount of lead leaching into drinking water."

[http://www.hc-sc.gc.ca/ewh-semt/contaminants/lead-plomb/asked\\_questions-questions\\_posees-eng.php#a8](http://www.hc-sc.gc.ca/ewh-semt/contaminants/lead-plomb/asked_questions-questions_posees-eng.php#a8)

Toronto Star, May 20:

"The (10 parts per billion threshold) is obsolete," says Dr. Bruce Lanphear, a health sciences professor who specializes in lead exposure in children at Simon Fraser University. "We've got science that is conclusive, definitive and evaluated by independent advisory boards but policy hasn't kept up with that."

Lanphear said Toronto's 13-per-cent failure rate is a serious concern. "That's excessive and unacceptable from a public health perspective."

\* Kathleen Cooper, senior researcher and lead expert with the Canadian Environmental Law Association, says there is "incredibly solid evidence to say there is no safe level (of lead)."

[http://www.thestar.com/news/gta/2014/05/20/water\\_quality\\_tests\\_data\\_shows\\_elevated\\_lead\\_levels\\_in\\_toronto\\_homes.html](http://www.thestar.com/news/gta/2014/05/20/water_quality_tests_data_shows_elevated_lead_levels_in_toronto_homes.html)

## **Sprovieri, John Councillor**

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**From:** Sprovieri, John Councillor  
**Sent:** 2017/01/13 1:37 PM  
**To:** Sprovieri, John Councillor  
**Subject:** FW: the province allows 2.5x more arsenic than Health Canada's guideline?

Hi John,

The EPA's "Public Health Goal" / "Maximum Contaminant Level Goal" (MCLG) for both arsenic and lead is zero.

**"Definitions: Maximum Contaminant Level Goal (MCLG)—The level of a contaminant in drinking water below which there is no known or expected risk to health. MCLGs allow for a margin of safety and are non-enforceable public health goals."** <https://www.epa.gov/ground-water-and-drinking-water/table-regulated-drinking-water-contaminants#one>

Christine

**So the province allows 2.5 times what HC advises, and Health Canada's guideline, MAC, 0.01 mg/L ALARA as low as reasonably achievable, is not even based on safety but on treatment achievability...**

From Canadian Environmental Law Association, a February 12, 2015 letter to Ministry of the Environment and Climate Change:

### **Arsenic**

**CELA supports lowering the Ontario Drinking Water Quality Standard (ODWQS) for arsenic to 0.010 mg/L from the previous standard of 0.025mg/L. This would bring Ontario's standard in line with Health Canada's 2006 guidelines and ODWAC's (Ontario Drinking Water Advisory Council's) recommendations to the Ministry in 2006. Similarly, CELA has also been advocating for revised standards. 1 According to Health Canada's guidelines, arsenic is highly toxic and a known human carcinogen with significant adverse impacts for human health. 2 In addition to cancer, long term exposure to arsenic can result in cardiovascular disease, diabetes, developmental effects, and neurotoxicity. 3**

**The ODWAC has affirmed that the standards for carcinogenic contaminants in Ontario should be as close to 'essentially negligible' as possible (one new cancer above background levels in a million people, after a lifetime (70 years) of exposure). 4 This revised ODWQS is not in the essentially negligible range; however, this is what is considered to be achievable at reasonable cost to drinking water treatment systems. 5 The estimated lifetime risk of excess internal organ cancers for this revised ODWQS is 3 to 39 additional cancers. While this is an improvement over the previous standard (8 to 97 additional cancers), 6 water treatment systems should be encouraged to reduce arsenic levels in drinking water as much as possible and the ODWQS should be further revised when feasible.**

...Also, a blog post on their website says: