Naturally produced noxious chemicals & toxins in the Great Lakes

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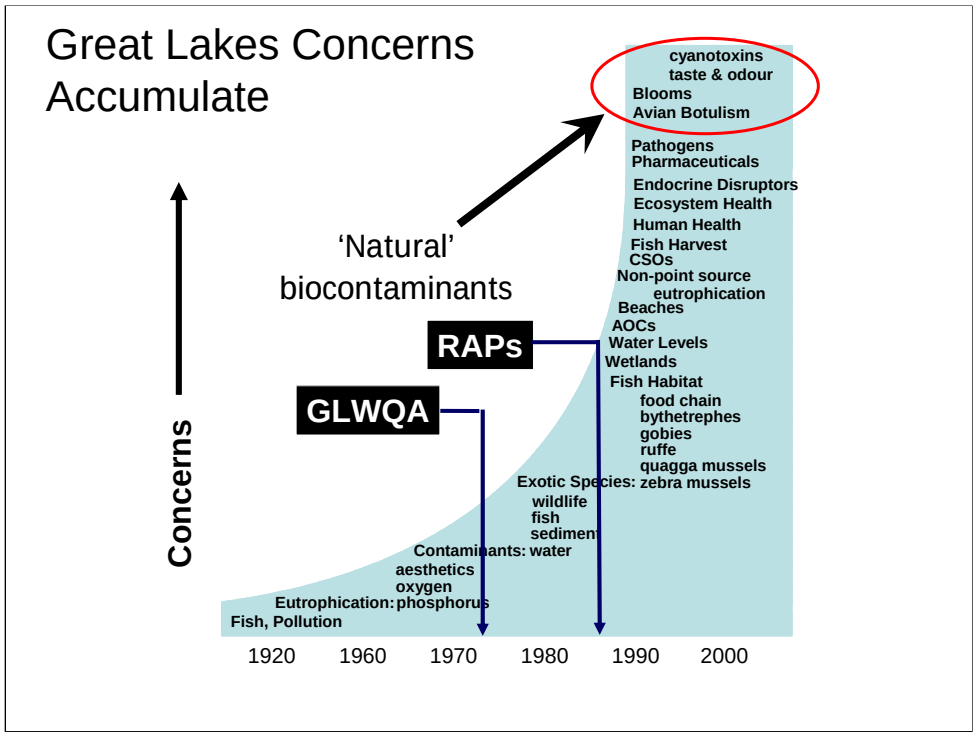
Good morning! It is a great pleasure to be here, and act as spokesperson on behalf of my coauthors, Greg Boyer, from SUNY (NY), and Elizabeth Hinchey Malloy, from Illinois-Indiana Sea Grant (and her colleagues).

I will be talking about '**Naturally produced noxious chemicals & toxins**'. It is a huge topic which can only be briefly touched on in this talk, but hopefully we will stimulate many of you to attend this afternoon's 2 workshops on these important issues.

This slide shows a **satellite image of a huge algal bloom in Lake Erie,** such disturbing images are becoming increasingly common on the web and other media.

It should be noted that the image was **taken on November 20, 2003** – very late in the so-called growing season

These **outbreaks, and their direct and indirect effects**, are closely linked to much of today's topic.



As we are all aware, the Great Lakes are **vital ecosystems and a major freshwater resource** for some 40m US-and Canadian people, (..and counting up!)

This Figure illustrates the **long history of human use and misuse** of this freshwater resource, which **continues today despite international lake-wide remedial and management action.**

The **effects** of many of these impacts have been **cumulative**, and **accompanied by new concerns.**

In particular, **two very conspicuous biological phenomena - avian botulism & algal blooms are an increasing** –are not only **highly visible to the public, but also have major ecological and socio economic impacts**

Clearly **traditional nutrient-productivity management models based on lakewide averages fail to address these oubreaks,** likely as a result of **the shift in inshore-offshore exchange** discussed in the previous talk

I will focus on three concerns related to these phenomena:

- Taste - odour
- Algal toxins
- Botulism

Outline

Naturally produced noxious chemicals & toxins

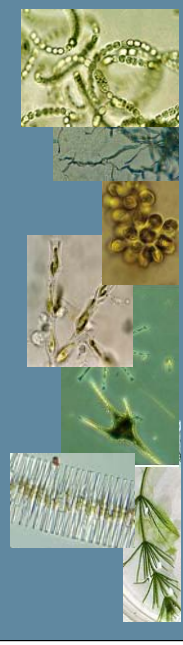
1. Taste & odour
2. Algal (cyanobacteria) toxins
3. Avian Botulism
   - Significance (effects, concerns etc.)
   - Characteristics (chemistry etc.)
   - Organisms
   - Great Lakes
     - where, when & how affected
     - monitoring/control/management
     - ecological/socioeconomic implications

This presentation will provide an overview of these three categories of 'noxious chemicals and toxins', and briefly introduce:

- their significance,
- their general characteristics
- their occurrence and levels in the Great Lakes
- monitoring/management programmes
- their ecological and socio economic implications

taste & odour

- major socio economic costs.
  water, food, beverage, aquaculture, tourism, recreation
- management/control issue.
  - – difficult to trace.
  - – resistant to treatment.



The first of these, **Taste and odour has a nasty habit of arriving at our taps.** It is an important

socio economic issue with substantial **costs to the water industry (millions annually!)…..and to food, aquaculture, tourist/recreation industries**

…an important **management and control issue for several reasons:**

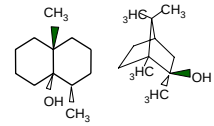
It is hard to trace. T&O can **originate from plankton, benthos and other 'hidden' zones.** It is caused by **volatile organic compounds** produced by a **diversity of species** (some pictured to the right),

The compounds are **highly potent and detectable** to humans at **very low levels** (ng/L).

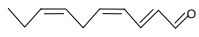
Some of the most **problematic compounds are stable** and **persist in the open water**, these compounds are also **resistant to conventional treatment**

Chemistry & taste

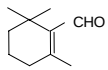
Terpenoids *('earthy/muddy/musty')*
→ **geosmin, 2-MIB**
→cyanobacteria, actinomycetes, fungi



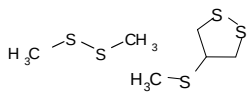
Fatty acid derivatives *('fishy'/'oily'/'rancid'/'cucumber')*
→ **decatrienal,** nonadienal, heptadienal
→ chrysophytes, diatoms, flagellates



Pigment derivatives *('hay'/'tobacco'/'floral')*
→ **β-cyclocitral,** β-ionone, etc
→ cyanobacteria



Sulphur compounds *('pungent / sulphurous /swampy / rotten')*
→ **thiols, sulphides**
→ cyanobacteria, *Chara*



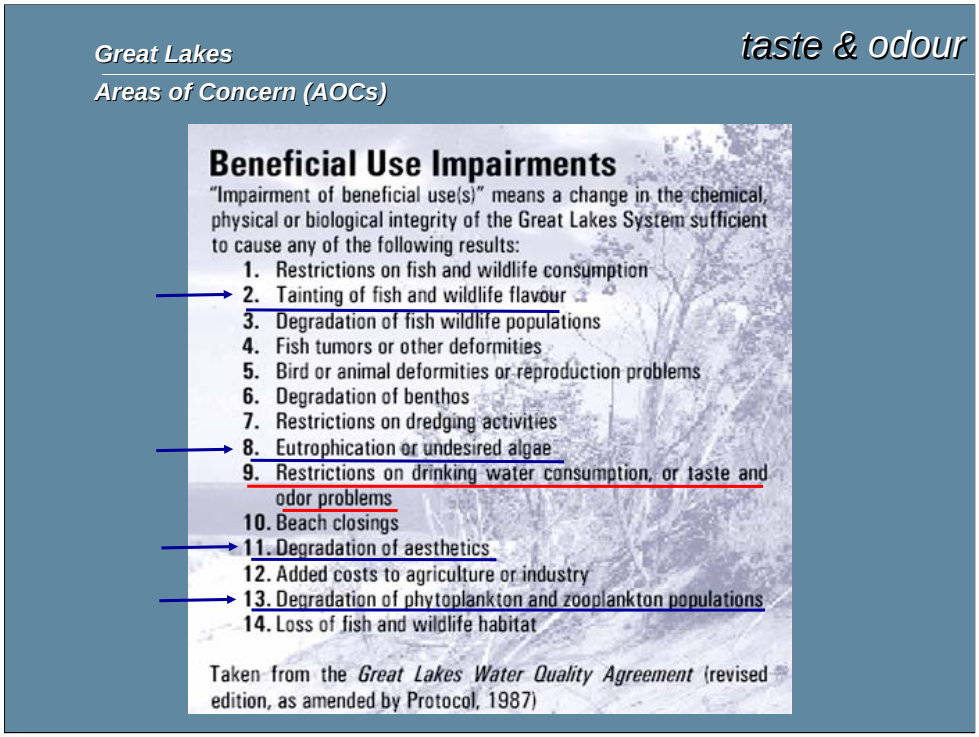
Aquatic organisms produce **numerous volatile compounds**, but only a few are consistently problematic. These fall into **4 main groups**, shown here

Terpenoids – particularly geosmin and 2-methylisoborneol, with a potent **muddy odour** common in **spring runoff or some summer blooms**, caused by some **cyanobacteria or bacteria** and **fungi**

Polyunsaturated fatty acid derivatives e.g. 2,4,7-decatrienal , with f**ishy/rancid** or **cucumber** odours (usually in **spring, fall,** or **under ice**)

Pigment derivatives with a **hay/tobacco/floral** odour e.g. β-carotene breakdown products (β-cyclocitral), produced by cyanobacteria

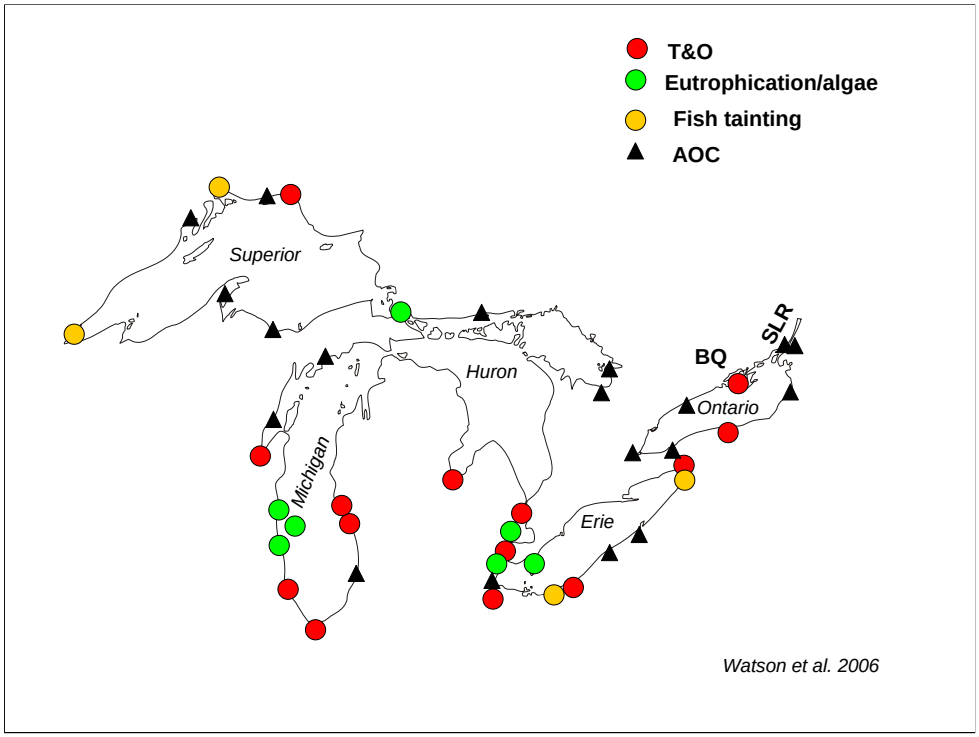
Sulphur compounds (thiols, mercaptans etc) with**swampy/ garlic/ sulphur odours**, often produced during anaerobic **degradation of plant & animal material**



Taste and odour is a key issue in the Gt. lakes, and not recent.

It was listed among **the 14 beneficial impaired uses** in AOCs *(underlined in red on this slide); note this is based on* **treated drinking water in municipal supplies**

T&O is related to **several other listed impairments**, usually associated with eutrophication *(underlined in blue).*



This map from a <u>recent EC report</u>, shows the distribution of **AOCs in the Gt Lakes** (**triangles**) and those which are affected by **T&O (in red)** or **related impairments** (Green and yellow)

<u>Some key points should be considered:</u>

<u>First</u>, taste and odour is **listed by over a third of the AOCs**, particularly in the **Lower Lakes**, where incidents are **most frequent in the summer and fall**, coincident with heightened biological activity.

<u>Second,</u> <u>There are no standards or guidelines for T&O</u>.

Furthermore **even though this is a listed impairment**, there has been **<u>little or no systematic or direct monitoring or quantification of the issue</u>**, **<u>instead</u>**, it is usually assessed by RAPS (i.e. **'deduced')** using '**proxy' measures (like nutrient levels).**

<u>Third,</u> this impairment is based on **treated drinking water in large municipal supplies**, and does not include

-small users or communities with **rudimentary / no treatment**

-many **areas not listed as AOCs** with **significant outbreaks** (eg the entire **NW shoreline of L. Ontario east** of **Colbourne** *(nr. 'BQ' on map)*

-**<u>source waters</u>!** And **many beaches and waterfronts affected by rotting mats** of algae and aquatic plants

Cyanobacteria Toxins

J. Great Lakes Res. 26(3):241–249
Internat. Assoc. Great Lakes Res., 2000

Isolation and Characterization of Microcystins, Cyclic Heptapeptide Hepatotoxins from a Lake Erie Strain of *Microcystis aeruginosa*

Scott M. Brittain[1], Jim Wang[3], Lisa Babcock-Jackson[2], Wayne W. Carmichael[1]*, Kenneth L. Rinehart[3], and David A. Culver[2]

FIG. 2. **Microcystis aeruginosa** *field collection sites: Put-In-Bay, Lake Erie, Ohio. "X" marks Hatchery Bay where > 1 µg/L microcystin was detected in October 1995.*

- Cyanobacterial toxins first reported in Lake Erie in the mid-1990s
- Blooms can be large and easily visible from space.
- Complex problems with many toxins and many toxic species involved.

The next section will cover the topic of **cyanobacteria toxins.**

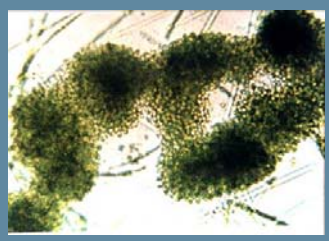
These have received increasing attention since the published report of a large heptatoxic bloom of the cyanobacterium *Microcystis* in L. Erie in the mid 1990s (referred to on the left).

These are large blooms and easily visible from space. *In the top right hand corner* is a false colour image of the W. basin of Lake Erie, clearly showing the emergence of a large cyanobacterial bloom from the Maumee River region.

In response to this initial bloom and other toxic events in L. Huron and L. Ontario, a number of large monitoring programs were initiated to address the occurrence and toxicity of these outbreaks. This includes a large regional program for Monitoring and Event Response of Harmful algal blooms in the lower Great Lakes, The Great Lakes Environmental Research Laboratories Oceans and Human Health Initiative, and Environment Canada program

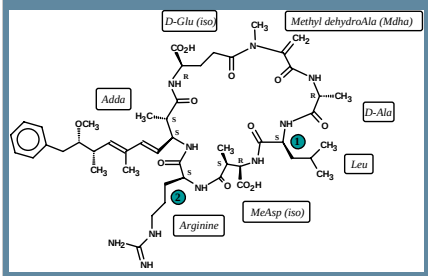
<u>Cyanobacterial toxins is a complex problem</u> with > 20 different species and 200 different toxins identified in freshwater systems. In the Great Lakes, the issue has now expanded beyond simple blooms of *Microcystis* in the W. basin of Lake Erie to include all five lakes and the St Lawrence River.

<u>In the next few slides, I will briefly summarize the major toxins found in the Great Lake ecosystem</u>



Microcystins

- *Microcystis aeruginosa*
- "non" (low) -N fixer.
- Widespread
  - Also produced by a number of other cyanobacteria species.



Peptide Toxins:
90+ structural variants
+ 200 other related compounds: nodularins, anabaenapeptins, etc.

Microcystins are hepatotoxic
LD-50: 25-60 μg kg^{-1}
"fast death factor"
Potent tumor promoter

The **most common** cyanobacteria toxins belong to a class of **hepatotoxic peptides** known as **microcystins *(shown here).*** Microcystins, as the name suggests, are commonly produced by species in the cyanobacteria genus ***Microcystis (shown above)*** from which they were first identified. ***Microcystis*** is **common** in the summer/fall phytoplankton of **eutrophic systems**.

Unfortunately the situation is not that simple. Individual **strains** of *Microcystis* **may or may not be toxic**, they may **change toxicity** with **environmental conditions**, and microcystins may be produced by a **several other cyanobacterial species simultaneously**.

Therefore management plans that focus on a single species are likely to fail.

Microcystins are **hepatotoxic.** They are a **potent tumor promoters** and have been associated with human liver tumors in Chinese studies.

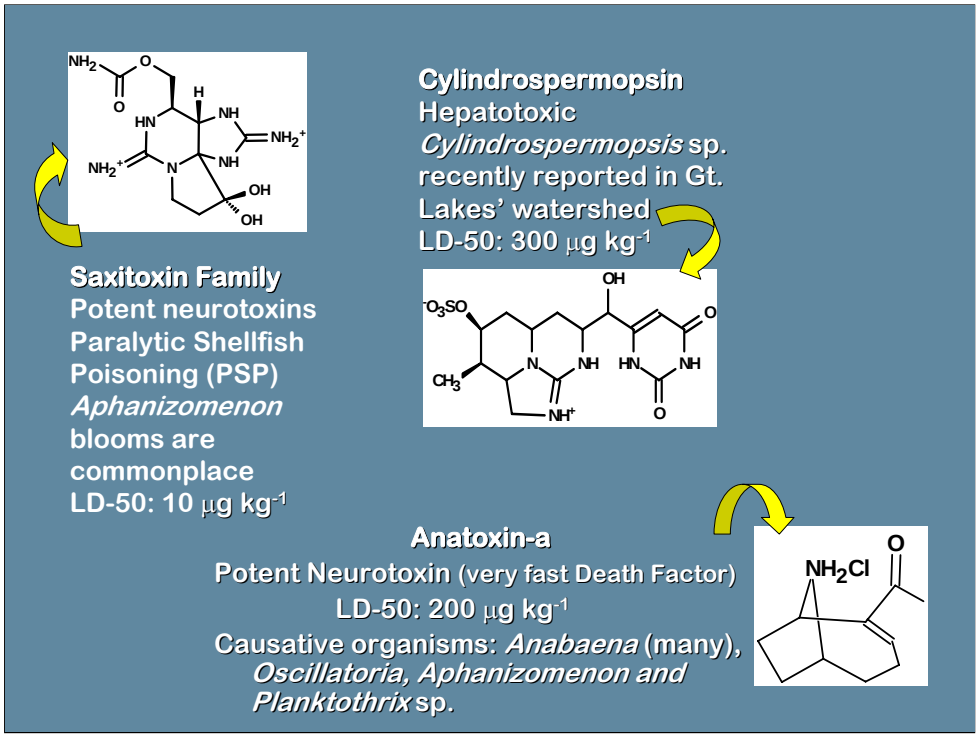
They are **resistant to degradation**, and can **bioaccumulate** in the food chain.

The **World Health Organization** has issued an advisory level of **1 ug /L in drinking water** and a **recreational** contact level of **10 ug/L**.

Health Canada has adopted a similar level of **1.5 ug /L** for drinking water.

The toxin is currently on the Critical Contaminant List of the US-EPA and under investigation for issuance of a US advisory and or regulatory level.

Concentrations above **1 ug / L are routinely exceeded in the w. basin of Lake Erie, the Bay of Quinte (L. Ontario), and in Saginaw Bay (L. Huron).**



In addition to the microcystins, several **other cyanobacterial toxins may be present in the Great Lakes, *illustrated in this slide***. The **potent neurotoxin anatoxin-a *(shown at the bottom)*** has been measured in both Lake Ontario and Erie at concentrations associated with animal fatalities in other systems.

Other potential toxins include **cylindrospermopsin *(top)***, a **hepatotoxin** produced by *Cylindrospermopsis* species. These taxa are more commonly found in warmer waters such as Florida but have recently been reported in smaller lakes in the Great lakes watershed and some of the near shore embayments of Lake Erie.

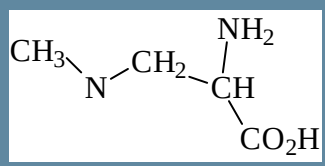
Other toxins such as the **saxitoxins *(seen at the left)*** may also be present**. These are potent neurotoxins** more commonly associated with **paralytic shellfish poisoning in marine systems** but also known to be **produced by selected freshwater *Aphanizomenon* species**.

The distribution, occurrence, persistence and potential health risks from these very potent toxins remain to be elucidated**.**

New Threats?

BMAA
Potent neurotoxin
associated with flying foxes & Parkinsonism dementia.

- many cyanobacteria species (aquatic & terrestrial) including
Microcystis, Anabaena, Aphanizomenon

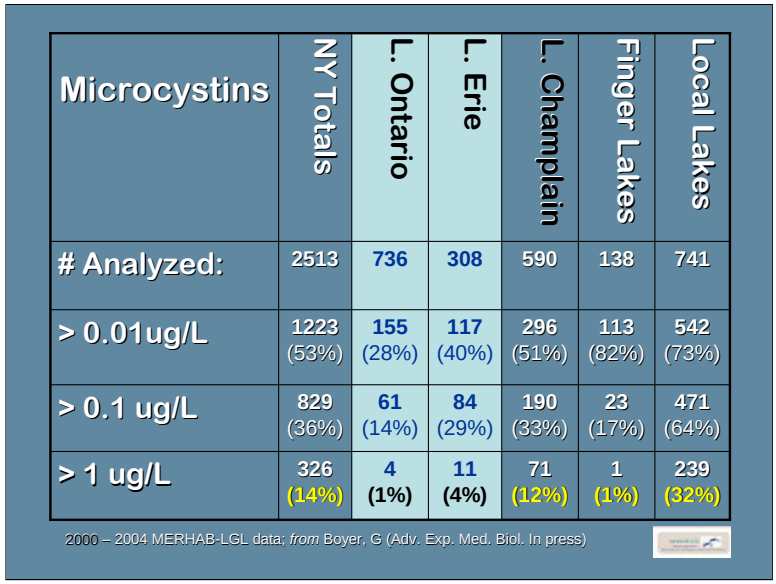


2006 bloom in Hamilton Harbour illustrating the high biomass that can occur in these blooms.

In **addition to the known cyanobacterial toxins** discussed in the previous section, Cyanobacteria produce numerous other **biologically active secondary metabolites**. **New toxins and potential health threats continue to be identified**.

One example is a new class of toxins known as the **beta methyl amino acid or BMAA**, This new class of **neurotoxins was recently linked with neurological symptoms associated with Parkinson's dementia**. BMAA is produced by **numerous species of Cyanobacteria**, **readily bio-accumulates** in the food chain and is **expected to be widespread through the Great Lakes**. Efforts to monitor its levels and abundance are currently underway in both the US and Canada.

The **inset shows a photograph** of a recent and prolonged cyanobacterial bloom in **Hamilton Harbour**, an eutrophic AOC at the W. end of Lake Ontario, and illustrates the **high surface densities** that these blooms can achieve



Microcystins	NY Totals	L. Ontario	L. Erie	L. Champlain	Finger Lakes	Local Lakes
# Analyzed:	2513	736	308	590	138	741
> 0.01ug/L	1223 (53%)	155 (28%)	117 (40%)	296 (51%)	113 (82%)	542 (73%)
> 0.1 ug/L	829 (36%)	61 (14%)	84 (29%)	190 (33%)	23 (17%)	471 (64%)
> 1 ug/L	326 (14%)	4 (1%)	11 (4%)	71 (12%)	1 (1%)	239 (32%)

2000 – 2004 MERHAB-LGL data; *from* Boyer, G (Adv. Exp. Med. Biol. In press)

The previous slides paint a dire situation for the Great Lakes. However it is important to put these numbers in perspective. The table on this slide summarizes 2000-2004 MERHAB data. Focusing L.Ontario and Erie **(highlighted in blue),** several key points can be made.

First. In the absence of clearly defined regulatory levels, **what constitutes a toxic event is often in the eye of the viewer.**

Modern tools for toxin analysis are extremely sensitive and detectable microcystin levels were measured in ~50% samples. This is in agreement with other studies out of the EU and culture collections. The Table also illustrates that the Great Lakes are not very different from other large waterbodies in New York, including the Finger Lakes and Lake Champlain.

Second, while high toxicity events are relatively rare, they do occur in all these lakes. High events exceeding the WHO guidelines for drinking water were found in 1% of the samples from L. Ontario, and 4% samples from L. Erie.

These numbers are especially disturbing because **the monitoring program was not targeted towards blooms**, but rather a randomized design to sample toxins regardless if Cyanobacteria were present. More targeted studies focusing only on bloom events suggest that higher toxin levels may occur much more frequently in those situations.

This can be **complicated by windborne accumulation.** Many cyanobacteria control their buoyancy and can rapidly accumulate on the surface of the water as in the last slide. Toxin levels in these scums have **exceeded 1000-5000 ug/L**.

You cannot determine if a cyanobacterial bloom is toxic by looking at it, and you must treat any bloom of potentially toxic species as toxic unless one knows otherwise.

General Conclusions:

- Microcystins: fairly common in Lake Ontario, Lake Erie & Lake Huron
  - Esp. more eutrophic embayments
- Other toxins e.g. Anatoxin-a & cylindrospermopsin are present in the Gt. Lakes.
  - Much lower concentrations than microcystins
  - Occurrence unrelated to microcystin-containing blooms
- Number of different toxin producers

  → management strategies focused on single species **unlikely to be successful**

General Conclusions for this section are:

Microcystins: fairly common in Lake Ontario, Lake Erie & Lake Huron, esp. more eutrophic embayments

Other cyanobacterial toxins such as the neurotoxin Anatoxin-a are present in the Great Lakes.

> These are found at much lower concentrations than microcystins, but their **health effects on humans and ecosystems are unknown.**

There are a number of different toxin producers; thus management strategies focused on single species **unlikely** to be successful

Botulism in the Great Lakes

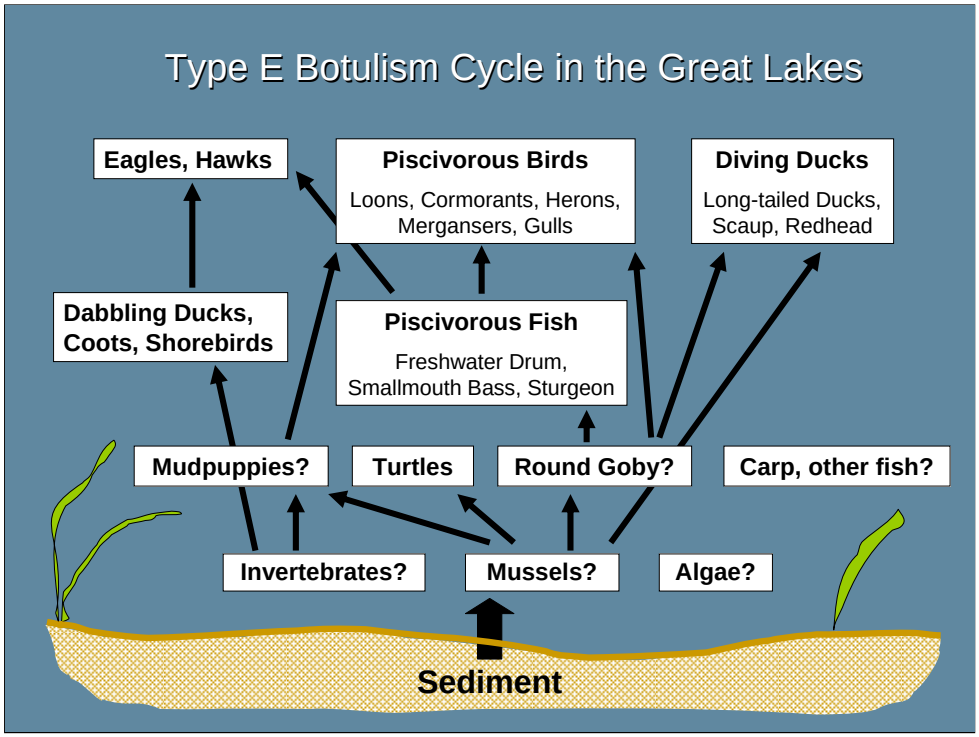
Photo Credit: Patricia Nelson, NYS Department of Environmental Conservation

Now I will discuss another biotoxin that is an important issue: **botulism,** which has caused numerous bird and fish kills in the Great Lakes.

Botulism is a **toxin** produced by the **bacterium *Clostridium botulinum* Type E.** These **ubiquitous anaerobic bacteria** are present in **soil and sediments** of aquatic systems. The toxin causes a **neuroparalytic disease** that is **transmitted through the ingestion of contaminated food**.

Botulism can result from the **ingestion of the neurotoxin or from colonization of the intestinal tract by the bacteria**.

Animals, **especially fish-eating birds, ingest the bacteria in their diet**, become paralyzed by the botulinum toxin, and **often die**, as graphically **illustrated in this slide. Their carcasses then become sites for more *Clostridium* growth**.

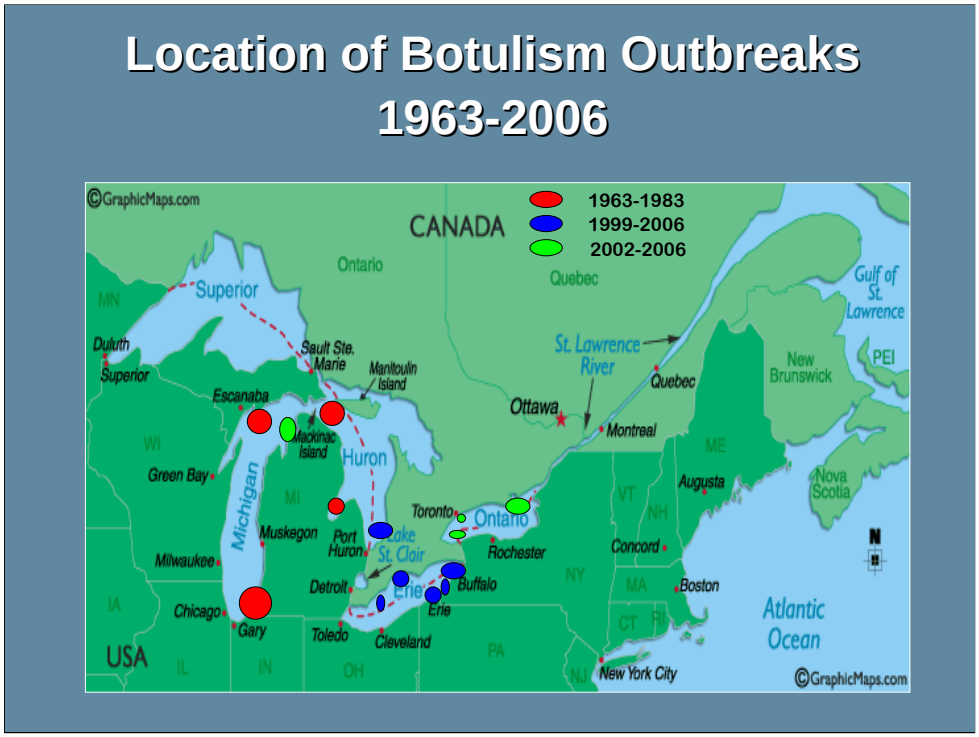


This slide depicts our **current understanding of the Type E botulism cycle in the Great Lakes**. Not all of the pathways are understood, nor are all of the environmental conditions required for an outbreak to occur.

It is known that the **botulism-producing bacteria thrive under oxygen-depleted conditions**, such as anoxic sediments. **Warming lake** temperatures may also **contribute to increased bacterial growth.**

Type E botulism is **unusual because it can concentrate in fish and affect a wide spectrum of wildlife**. Dead or dying fish containing botulism attract **scavengers like gulls and terns** that die when they eat the contaminated fish. **Merganser ducks and loons ingest the toxin by eating live fish.**

Botulism impacts many components of the food web - waterfowl , fish and invertebrates. But the full ecosystem impacts are yet unknown.



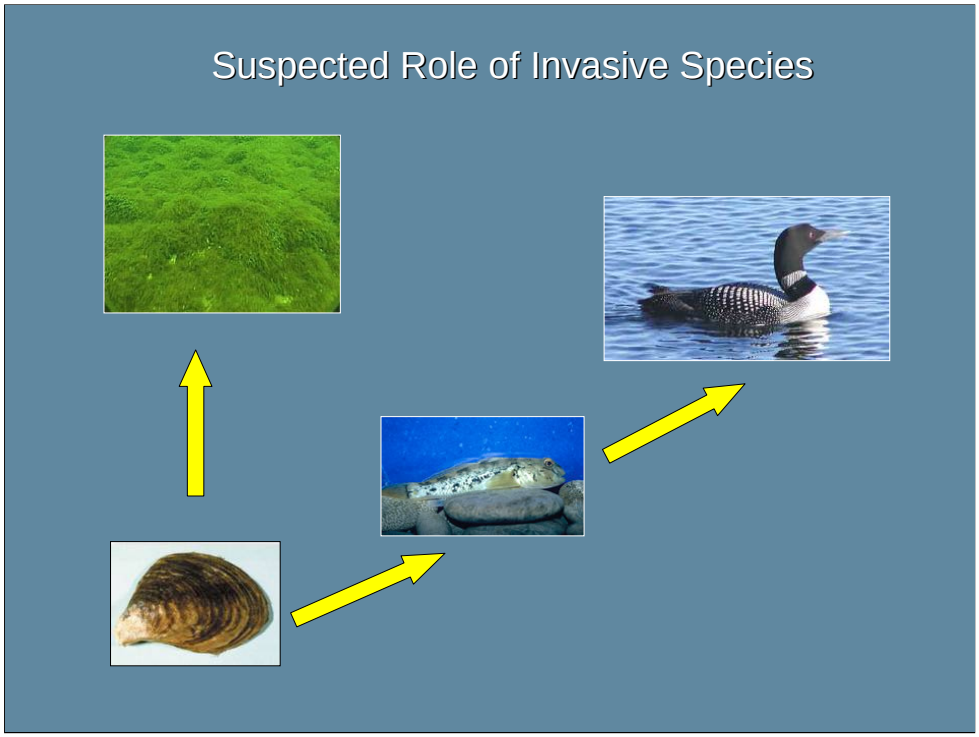
This map shows the **location of botulism outbreaks that resulted in bird kills over the last 40 years in the Gt Lakes**. Botulism was **first detected in L. Michigan in the 1960s**, with outbreaks in **Lakes Michigan and Huron until the 1980s *(red circles)***.

After **subsiding** in the late 1980s, outbreaks **re-intensified** in the **late 1990s and early 2000s in L. Huron, Erie and Ontario (blue circles)**

Although the past few summers have experienced fewer botulism-related waterbird die-offs**, it appears that they have increased in 2006**. This summer, die-offs were reported in Lake Ontario. **In the past two weeks, Lake Ontario and Erie** have been extremely hard hit**. The Lake Ontario bird mortality in this event was among the largest on record, and affected common loons, grebes, and merganser ducks**. It was especially severe for migrating loons in the E end of Lake Ontario.

It is also important to note that **in 2006 botulism outbreaks returned to L. Michigan for the first time in recent history**. An estimated **2,600 waterbirds** were lost along the beaches of Sleeping Bear Dunes National Lakeshore alone. The species hardest hit were horned grebes, mergansers, common loons, cormorants and gulls.

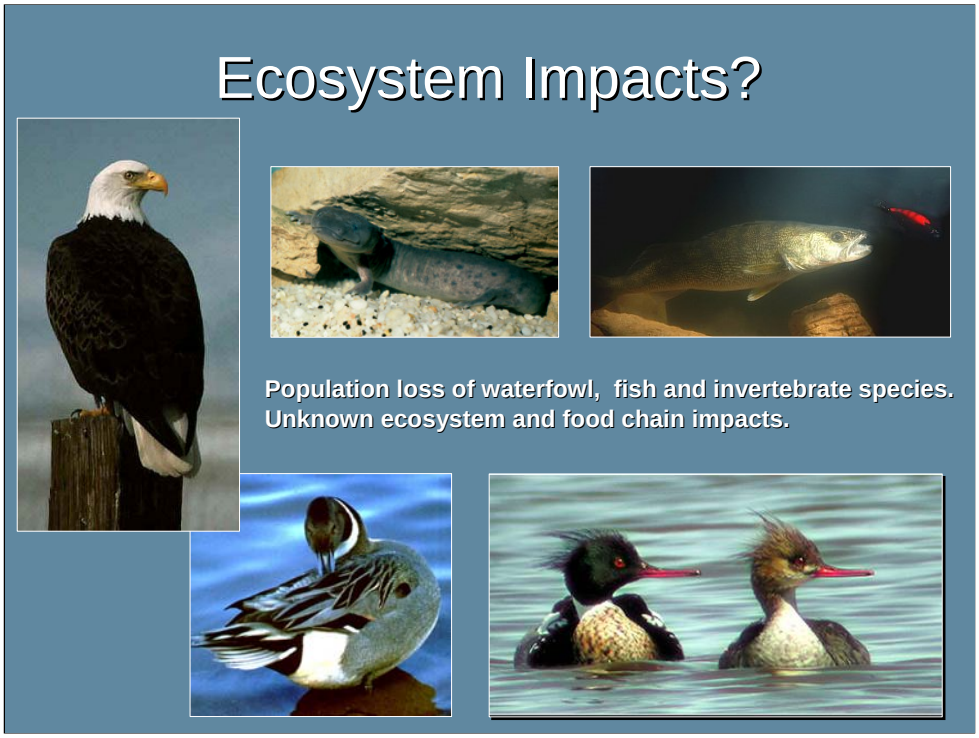
Note the general movement of the outbreaks from West to East….



I mention that trend, because **invasive species have been implicated in the progression of botulism through the Great Lakes and in the increased number and severity of outbreaks in recent years.**

It is hypothesized that the **West to East movement of botulism has tracked the West to East invasion of round gobies in the Great Lakes**. ***As diagrammed here***, **gobies feed on invasive dressenid mussels**. It is believed that the **mussels concentrate the botulism toxin as they filterfeed**. When these **mussels are eaten by gobies, the botulism is concentrated further**. The affected gobies exhibit an erratic thrashing behaviour which attract **predatory fish, mudpuppies, mergansers, loons and other birds.**

In addition, the **mussels may be creating an environment that is more favorable to botulism** production through their filtering activities which have increased **water clarity and nutrient recycling** in many areas of the Great Lakes. This **increases the growth of aquatic plants and algae such as *Cladophora***. The oxygen-deprived environments provided by their decaying biomass promotes botulism growth

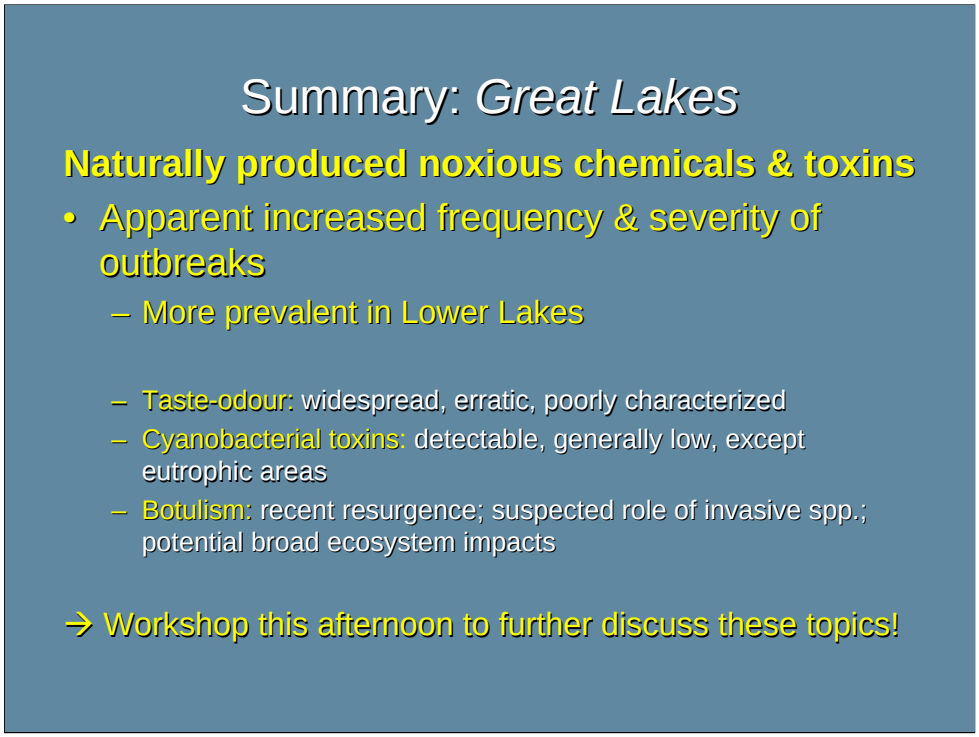


<u>So what are the ecosystem impacts of avian botulism in the Great Lakes?</u>

Many species are affected, but the major food web pathways and environmental causes remain poorly understood.

Invasive mussels and gobies are believed to play roles in the recent resurgence of outbreaks.

However, much of the complex cycle and foodweb links remain to be elucidated to better predict the occurrence and impacts of this toxin in these ecosystems.



To summarize what we know about **Naturally produced noxious chemicals & toxins in the Great Lakes:**

There is an apparent **increased frequency & severity of outbreaks** .

These appear to be **more prevalent in Lower Lakes**. This may be a reflection of the higher nutrient levels

Taste-odour: widespread, erratic, poorly characterized

Cyanobacterial toxins: detectable, generally low in the open waters but can often exceed advisory levels in more eutrophic inshore areas.

Botulism: has shown a recent resurgence suspected to be linked to invasive spp.

Both botulism and cyanobacterial toxins are likely to have broad ecosystem impacts, the nature of which we have yet to understand.

→ Workshop this afternoon to further discuss these and other topics!