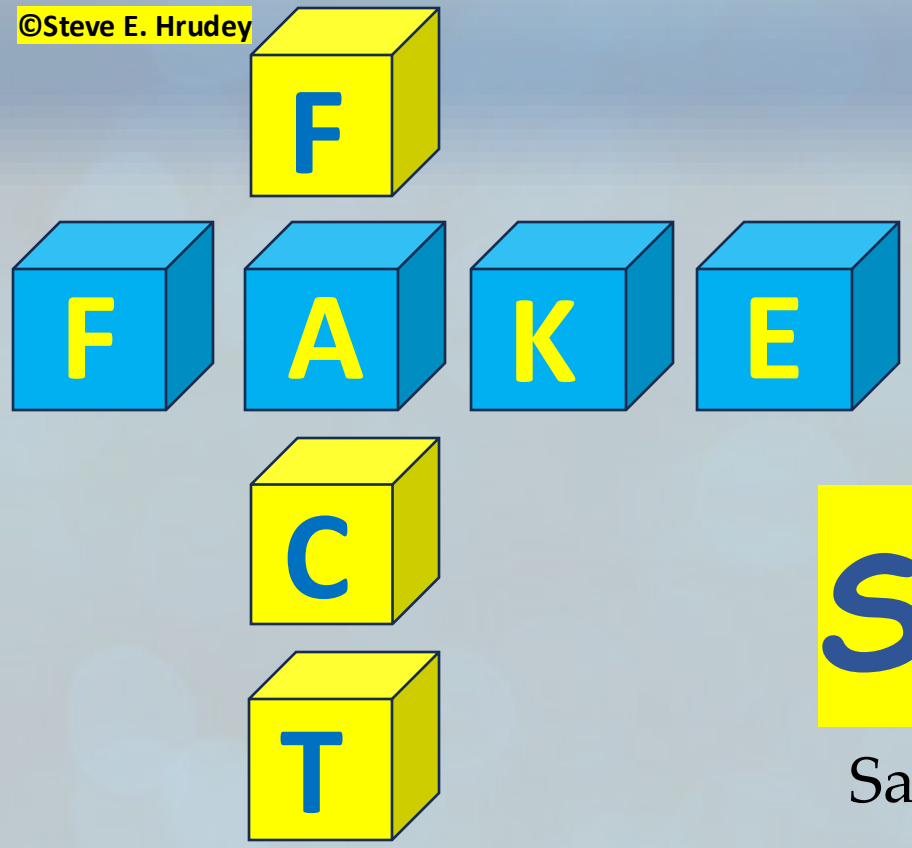


Misinformation vs. Safe Drinking Water

Saskatchewan Water & Wastewater Association
Annual Conference & Trade Show
Saskatoon, Saskatchewan
November 7, 2024

Steve E. Hrudey
Professor Emeritus
Analytical & Environmental Toxicology, Faculty of Medicine & Dentistry
UNIVERSITY OF ALBERTA

Use Caution with Popular Mythology

Protecting Public Health MUST Be Your JOB 1

I assume (***at least I hope***) that no one here advocates allowing drinking water to make consumers ill

Presentation Outline

Preamble – Risk Management

An Authentic Case of Myths Killing People

Popular Mythology to Address

1. Natural is inherently safer than synthetic
2. Contaminant detection means a likely health risk
3. Environmental contaminants are causing a cancer epidemic
4. Must regulate contaminants regardless of drinking water exposure
5. No safe level for some drinking water contaminants

Turning Hindsight Into Foresight – Learning From Experience

Practical Actions

Concluding Thoughts

Preamble - Risk Management

Risk Management for drinking water
(O'Connor, 2002, Walkerton Inquiry Part 2)
to be effective, seeks:

- *"being preventive rather than reactive;*
- *distinguishing greater risks from lesser ones, and dealing first with the former;*
- *taking time to learn from experience; and*
- *investing resources in risk management that are proportional to the danger posed"*

Preamble - Risk Management

Risk Management, to be successful, requires effective action

- Effective action depends upon there being truths that can be verified by credible evidence
- Affected or responsible parties must believe the need for, or merits of, proposed actions
- "***Normalization***" means popular beliefs that have become common knowledge being accepted as true because of frequent repetition
- **5 Popular Myths** interfering with effective risk management are inaccurate beliefs that have become normalized in our society
- There are many more myths than we have time to discuss today

But First…

What may be the most dangerous recent public health myth

- This example is dramatically worse than anything bearing directly on your responsibility to provide safe drinking water: **bad faith + dishonesty**
- Arguably, this dangerous myth has **contributed to** many thousands of preventable deaths
- Dr. Carl Sagan (1934-1996), astronomer and science communicator wisely stated: ***"Extraordinary claims require extraordinary evidence"***

United States: COVID-19 weekly death rate by vaccination status, All ages

Our World in Data

Death rates are calculated as the number of deaths in each group, divided by the total number in this group.
This **rate** is given per 100,000 people

This, IS extraordinary evidence -
from a historically **huge** unnatural experiment

Unvaccinated
Vaccinated **without** bivalent booster
Vaccinated **with** bivalent booster

Data source: Centers for Disease Control and Prevention, Vaccine Breakthrough/Surveillance and Analytics Team
Note: The mortality rate for the 'All ages' group is age-standardized to account for the different vaccination rates of older and younger people.
OurWorldInData.org/coronavirus | CC BY

What Is The Evidence for Vaccine Opposition?

The "*Foundations*" for vaccine opposition can be traced to a remarkably flawed study by an English "*medical researcher*"

- Andrew Wakefield was the principal investigator and lead author of a "***study***" at the London Royal Free Hospital
- He published a 1998 paper in *The Lancet* one of the two top British medical journals
- The "*study*" purported to demonstrate evidence of a causal link between the MMR (measles, mumps, rubella) vaccine and autism in children
- Publicity around the the "*study*" led to a severe drop in MMR vaccination rates ultimately causing childhood deaths from measles
- The "*study*" had a suspect history and many **severe conflicts of interest**

Early report

A.J. Wakefield

Ileal-lymphoid-nodular hyperplasia, non-specific colitis, and pervasive developmental disorder in children

A J Wakefield, S H Murch, A Anthony, J Linnell, D M Casson, M Malik, M Berelowitz, A P Dhillon, M A Thomson, P Harvey, A Valentine, S E Davies, J A Walker-Smith

Summary

Background We investigated a consecutive series of children with chronic enterocolitis and regressive developmental disorder.

Methods 12 children (mean age 6 years [range 3–10], 11 boys) were referred to a paediatric gastroenterology unit with a history of normal development followed by loss of acquired skills, including language, together with diarrhoea and abdominal pain. Children underwent gastroenterological, neurological, and developmental assessment and review of developmental records. Ileocolonoscopy and biopsy sampling, magnetic-resonance imaging (MRI), electroencephalography (EEG), and lumbar puncture were done under sedation. Barium follow-through radiography was done where possible. Biochemical, haematological, and immunological profiles were examined.

Findings Onset of behavioural symptoms was associ... by the parents, with measles, mumps, and rub... vaccination in eight of the 12 children, with measl... infection in one child, and otitis media in an... All 12 children had intestinal abnormalities ...ging from lymphoid nodular hyperplasia to a... oid ul...ration. Histology showed patchy chronic inflam...tion ... in 11 children and reactive ileal lympho... perplasia in seven, but no granulomas. Be...vioural diso...s included autism (nine), disintegrativ...syc...sis (one), a... possible postviral or vaccinal encephalitis ...o). There were no focal neurological ab...rmalities and ...RI and EEG tests were normal. Abno...al laboratory results ...ere significantly raised urinary ...ethylmal...c acid compared with age-matched contro... (p=0.003), low ... haemoglobin in four children, a... low s...m IgA in ...r children.

Inter...tation ...e ident... associated gastrointestinal di...se and ...evelopmental regression in a group of previ...ly n...mal ch...n, which was generally associated in time ... possible environmental triggers.

Lancet 199... **351:** 637–41
See Commentary page

Inflammatory Bowel Disease Study Group, University Departments of Medicine and Histopathology (A J Wakefield FRCS, A Anthony MB, J Linnell PhD, A P Dhillon MRCPath, S E Davies MRCPath) **and the University Departments of Paediatric Gastroenterology** (S H Murch MB, D M Casson MRCP, M Malik M... M A Thomson FRCP, J A Walker-Smith FRCP), **C... Psychiatry** (M Berelowitz FRCPsych), **Neurology ... Radiology** (A Valentine FRCR), **Royal Free Hospital and School of Medicine, London NW3 2QG, UK**

Correspondence to: Dr A. J. Wakefield

THE LANCET

Introduction

We saw several children who, after a p...d of apparent normality, lost acquired skills, includ...g com...nication. They all had gastrointestinal ...mptoms, ...luding abdominal pain, diarrhoea, and ...ating and, i... some cases, food intolerance. We d...scribe... clinical f...dings, and gastrointestinal featur... of these ch...en.

Patients and meth...ds

12 children, cons...tively ...red to t... department of paediatric gastr...terology ... a hi...y of a pervasive developmental ...der with loss ...ed skills and intestinal symptoms ...arrh... abdominal ...in, bloating and food intolerance), were inv...gated. All children were admitted to the ward f... week, accomp...ed by their parents.

Clinical investigations

...took histori... including details of immunisations and e...posure to infect...us diseases, and assessed the children. In 11 cas... the histor...as obtained by the senior clinician (JW-S). Neuro...l and psychiatric assessments were done by consultant staff (PH, MB) with HMS-4 criteria.[1] Developmental ...s included a review of prospective developmental records from parents, health visitors, and general practitioners. Four children did not undergo psychiatric assessment in hospital; all had been assessed professionally elsewhere, so these assessments were used as the basis for their behavioural diagnosis.

After bowel preparation, ileocolonoscopy was performed by SHM or MAT under sedation with midazolam and pethidine. Paired frozen and formalin-fixed mucosal biopsy samples were taken from the terminal ileum; ascending, transverse, descending, and sigmoid colons, and from the rectum. The procedure was recorded by video or still images, and were compared with images of the previous seven consecutive paediatric colonoscopies (four normal colonoscopies and three on children with ulcerative colitis), in which the physician reported normal appearances in the terminal ileum. Barium follow-through radiography was possible in some cases.

Also under sedation, cerebral magnetic-resonance imaging (MRI), electroencephalography (EEG) including visual, brain stem auditory, and sensory evoked potentials (where compliance made these possible), and lumbar puncture were done.

Laboratory investigations

Thyroid function, serum long-chain fatty acids, and cerebrospinal-fluid lactate were measured to exclude known causes of childhood neurodegenerative disease. Urinary methylmalonic acid was measured in random urine samples from eight of the 12 children and 14 age-matched and sex-matched normal controls, by a modification of a technique described previously.[2] Chromatograms were scanned digitally on computer, to analyse the methylmalonic-acid zones from cases ...trols. Urinary methylmalonic-acid concentrations in ... and controls were compared by a two-sample *t* test. ... creatinine was estimated by routine spectrophotometric assay.

Children were screened for antiendomyseal antibodies and boys were screened for fragile-X if this had not been done

Behavioural diagnosis, several inaccurate

Exposure identified mostly by **parents**

Table 2 **Neuropsychiatric Diagnoses**

Child	Behavioural diagnosis	Exposure identified by parents or doctor	Interval from exposure to first behavioural symptom	Features associated with exposure
1	Autism	MMR	1 week	Fever/delirium
2	Autism	MMR	2 weeks	Self injury
3	Autism	MMR	48 h	Rash and fever
4	Autism? Disintegrative disorder?	MMR	Measles vaccine at 15 months followed by slowing in development. Dramatic deterioration in behaviour immediately after MMR at 4·5 years	Repetitive behaviour, self injury, loss of self-help
5	Autism	None—MMR at 16 months	Self-injurious behaviour started at 18 months	
6	Autism	MMR	1 week	Rash & convulsion; gaze avoidance & self injury
7	Autism	MMR	24 h	Convulsion, gaze avoidance
8	Post-vaccinial encephalitis?	MMR	2 weeks	Fever, convulsion, rash & diarrhoea
9	Autistic spectrum disorder	Recurrent otitis media	1 week (MMR 2 months previously)	Disinterest; lack of play
10	Post-viral encephalitis?	Measles (previously vaccinated with MMR)	24 h	Fever, rash & vomiting
11	Autism	MMR	1 week	Recurrent "viral pneumonia" for 8 weeks following MMR
12	Autism	None—MMR at 15 months	Loss of speech development and deterioration in language skills noted at 1...	

MMR=measles, mumps, and Rubella va...

- ❑ Only **12** child subjects
- ❑ Were referred by an **anti-vaxx** group ***"JABS"***
- ❑ **Enormous** selection bias

What Is The Evidence for Vaccine Opposition?

Wakefield proved to be wholly unqualified to lead this research

- Wakefield had initially been trying to prove that Crohn's disease was caused by chronic measles viral infection of the gut
- The hypothesis that MMR vaccine caused autism was originally proposed to Wakefield by an anti-Vaxx activist
- A lawyer working for anti-Vaxx activists led a class action lawsuit & retained Wakefield before the *Lancet* paper to find evidence for the autism theory with a £150/hr fee that, in total, paid Wakefield **£435,643** plus expenses
- Wakefield used media publicity from the *Lancet* paper to advocate abandoning MMR for a measles-only vaccine, **AFTER** obtaining a patent for **his** measles vaccine
- UK General Medical Council held a **£6 million, 217 day** Inquiry finding Wakefield: "***dishonest***", "***unethical***" and "***callous***" to rescind his medical license in 2010.
- Since moving to Texas, Wakefield has become a leading anti-vaxxer

Prevalent Myth 1

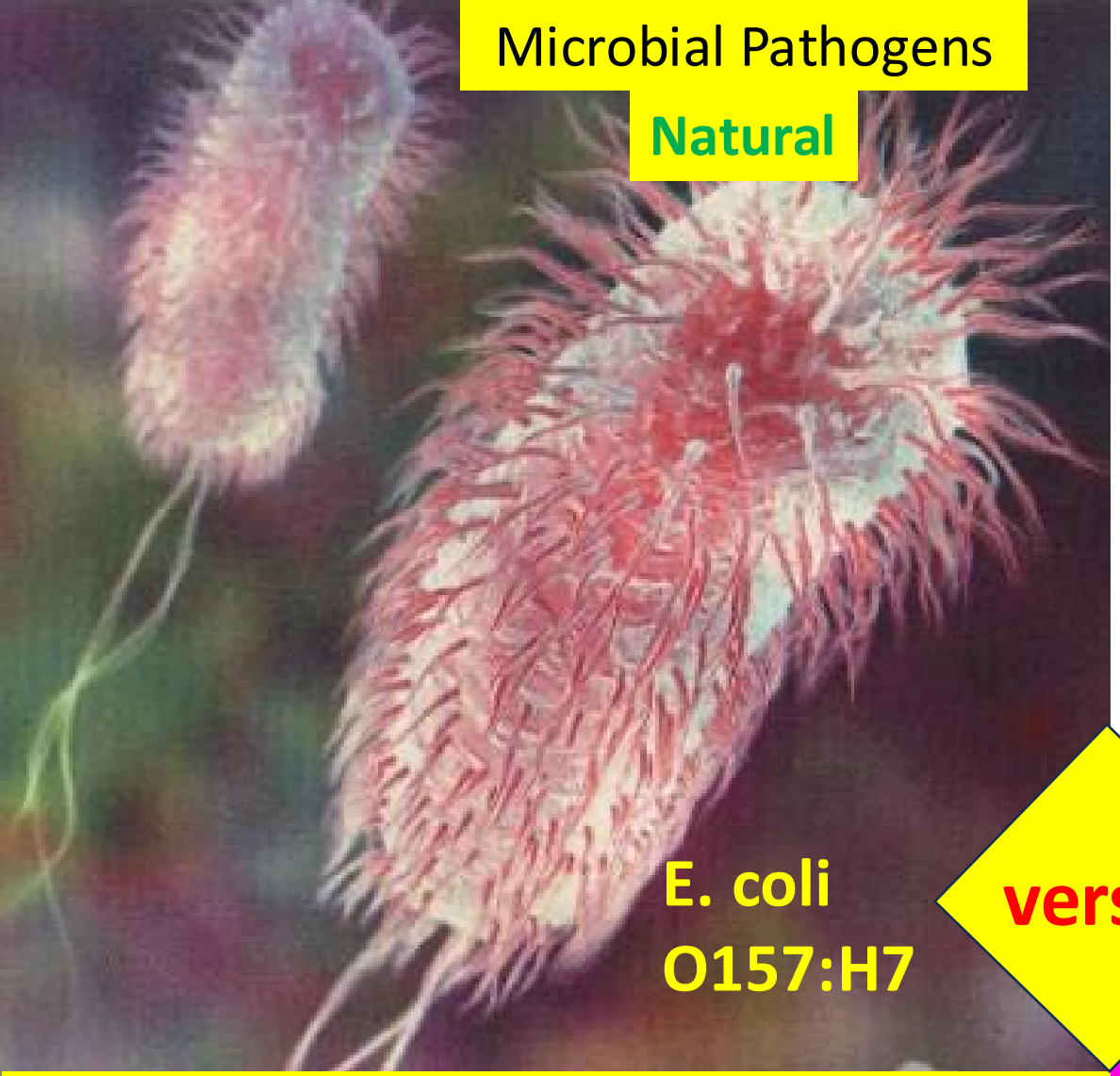
Natural is inherently safer than synthetic


ALL Natural

Natural but Human-influenced

Microbial Pathogens

Natural

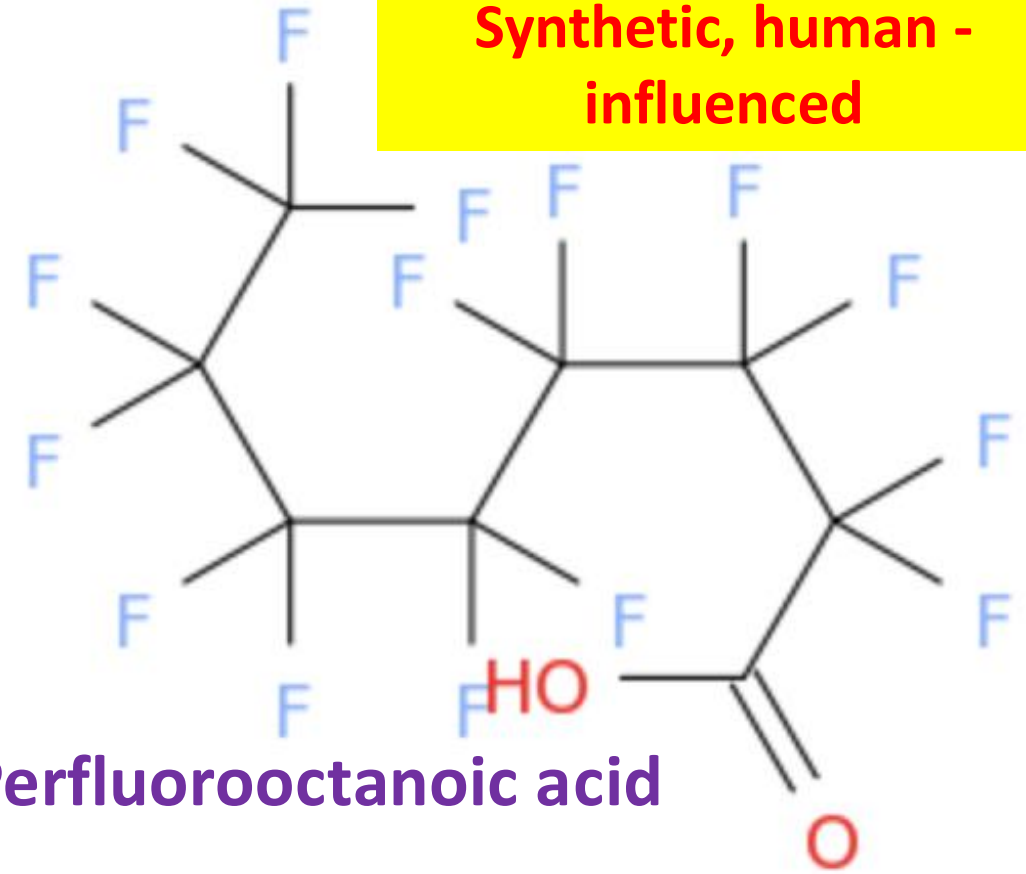


E. coli O157:H7

versus

Chemical Contaminants

Synthetic, human - influenced



Perfluorooctanoic acid

Chloroform

We know for **certain** that microbial pathogens have killed consumers via drinking water exposure

We suspect, with **considerable uncertainty,** that some chemicals **MAY** cause human illness via drinking water exposure, **evidence is largely inferential**

What Chemicals ARE a Risk Management Priority for Drinking Water?

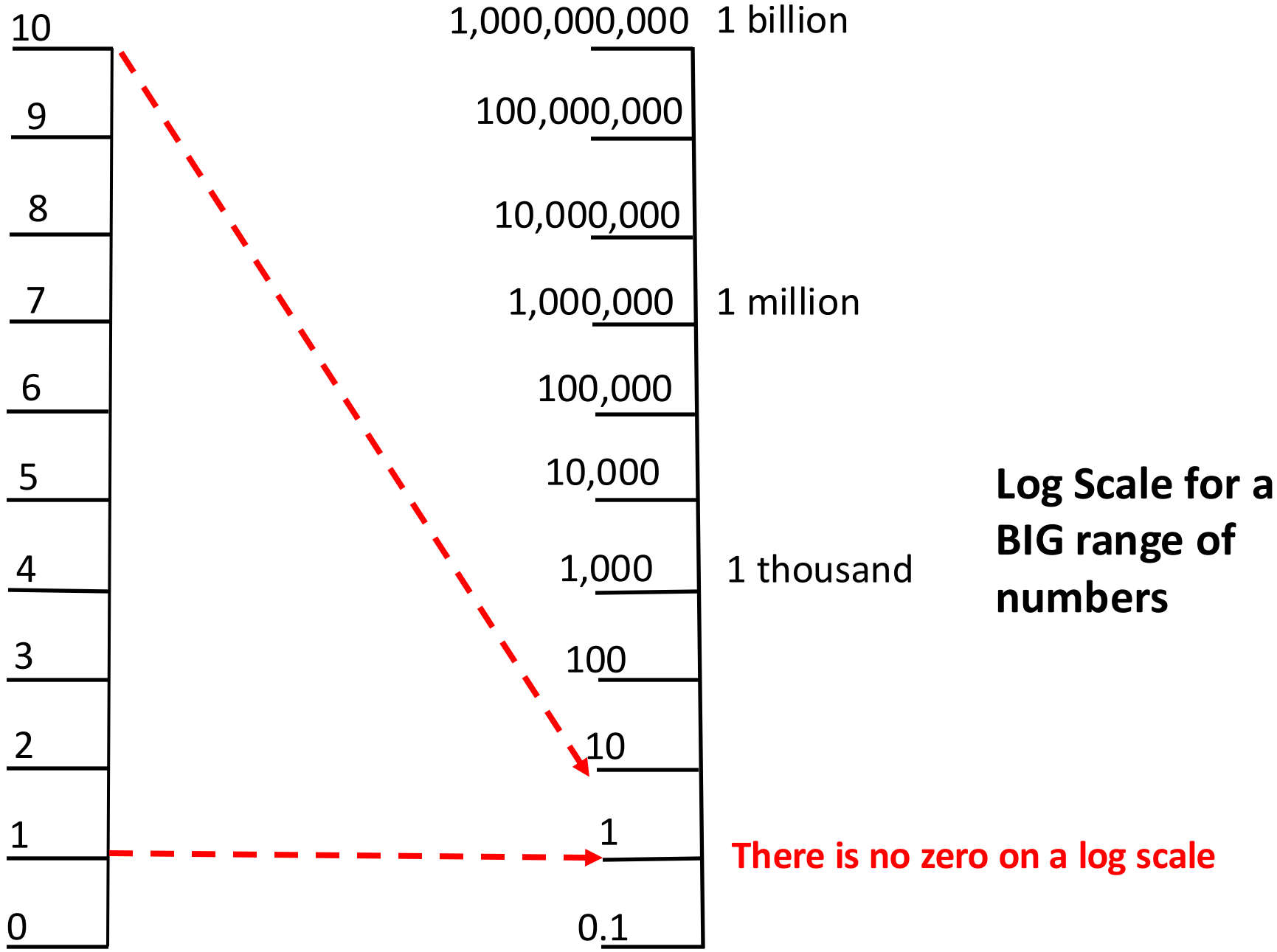
World Health Organization (Thompson et al. 2007) **assessed Priority Chemicals based on credible evidence of human health risks from drinking water**

- NOT all chemicals that could be a human health risk – **only SOME ARE a human health risk FROM CONSUMING DRINKING WATER**
- **arsenic**, **fluoride**, **selenium**, **lead, nitrate**
- **3** of the above 5 occur in drinking water from natural sources
- lead is a natural element, but exposure typically from plumbing sources
- nitrate can be natural but is most commonly a result of fertilization
- 5 among 52 chemicals (or groups) in GCDWQ that have health-based MACs

Prevalent Myth 2

Contaminant detection means a LIKELY health risk

Understanding contaminants requires understanding scale



A Quantitative Perspective for Health Risks

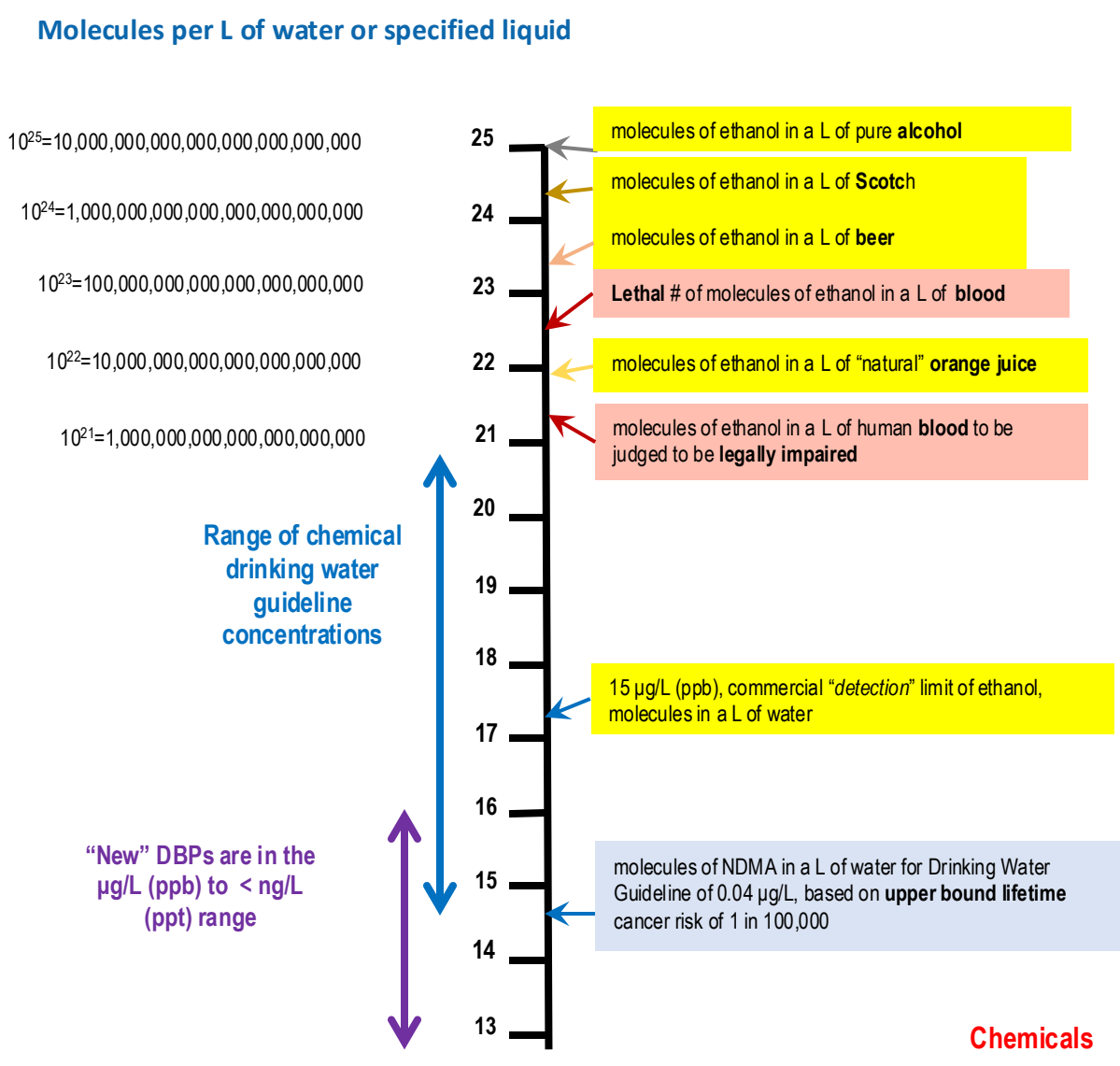
TOXICOLOGY is the Basic Science of Poisons

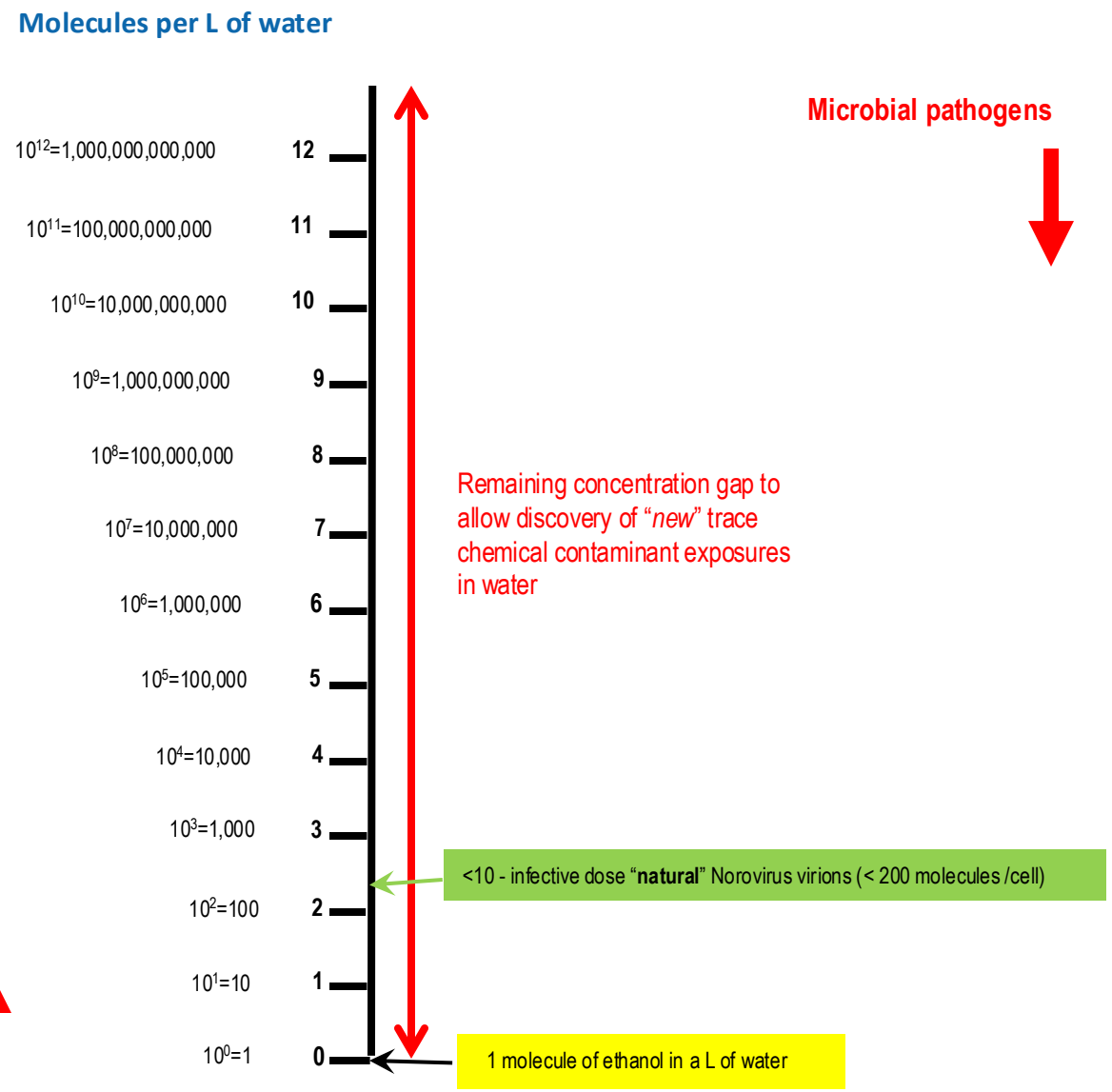
- *"The dose makes the poison"*
- Toxicology provides the medical science foundations for understanding the mechanisms of toxic action
- For trace contaminants, toxicology largely relies upon experiments with animal models
- Public Health deals with inevitable uncertainties by applying caution
- Consider a current relevant example of *"The dose makes the poison"* – Botulinum toxin

A Quantitative Perspective for Health Risks

- *"Botulinum toxins are one of the most lethal substances known. Botulinum toxins block nerve functions and can lead to respiratory and muscular paralysis"* WHO (2023a)
- Botulinum toxins are up to 6 million times more lethal to humans than sodium cyanide
- Botulinum toxin also known as "Botox".
- This extremely lethal toxin is widely being marketed for cosmetic use for removing facial wrinkles

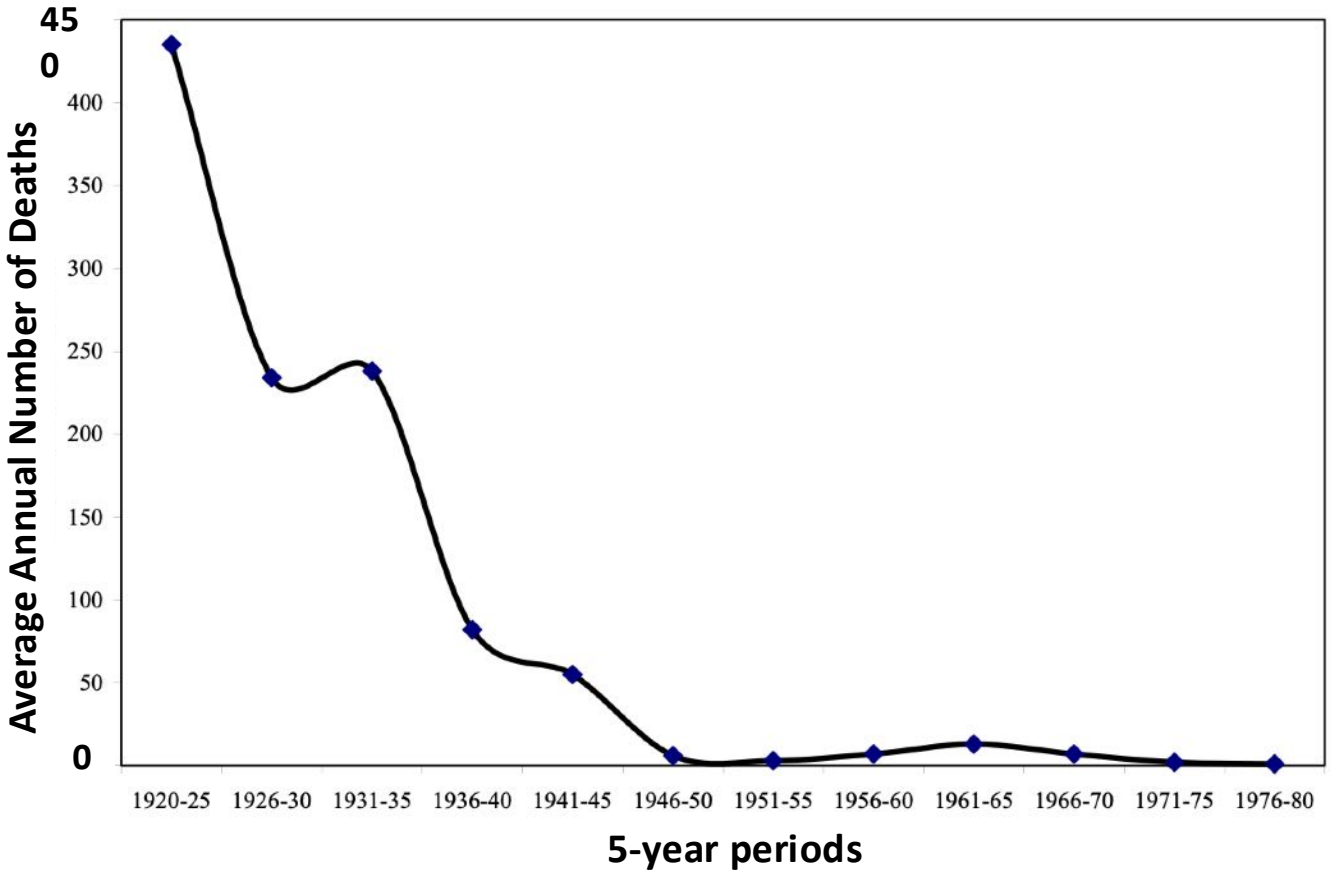
We will illustrate quantitative realities using another much less toxic substance that most of us have been exposed to (some without knowing)
Ethyl alcohol (ethanol) IS lethally toxic in sufficient dose





Evidence for Drinking Water Health Risks

World Health Organization (WHO 2023b) currently estimates **505,000 deaths per year** are caused by diarrheoal diseases from **microbial pathogen-**contaminated drinking water (e.g., cholera, diarrhoea, dysentery, hepatitis A, typhoid)



Number of Deaths (per 5 years) from waterborne outbreaks in the **U.S. 1920 – 1980** (Craun 1986)

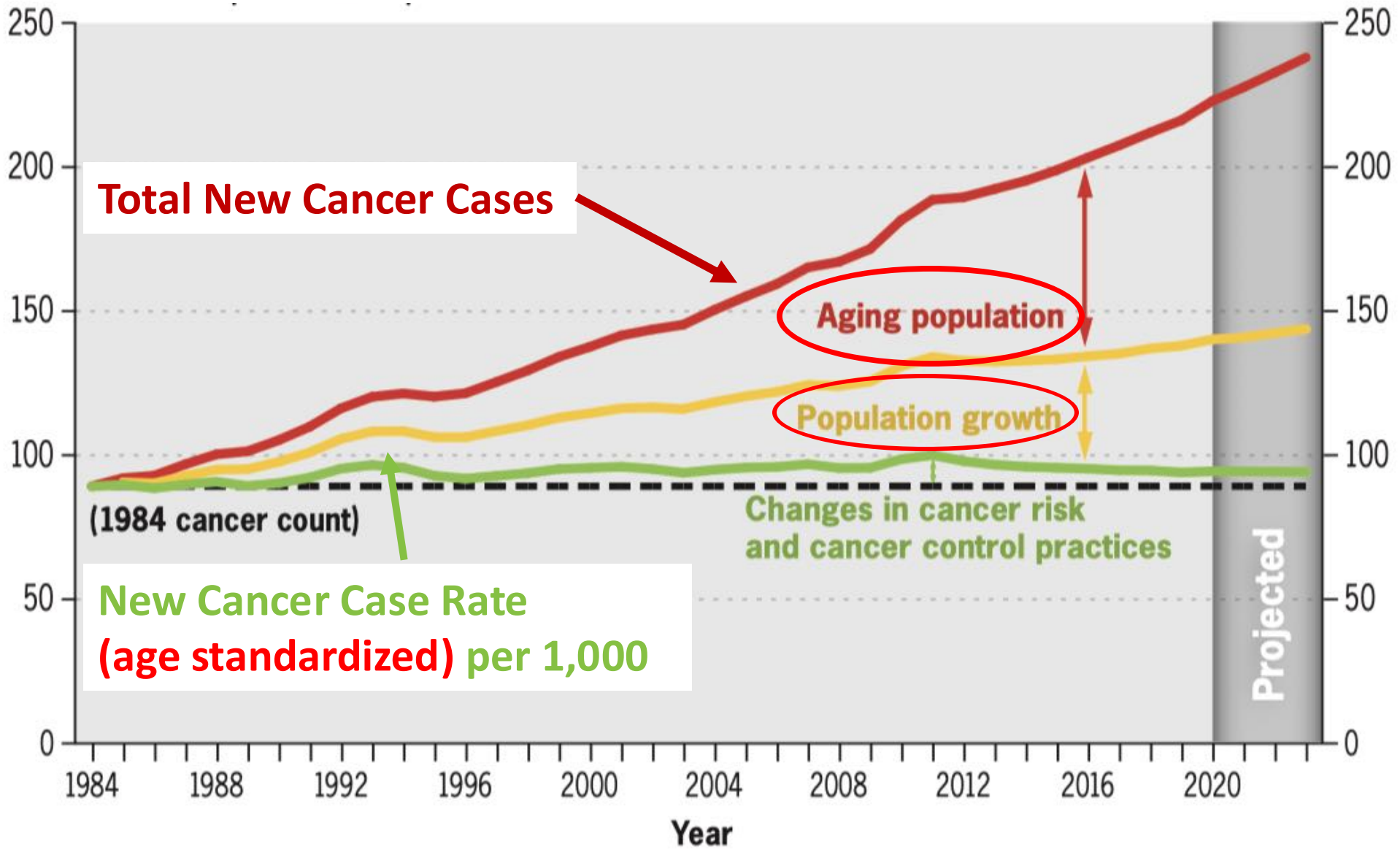
Prevalent Myth 3

Environmental contaminants are causing a Cancer EPIDEMIC

Origins of Cancer Epidemic Claims

- Rachel Carson's iconic 1962 book "*Silent Spring*" about irresponsible, excessive use or pesticides launched modern environmental movement
- Link of excessive DDT use and decline of raptors was a major discovery
- Carson also devoted an entire chapter to growing cancer risk caused by human-made environmental pollution
- Influenced by early WHO estimates that more than 90% of cancers were caused by "*extrinsic*" (non-inherited, genetic) factors
- "*Extrinsic*" covered life-style factors (e.g., smoking, diet, alcohol consumption, sexual behaviours, etc.)
- "*Extrinsic*" became widely termed "*environmental*" which was appropriated to mean environmental contaminants

Cancer Incidence (New Cases)



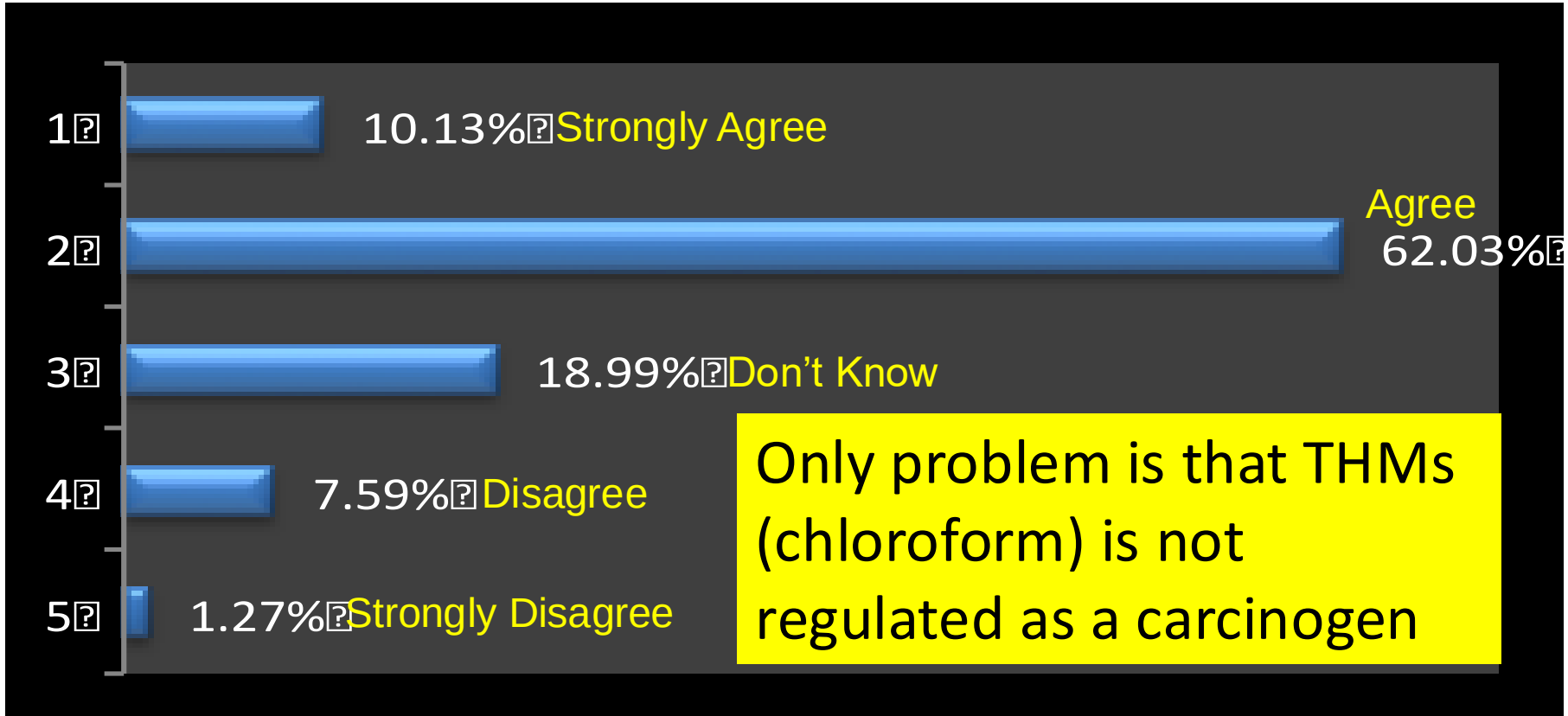
cancer.ca/Canadian-Cancer-Statistics-2023-EN

"The number of cancer cases diagnosed each year has been increasing largely due to the growing and aging population. When the effect of age and population size are removed, the risk of cancer has been decreasing."

"Overall, age-standardized cancer rates have declined : 1.2% annually since 2011 for males and : 0.4% annually since 2012 for females."

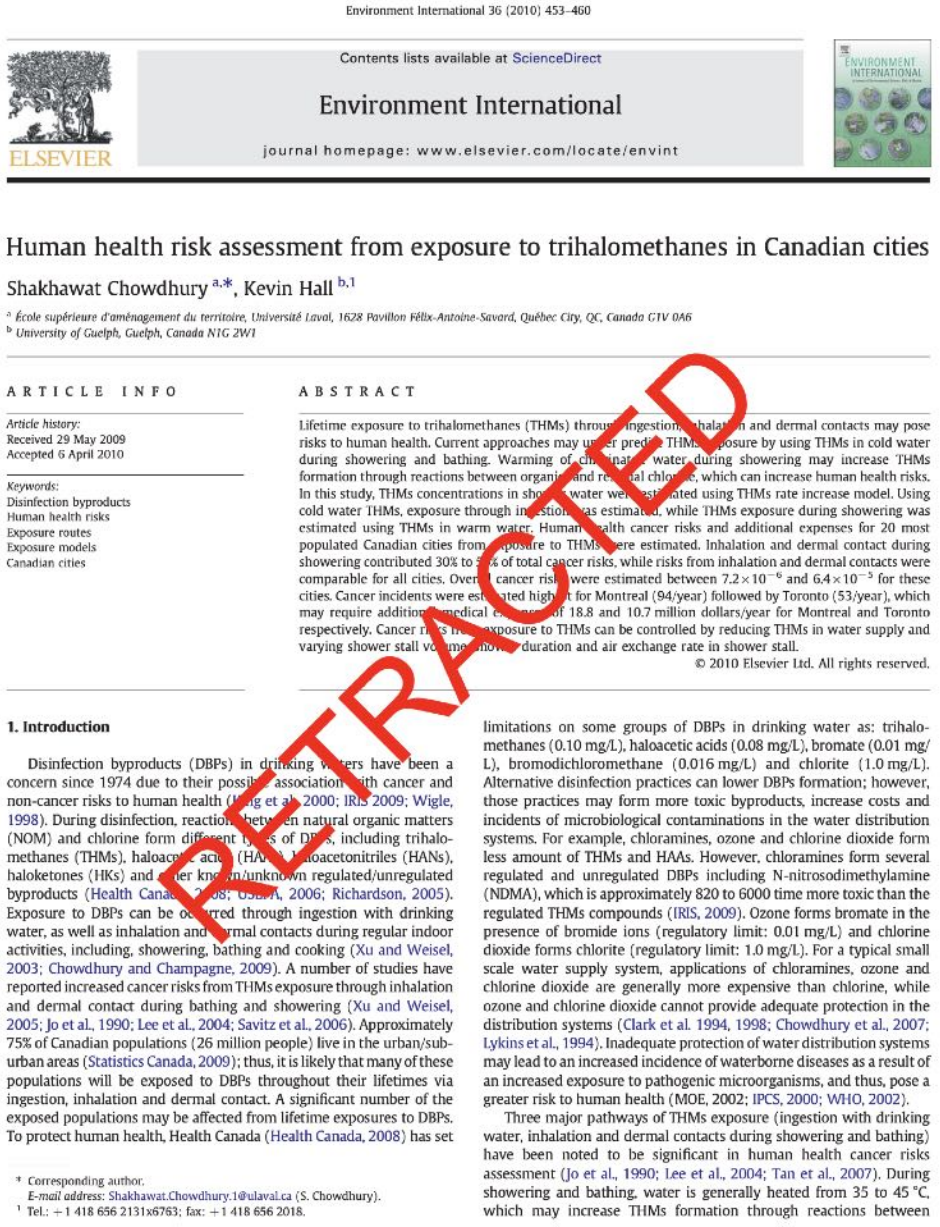
Are Cancer Risks from DBPs Misunderstood?

- Survey done at a drinking water professionals' seminar – BCWWA, Penticton, April 25, 2012.
- Attendees were asked for agreement or disagreement with: "*Chloroform, the most common THM, has a drinking water guideline mainly to manage cancer risk*"



Cancer Concerns for THMs

- Example: Chowdhury & Hall 2010
- Abstract: "*Human health cancer risks … for 20 most populated Canadian cities from exposure to THMs was estimated*"
- "***Cancer incidents* [sic]** *were estimated highest for* ***Montreal (94/year) followed by Toronto (53/year)*** *…*"
- The cancer predictions were **totally erroneous** - primary author was told this but did not disclose that to the Journal
- Misleading reports keep happening, see Cotruvo et al. 2020



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RETRACTED

Human health risk assessment from exposure to trihalomethanes in Canadian cities

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ABSTRACT

Lifetime exposure to trihalomethanes (THMs) through ingestion, inhalation and dermal contacts may pose risks to human health. Current approaches may underpredict THMs exposure by using THMs in cold water during showering and bathing. Warming of chlorinated water during showering may increase THMs formation through reactions between organics and residual chlorine, which can increase human health risks. In this study, THMs concentrations in shower water were estimated using THMs rate increase model. Using cold water THMs, exposure through ingestion was estimated, while THMs exposure during showering was estimated using THMs in warm water. Human health cancer risks and additional expenses for 20 most populated Canadian cities from exposure to THMs were estimated. Inhalation and dermal contact during showering contributed 30% to 57% of total cancer risks, while risks from inhalation and dermal contacts were comparable for all cities. Overall cancer risks were estimated between 7.2×10^{-6} and 6.4×10^{-5} for these cities. Cancer incidents were estimated highest for Montreal (94/year) followed by Toronto (53/year), which may require additional medical expenses of 18.8 and 10.7 million dollars/year for Montreal and Toronto respectively. Cancer risks from exposure to THMs can be controlled by reducing THMs in water supply and varying shower stall volume, shower duration and air exchange rate in shower stall.

1. Introduction

Disinfection byproducts (DBPs) in drinking waters have been a concern since 1974 due to their possible association with cancer and non-cancer risks to human health (Ding et al., 2000; IRIS 2009; Wigle, 1998). During disinfection, reactions between natural organic matters (NOM) and chlorine form different types of DBPs, including trihalomethanes (THMs), haloacetic acids (HAAs), haloacetonitriles (HANs), haloketones (HKs) and other known/unknown regulated/unregulated byproducts (Health Canada, 2008; USEPA, 2006; Richardson, 2005). Exposure to DBPs can be occurred through ingestion with drinking water, as well as inhalation and dermal contacts during regular indoor activities, including, showering, bathing and cooking (Xu and Weisel, 2003; Chowdhury and Champagne, 2009). A number of studies have reported increased cancer risks from THMs exposure through inhalation and dermal contact during bathing and showering (Xu and Weisel, 2005; Jo et al., 1990; Lee et al., 2004; Savitz et al., 2006). Approximately 75% of Canadian populations (26 million people) live in the urban/sub-urban areas (Statistics Canada, 2009); thus, it is likely that many of these populations will be exposed to DBPs throughout their lifetimes via ingestion, inhalation and dermal contact. A significant number of the exposed populations may be affected from lifetime exposures to DBPs. To protect human health, Health Canada (Health Canada, 2008) has set limitations on some groups of DBPs in drinking water as: trihalomethanes (0.10 mg/L), haloacetic acids (0.08 mg/L), bromate (0.01 mg/L), bromodichloromethane (0.016 mg/L) and chlorite (1.0 mg/L). Alternative disinfection practices can lower DBPs formation; however, those practices may form more toxic byproducts, increase costs and incidents of microbiological contaminations in the water distribution systems. For example, chloramines, ozone and chlorine dioxide form less amount of THMs and HAAs. However, chloramines form several regulated and unregulated DBPs including N-nitrosodimethylamine (NDMA), which is approximately 820 to 6000 time more toxic than the regulated THMs compounds (IRIS, 2009). Ozone forms bromate in the presence of bromide ions (regulatory limit: 0.01 mg/L) and chlorine dioxide forms chlorite (regulatory limit: 1.0 mg/L). For a typical small scale water supply system, applications of chloramines, ozone and chlorine dioxide are generally more expensive than chlorine, while ozone and chlorine dioxide cannot provide adequate protection in the distribution systems (Clark et al. 1994, 1998; Chowdhury et al., 2007; Lykins et al., 1994). Inadequate protection of water distribution systems may lead to an increased incidence of waterborne diseases as a result of an increased exposure to pathogenic microorganisms, and thus, pose a greater risk to human health (MOE, 2002; IPCS, 2000; WHO, 2002).

Three major pathways of THMs exposure (ingestion with drinking water, inhalation and dermal contacts during showering and bathing) have been noted to be significant in human health cancer risks assessment (Jo et al., 1990; Lee et al., 2004; Tan et al., 2007). During showering and bathing, water is generally heated from 35 to 45 °C, which may increase THMs formation through reactions between

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Prevalent Myth 4

Must Regulate contaminants regardless of drinking water exposure

"Regulation" Regardless of Relative Exposure

Managing risk for any exposure route should protect public health

- If 99% of public exposure to a contaminant comes from sources other than drinking water, regulating drinking water exposure can only control less than 1% of risk
- Water Research Foundation contracted us to study the case of nitrosamines (such as NDMA) that are clearly carcinogenic to mammals and occur as disinfection by-products in some drinking waters
- US EPA had collected 18,000 drinking water samples to assess need to regulate nitrosamines under the Safe Drinking Water Act
- We studied the evidence for relative exposure to nitrosamines via drinking water

"Regulation" Regardless of Relative Exposure

Drinking Water as a Proportion of Total Human Exposure to Volatile *N*-Nitrosamines

Steve E. Hrudey,[1,*] Richard J. Bull,[2] Joseph A. Cotruvo,[3] Greg Paoli,[4] and Margaret Wilson [4]

2013 *Risk Analysis* 34(5):791-793

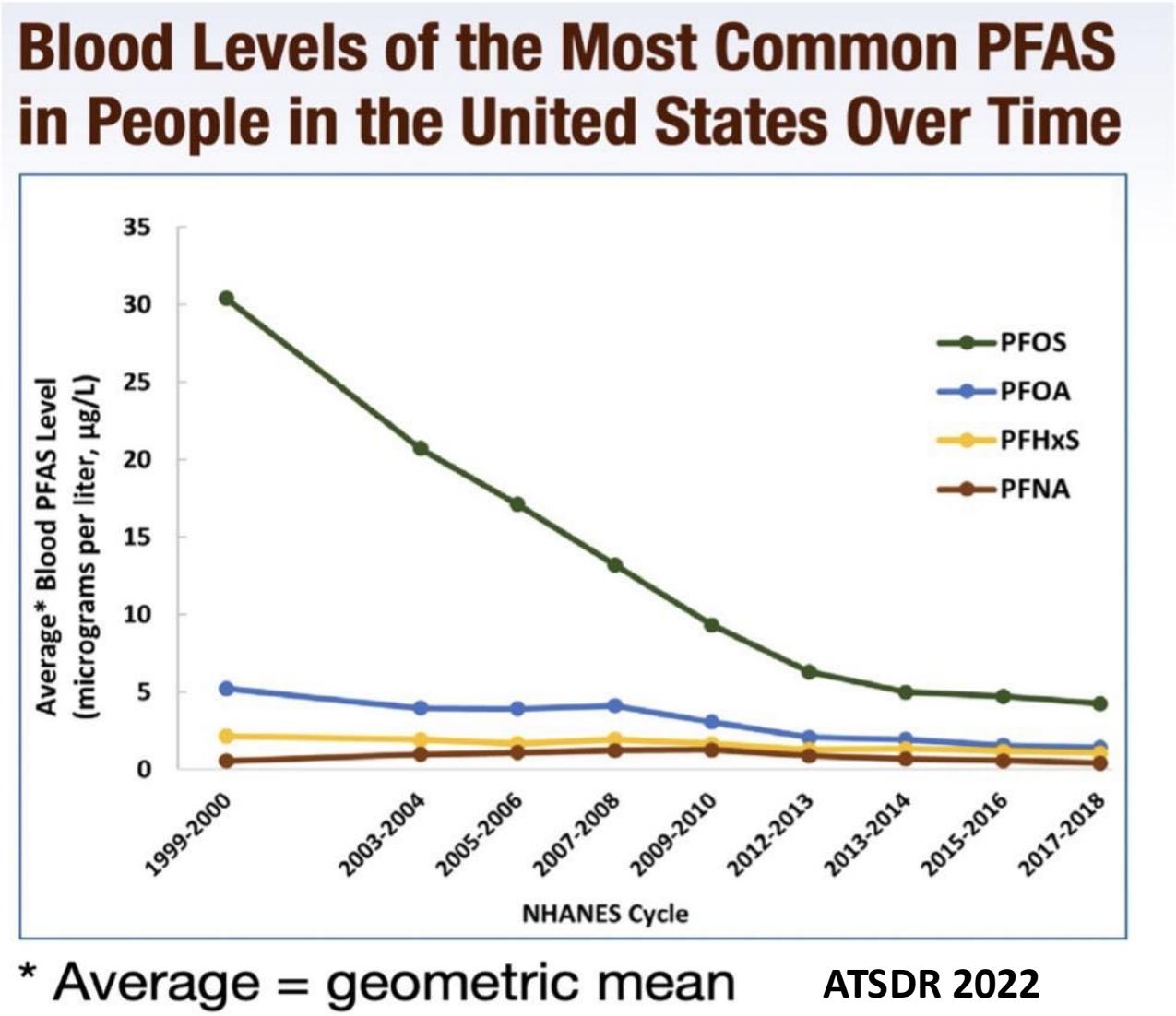
Some volatile *N*-nitrosamines, primarily *N*-nitrosodimethylamine (NDMA), are recognized as products of drinking water treatment at ng/L levels and as known carcinogens. The U.S. EPA has identified the *N*-nitrosamines as contaminants being considered for regulation as a group under the Safe Drinking Water Act. Nitrosamines are common dietary components, and a major database (over 18,000 drinking water samples) has recently been created under the Unregulated Contaminant Monitoring Rule. A Monte Carlo modeling analysis in 2007 found that drinking water contributed less than 2.8% of ingested NDMA and less than 0.02% of total NDMA exposure when estimated endogenous formation was considered. Our analysis, based upon human blood concentrations, indicates that endogenous NDMA production is larger than expected. The blood-based estimates are within the range that would be calculated from estimates based on daily urinary NDMA excretion and an estimate based on methylated guanine in DNA of lymphocytes from human volunteers. Our analysis of ingested NDMA from food and water based on Monte Carlo modeling with more complete data input shows that drinking water contributes a mean proportion of the lifetime average daily NDMA dose ranging from between 0.0002% and 0.001% for surface water systems using free chlorine or between 0.001% and 0.01% for surface water systems using chloramines. The proportions of average daily dose are higher for infants (zero to six months) than other age cohorts, with the highest mean up to 0.09% (upper 95th percentile of 0.3%).

KEY WORDS: Comparative exposure assessment; dietary intake; endogenous formation; NDMA

Evidence for NDMA DW exposure

- For free chlorine, surface water systems lifetime daily NDMA dose from drinking water is between **0.0002% and 0.001%** of total daily dose
- For chloramine, surface water systems lifetime daily NDMA dose from drinking water is **0.001% to 0.01%** of total daily dose
- **SDWA requires that:** *"…regulation of such contaminant presents a meaningful opportunity for health risk reduction for persons served by public water systems"*
- **US EPA has NOT regulated NDMA**

"Regulation" Regardless of Relative Exposure



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- **SDWA requires that:** *"…regulation of such contaminant presents a meaningful opportunity for health risk reduction for persons served by public water systems*"
- **US EPA has NOT regulated NDMA**

Prevalent Myth 5

NO SAFE LEVEL for some drinking water contaminants

No Safe Level for Some DW Contaminants

What does Safe Drinking Water Mean?

- **The Environmental Management and Protection Act, 2010:**

Duty to provide safe drinking water. *33. Every person responsible for a waterworks that is used to provide water intended for human consumption shall ensure that the water supplied by the waterworks is* ***safe*** *for human consumption.*

- **Neither the Ontario Safe Drinking Water Act (SDWA) nor U.S. SDWA define "*safe*" or "*safe* drinking water"**
- **WHO DW Guidelines define "*safe* drinking water" as water that "**does not represent any significant risk to health over a lifetime of consumption…."
- **Walkerton Inquiry Part 2: A Strategy for Safe Drinking Water stated: "**to ensure that Ontario's drinking water systems deliver water with a level of risk so negligible that a **reasonable and informed person** would feel "*safe*" drinking the water"

No Safe Level for Some DW Contaminants

Why is the No Safe Level Theory Inaccurate?

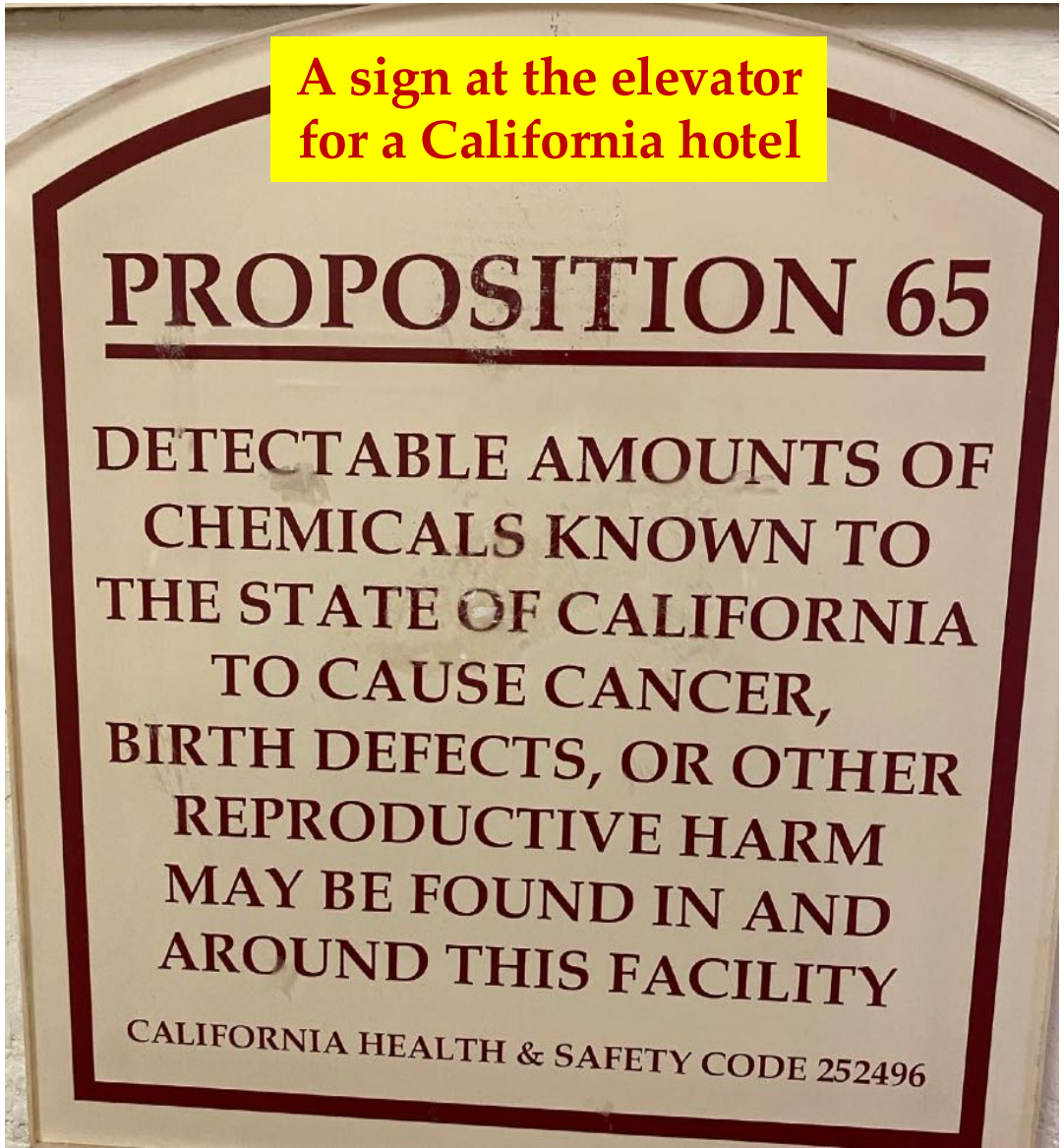
- **Requiring Absolute Zero Risk to Define "*Safe*" is Not Defensible**
- There can be contaminants so toxic at trace levels that a "***safe***" level has not yet been established by evidence
- Lead (Pb) poses a threat to cognitive development in young children, a very difficult adverse effect to study and characterize
- The **LEAD** Guideline for Canadian Drinking Water Quality **(**FPT-CDW 2019**)** states: "*The consensus in the scientific literature is that a safe level of exposure to lead in children* ***has not been identified***"
- That statement is defensible because it means that available scientific methods for defining a threshold below which adverse health effects **do not occur** are not sensitive enough to **reliably define a low "*safe*" level**

No Safe Level for Some DW Contaminants

Why is the No Safe Level Theory Inaccurate?

- The U.S. CDC has monitored blood levels of lead in children for decades and has defined a Reference Level for lead in blood (BLRV) of 3.5 µg/dL
- Detection limit for lead in blood is 0.07 µg/dL, 2% of BLRV
- This detection limit for lead in blood corresponds to 35×10^{14} (3,500,000,000,000) atoms of lead per dL
- There is an enormous range between "*detectable*" and absolute zero
- Not being able to detect a threshold below which there is no adverse health effect is a function of the capability of health effects studies AND the enormous range of possible contaminant concentrations above ZERO
- U.S. EPA proposed in April 2023 to prohibit water utilities from describing their drinking water as **safe**, under the Safe Drinking Water Act (Hrudey 2024)

No Safe Level for Some Contaminants



A sign at the elevator for a California hotel

What should you do with this warning?

- ❑ Stop breathing?
- ❑ Write your will?
- ❑ Run?

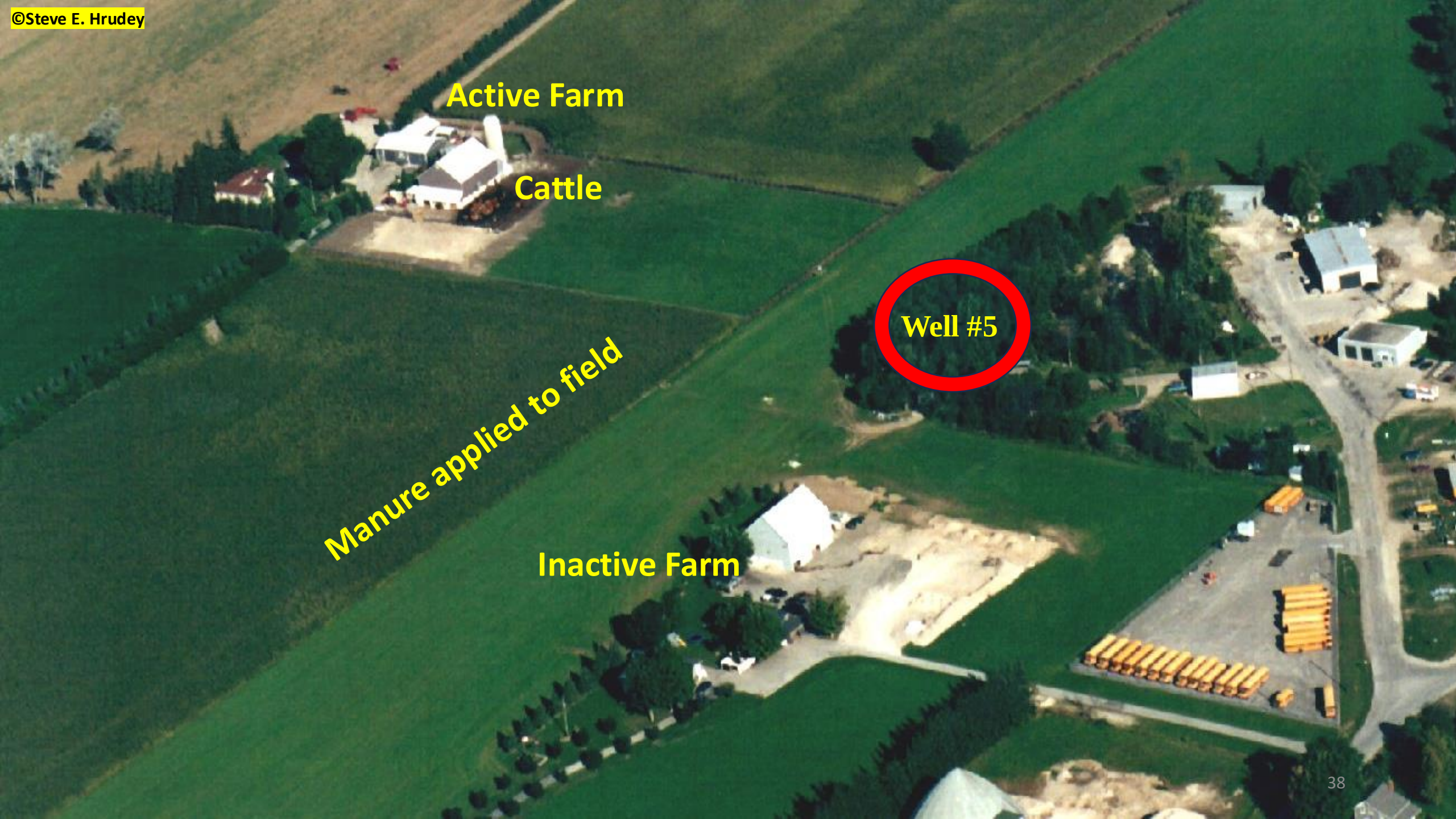
Risk Management / Risk Communication should inform the public not confuse them

Turning Hindsight Into Foresight – Learning from Experience

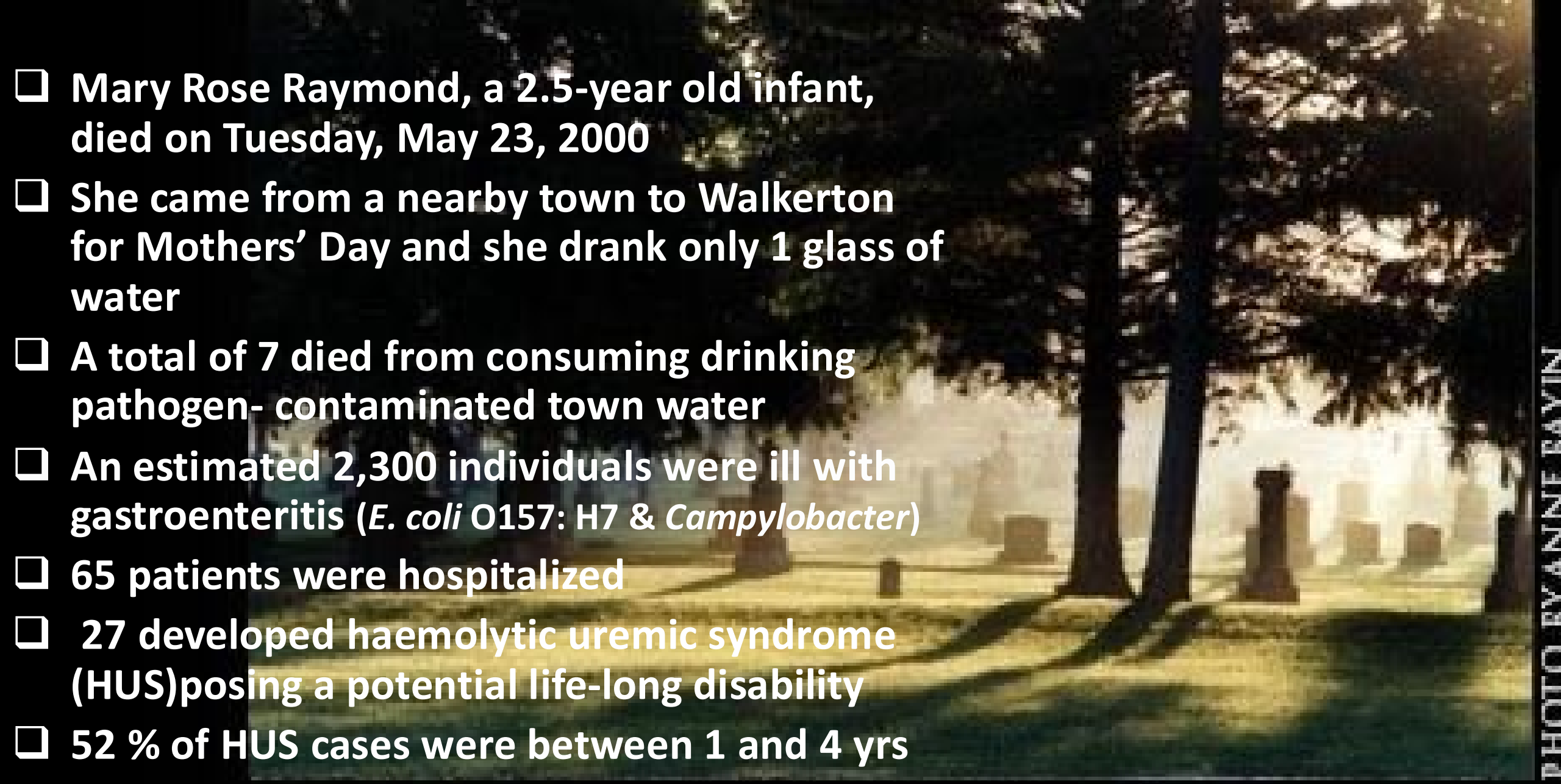
- Fortunately, we do know how to ensure safe drinking water
- The challenge is to consistently do what we know how to do
- By adopting and maintaining an effective multiple barrier approach, failures can be prevented
- Effective risk management requires learning from experience

Walkerton, Ontario, May 2000

- This disaster happened 24 years ago, so anyone under 42 was not yet an adult when it happened.
- Ontario was the unquestioned leader in water management in the 1960s
- Decades of complacency and neglect laid the foundations for tragedy
- Hydrogeologist commissioning Well 5 in 1978 warned that it was vulnerable to agricultural waste contamination
- Effective chlorination was essential
- Operators & management did not understand need for disinfection and what chlorine residual informed about possible contamination


©Steve E. Hrudey
Active Farm
Cattle
Well #5
Manure applied to field
Inactive Farm

- Mary Rose Raymond, a 2.5-year old infant, died on Tuesday, May 23, 2000
- She came from a nearby town to Walkerton for Mothers' Day and she drank only 1 glass of water
- A total of 7 died from consuming drinking pathogen- contaminated town water
- An estimated 2,300 individuals were ill with gastroenteritis (*E. coli* O157: H7 & *Campylobacter*)
- 65 patients were hospitalized
- 27 developed haemolytic uremic syndrome (HUS)posing a potential life-long disability
- 52 % of HUS cases were between 1 and 4 yrs



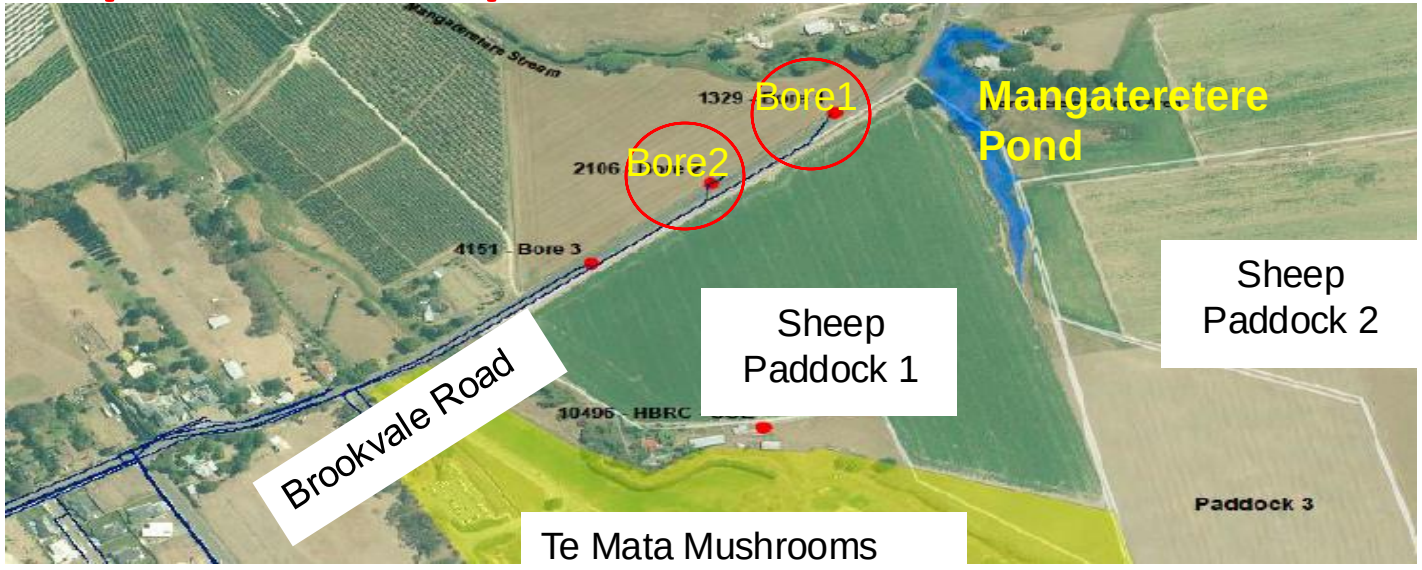
Walkerton Cemetery

What caused Walkerton?

- The water operators were long-term residents of Walkerton and those who died or were violently ill were their neighbours in the community
- The operators did not understand that pathogen contaminated drinking water could kill consumers
- They only chlorinated because they were told to, but had no idea about serious health risks from failing to disinfect
- They did not understand that monitoring chlorine residual could tell them if water was contaminated
- If the operators were incompetent, what does that say about their management and the regulators?

Havelock North, New Zealand, August 2016

- North Havelock, Hawkes Bay, New Zealand had a regional exposed population of more than 14,000
- 6,300 to 8,300 cases of illness including 42 hospitalizations and 3 cases of Guillain-Barré syndrome
- 4 deaths among elderly patients caused by *Campylobacter* infection
- The water supply from shallow Bores #1 and #2 located along Brookvale Road among **sheep and cattle pastures** and a **mushroom farm**



Havelock North, New Zealand - August 2016

- Bore #1 was screened from 11.4 to 17.4 m in an aquifer classified as "***secure***" by then existing NZ DW Standards
- "***secure***" groundwater was delivered without chlorination
- When well pump was on, pond would dry up

Well head pump was below ground level and would flood during periods of high rain. Not sealed!

Havelock North, New Zealand - August 2016

Heavy rain flooded sheep pastures that caused sheep manure-contaminated water to drain to Mangateretere Pond - a widening of a small stream 90m from Bore #1 – contaminating the shallow aquifer

The sanitary status of the pumphouse for the Havelock North shallow wells

Is it any surprise that this water supply led to a disastrous outbreak?

Perhaps the only surprise is that it took a decade since their previous outbreak to kill some of their consumers

Some Havelock North Inquiry Findings

Failures in Risk Management and Complacent Culture

- Livestock faecal risk was not recognized as a serious health risk
- A 2008 Water Safety Plan categorized the risk of contamination of surface sources was "*unlikely*" and the **consequences as only "*moderate*"**
- Failed to investigate or determine source of *E. coli* contamination
- Bore head contamination risks were mentioned over, and over again, between 2009 and 2014 with nothing done about it
- Local government was reluctant to chlorinate or maintain chlorination beyond a bare 3 day minimum after an *E. coli* detection
- **OVERALL, where were the operators who MUST KNOW BETTER?**

Northampton, England 2008

Pathogen contamination can happen to anyone!

- This system had been operated for 52 years, as of 2008
- Source water was treated by:

  pre-ozonation, chemical coagulation, clarification, filtration, ozonation, GAC adsorption, chloramination and buffering for control of lead
- The raw water was found to be generally free of *Cryptosporidium* oocysts
- Performed voluntary continuous *Crypto* monitoring
- Discovered oocysts at 8:00 PM one evening
- Rechecked then called a boil water advisory by 3:30 AM

Northampton, England 2008

Outbreak Experience: June – July 2008

- Inspection revealed wire mesh covers for two ventilation c GAC backwash tank and one access hatch were damaged
- The gaps provided access for "*small*" animals to the GAC b
- On the evening of June 27, a small, "*fresh*" carcass of a rab below the inlet pipe of the chlorine contact chamber
- Rabbit infected with a new strain - *Cryptosporidium cuniculus* - that was also found in treated water
- Caused 22 lab-confirmed cases and an estimated **422 Crypto cases** among 270,000 consumers
- Much larger outbreak would have occurred if not for rapid response
- Total cost to Anglian Water = £4.9 million (US$9.7 million)

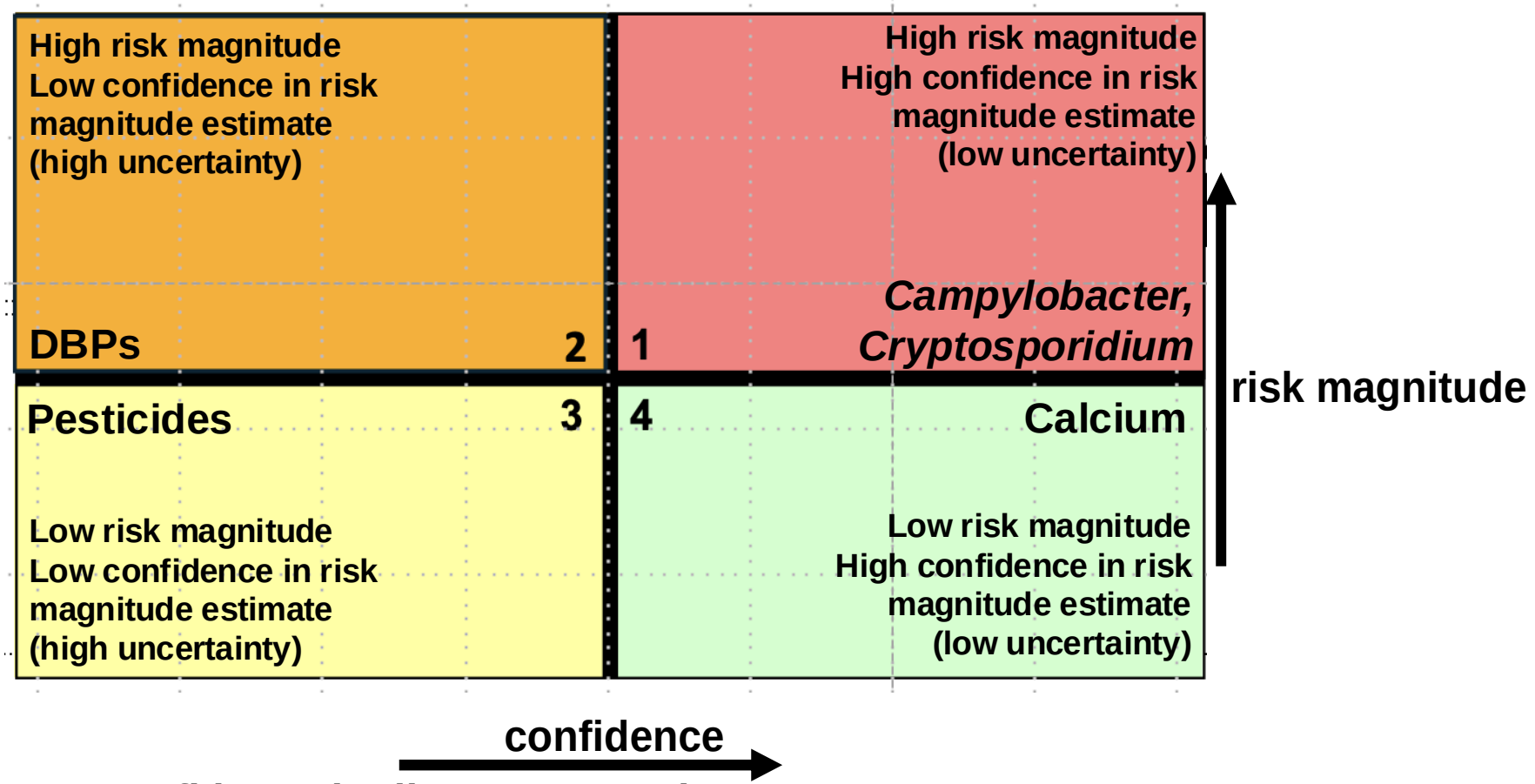
Recent Large DW Outbreaks (>1,000 cases) in Developed Nations - including fatal outbreaks

Year	Location	Pathogen	Cases	Hospital Admissions	Death	Comments
2010	Ostersund, Sweden	*Cryptosporidium*	27,000	~270	-	Sewage from a single family contaminated the water intake
2011	Skellfetea, Sweden	*Cryptosporidium*	18,500	N.R.	-	Likely that community sewage contaminated the water intake
2012	Ellasona, Greece	*Cryptosporidium*	3,600	N.R.	-	a spring was contaminated
2012	Darfield, New Zealand	*Campylobacter*	828-1,987	46	-	Livestock contamination, chlorination inoperative
2013	Baker City, OR, USA	*Cryptosporidium*	2,780	N.R.	-	Livestock contamination, no Cryptosporidium barrier
2015	Prague, Czech Republic	Norovirus	11,500	33	-	Sewage infiltration of mains repair
2016	Havelock N. New Zealand	*Campylobacter*	5,500	45	4	Livestock contaminated shallow wells – no chlorination
2019	Askoy, Norway	*Campylobacter*	>2,000	76	2	Untreated water contaminated in storage – wildlife source

Practical Actions for Safe Drinking Water

- **Quality Management for Risk Management**
  - During 1990s, many came to realize that an emphasis on monitoring treated water quality for quantitative guidelines or regulations is not preventive
  - Need a focus on operational confirmation that multiple barriers are functional
  - Australian DWG have provided this approach since 2004
- **Drinking Water Safety Plans**
  - In parallel with ADWG, WHO introduced the Water Safety Plan approach in 2004, that is now adopted to varying degrees in over 93 countries (see Hrudey et al. 2024)
- **Recognizing priorities**
  - Logical, high-level approach to to ensure effective risk management

Priorities for Health Risks in Drinking Water



Confidence in disease causation at or below **levels** found in drinking water

Risk magnitude = probability x consequences
At or below levels which **have occurred** in drinking water
Higher prevalence will increase probability

Hrudey et al. 2012. Managing uncertainty in the provision of safe drinking water.

Concluding Thoughts

- More stringent guideline numbers will **NOT** ensure safe drinking water
- Meeting the regulations of that time **could have prevented** Walkerton
- Regulation needs to focus on achieving competence at all levels
- A regulatory focus on monitoring for multiple chemicals **must not distract** from the **greatest risks – microbial pathogens**
- Regulation needs to focus on good practice, e.g., **DWSP**s
- **What Do YOU know about YOUR plant that COULD go wrong?**

The Bottom Line

You can have cheap water

Or you can have safe water

But you cannot have cheap, SAFE water!

Public Health Risk Assessment and Risk Management for Safe Drinking Water

Steve E. Hrudey

THE GROUNDWATER PROJECT

Should appear before Nov 15

Search:

https://gw-project.org/books/

THE GROUNDWATER PROJECT

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Thank you for your attention

Questions?

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