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MINIREVIEW

Toxin-producing Cyanobacteria in Freshwater: A Review of the Problems, Impact on Drinking Water Safety, and Efforts for Protecting Public Health

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Cyanobacteria have adapted to survive in a variety of environments and have been found globally. Toxin-producing cyanobacterial harmful algal blooms (CHABs) have been increasing in frequency worldwide and pose a threat to drinking and recreational water. In this study, the prevalence, impact of CHABs and mitigation efforts were reviewed, focusing on the Lake Erie region and Ohio's inland lakes that have been impacted heavily as an example so that the findings can be transferrable to other parts of the world that face the similar problems due to the CHABs in their freshwater environments. This paper provides a basic introduction to CHABs and their toxins as well as an overview of public health implications including exposure routes, health effects, and drinking water issues, algal bloom advisory practices in Ohio, toxin measurements results in Ohio public water supplies, and mitigation efforts.

Keywords: cyanobacteria, toxins, freshwater, public health, drinking water safety

Introduction

Cyanobacterial harmful algal blooms (CHABs) are excessive growths of cyanobacteria (also known as blue-green algae) in water. Not all cyanobacteria produce toxins, but it has been hypothesized that the aquatic environment with increasing water temperatures will favor the bloom of toxin-producing harmful cyanobacteria, such as *Microcystis* sp. (Paerl, 2008; Davis *et al.*, 2009; Ye *et al.*, 2011). These blooms can produce a variety of cyanotoxins as well as taste and odor compounds that pose a nuisance to water supplies worldwide.

Cyanobacteria are organisms that share characteristics with both bacteria and true algae. Like algae, cyanobacteria contain chlorophyll and other pigments, and can perform photosynthesis. Some cyanobacteria are also able to fix nitrogen via specialized cells (Herrero *et al.*, 2001). In addition, some species can also regulate their buoyancy using specialized intracellular gas vesicles and move vertically within the water column to optimize growth (Porat *et al.*, 2001). Blooms can have a wide variety of appearance, forming colonies, mats, and scums that can range from blue-green to black in color.

Increasing frequency and causes of CHABs

Cyanobacteria have adapted to survive in a variety of environments and have been found across the United States and worldwide. CHABs do occur naturally and are not a sudden or new occurrence. However, there is a general consensus among experts that the incidence of CHABs has been increasing in the U.S. and worldwide in recent decades (Chorus and Bartram, 1999; Carmichael, 2008; Hudnell, 2010). This trend is reflected in the increasing number of published re-

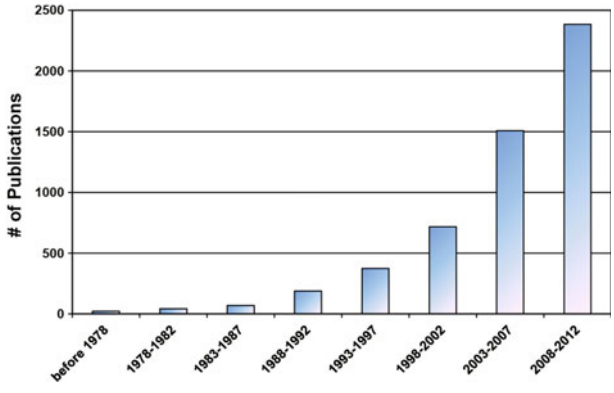

Fig. 1. The increasing trend of published scientific studies about toxic cyanobacteria worldwide. The key words used for searching in database [Web of Science (SCI & SSCI), PubMed and National Library of Medicine] were 'toxic cyanobacteria', 'harmful algal bloom', 'HAB', 'harmful algae', 'cyano HAB' and 'toxic blue-green algae' with EndNote (X4, Thomson Reuters). Journal articles and books were included. It should be noted that the census for 2012 publications was done as of November 2012, so it may underestimate the actual number accumulated by the end of 2012.

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Table 1. Cyanobacteria genera and their associated toxins (modified from Lopez *et al.*, 2008)

Toxin types	Cyanobacteria genera
Microcystins	*Anabaena*, *Aphanocapsa*, *Hapalosphon*, *Microcystis*, *Nostoc*, *Planktothrix (Oscillatoria)*
Nodularins	*Nodularia spumigena*
Saxitoxins	*Anabaena*, *Aphanizomenon*, *Cylindrospermopsis*, *Lyngbya*
Anatoxins	*Anabaena*, *Aphanizomenon*, *Planktothrix (Oscillatoria)*
Cylindrospermopsin	*Aphanizomenon*, *Cylindrospermopsin*, *Umezakia*
Lipopolysaccharide (LPS)	*Aphanizomenon*, *Planktothrix (Oscillatoria)*
Lyngbyatoxins	*Lyngbya*
Beta-N-methylamino-L-alanine (BMAA)	*Anabaena*, *Cylindrospermopsin*, *Microcystis*, *Nostoc*, *Planktothrix (Oscillatoria)*

ports and studies regarding CHABs over time shown in Fig. 1.

While this may also reflect the increasing awareness, surveillance, and reporting of HAB occurrences, it is unlikely that the trend is due to this alone. In the United States (US), as of 2005, at least 35 states have documented CHABs (Lopez *et al.*, 2008). With the increasing frequency and severity of these blooms, many states have developed and instituted response strategies, including Ohio. In addition to increasing frequency and severity of CHAB events, there has also been an increase in the geographic distribution of blooms in the U.S. and world-wide, with blooms appearing in areas previously unaffected (Lopez *et al.*, 2008).

The factors that promote HABs are relatively well-known and understood. Favorable conditions for a CHAB include excessive nutrients (mainly nitrogen and phosphorous), warmth (over 20°C), sunlight, and quiescent water (Hudnell, 2010). Human activities leading to the eutrophication of lakes have been cited as a major cause of increasing CHABs (Paerl, 2008). Excessive nutrients enter freshwater bodies through a variety of point and non-point sources. Point sources, which are controlled by U.S. Environmental Protection Agency (US EPA) National Pollution Discharge Elimination System (NPDES) permits, include waste water treatment plants and industry effluents. Non-point sources, which are often more difficult to identify and manage, include runoff from agriculture, urban and suburban lawn fertilizer, sewer overflows, and wildlife. In freshwater environments, phosphorous from various sources has been shown to be the limiting nutrient (Paerl, 2008). In addition to excessive nutrients flowing into waters, the expansion of human populations and agricultural areas has also led to the depletion of wetlands, which serve as buffer zones and filter nutrients before they enter the water, further exacerbating the nutrient problem (Hudnell, 2010). Other factors such as the abundance or presence of other algal species and grazers in the aquatic ecosystem may also influence the dominance of cyanobacterial species (Anderson *et al.*, 2002).

Impacts of CHABs

Cyanobacteria blooms can have major impacts on aquatic ecosystems. Severe blooms in lakes have ultimately led to oxygen depletion in the water, resulting in massive fish kills. Toxins produced by cyanobacteria have also been linked to mortality in aquatic macro-invertebrates, waterfowl, and other predators (Havens, 2008). CHABs also have heavy economic impacts on local communities. Toxic blooms reduce business during normally tourism-heavy weekends and holidays, resulting in heavy revenue losses. There are also considerable costs associated with mitigation of the blooms and lake restoration, as seen in the recent case of Ohio's Grand Lake St. Marys. In a 2009 paper, a conservative estimate of the economic costs of eutrophication in U.S. freshwaters was determined to be over $2.2 billion annually (Dodds *et al.*, 2009).

Cyanobacteria and their toxins

Harmful cyanobacteria are getting special attention because they are known to produce various types of cyanotoxins, such as hepatotoxins, neurotoxins, cytotoxins, irritants and/or gastrointestinal toxins (Carmichael, 1997). There are a variety of toxin-producing cyanobacteria genera and cyanotoxins.

Table 2. 24-h intraperitoneal LD_{50}s of rodents with selected cyanotoxins in comparison to other well-known toxins (from Hudnell, 2010 with permission)

Compounds and rodent 24 h intraperitoneal LD50s (μg/kg)				
Cyanotoxins	LD_{50}	EPA priority	Comparison	LD_{50}
Saxitoxins[a]	10	Medium/high	Ricin[f]	22
Anatoxin-a(s)[b]	20	Medium/high	Cobra venom[g]	185
Microcystin-LR[c]	50	Highest	Sarin[h]	218
Anatoxin-a[d]	200	Highest	Curare[i]	500
Cylindrospermopsin[e]	300/180	Highest	Strychnine[j]	2500/980

[a] Alkaloid, voltage-gated sodium channel antagonist, Kuiper-Goodman *et al.* (1999).
[b] Alkaloid (similar to organophosphate pesticide), acetylcholinesterase inhibitor, Carmichael *et al.* (1990).
[c] Cyclic peptide, protein phosphatase inhibitor, cytoskeleton damage, other, Kuiper-Goodman *et al.* (1999).
[d] Alkaloid, nicotinic acetylcholine receptor agonist, Carmichael (1997).
[e] Alkaloid, protein synthesis inhibitor, other 300/180 = 24 h/5 days, Hawkins *et al.* (1997).
[f] Glycoprotein, from castor bean (*Ricinus communis*), protein synthesis inhibitor, other, SOPCFC (2008).
[g] Polypeptide, from Egyptian *Naja haje haje* cobras, nicotinic acetylcholine receptor antagonist, Fry (2009).
[h] Organophophospate, synthetic, acetylcholinesterase inhibitor, US Army ERDEC (1994); ORNL (1996).
[i] Alkaloids (e.g., *d*-tubocurarine), poison arrow toxin plants (e.g., *Strychnos toxifera*), nicotinic acetylcholine receptor antagonist, Wenningmann and Dilger (2001).
[j] Alkaloid, from *Strychnos* plants, glycine receptor antagonist, 2500/980 = rat/mouse, IPCS (2009).

Table 3. WHO recreational water guidelines for human health risk (modified from WHO 2003).

Probability of adverse health effects	Cell concentration (per ml)	Chlorophyll-a concentration (μg/L)
Relatively low	<20,000 cells	<10
Moderate	20,000–100,000 cells	10–50
High	>100,000 cells (visible scum)	visible scum

Table 1 includes a list of cyanobacteria genera and associated toxins. As seen in the table, the same toxins can be produced by multiple genera of cyanobacteria. For example, microcystins, the most well-known of the cyantoxins, can be produced by *Microcystis*, *Anabaena*, *Nostoc*, *Oscillatoria*, and *Hapalosphon* (Lopez *et al*., 2008). Cyanobacteria genera can also produce multiple types of toxins. There are also structural variants of the cyanotoxins. Microcystin-LR is the most well-known and studied microcystin. Structurally, microcystins are cyclic peptides and have over 80 known structural variants. Further adding to their complexity, there are toxic and non-toxic strains of cyanobacteria, even within the same species, which often coexist in the environment (Davis *et al*., 2009).

While the factors that stimulate or promote blooms are well-known, the factors that trigger toxin-production or the dominance of toxic versus non-toxic strains are considerably less-understood. There is growing evidence concerning the role of nutrients in toxin concentrations and the dominance of toxic strains (Anderson *et al*., 2002; Davis *et al*., 2009). Several studies have suggested that climatic warming and continued eutrophication of lakes will favor toxic strains over non-toxic strains. A laboratory study by Vézie, *et al*. (2002) examined the effect of varying phosphorous and nitrogen levels on two toxic and two non-toxic strains of *Microcystis*. The study found that the non-toxic strains required less nutrients for growth at low nutrient levels, but the toxic strains outgrew the non-toxic strains at high nutrient concentrations.

Public Health Implications

Toxin potency

The potential public health risk due to exposure to CHAB toxins has been increasingly gaining the attention of scientists and government agencies, as well as the media and the public.

Cyanobacteria are known to produce a variety of hepatotoxins, neurotoxins, and dermatoxins, "several of which are among the most potent toxins known" (Hudnell, 2010). Table 2 shows the LD_{50}s (lethal dose for 50% of the research rodents) of five cyanotoxins compared to five well- known toxins such as ricin, cobra venom, etc. within 24 h of an intraperitoneal injection.

Current guidelines and regulations

The World Health Organization (WHO) safe guideline for microcystin-LR in source water for drinking water is 1 μg/L (Chorus and Bartram, 1999). For recreational waters, the WHO has developed a series of guideline values, with "incremental severity and probability of health effects" defined at three levels (WHO, 2003). Potential human health risk in recreational waters is considered low at 20,000 cells/ml (up to 10 μg/L chlorophyll-a). A cell concentration of 20,000 to 100,000 cells/ml (10 to 50 μg/L chlorophyll-a) is considered a moderate risk to human health. A high risk corresponds to a cell concentration of over 100,000 cells/ml (visible cyanobacterial scum formation). Table 3 is a condensed table of the WHO guidelines for recreational waters according to potential human health risk.

Currently, there are no national primary drinking water regulations for cyanotoxins in the US. Under the Safe Drinking Water Act (SDWA), the US EPA periodically compiles and publishes a Contaminant Candidate List (CCL) consisting of priority contaminants that are currently unregulated and "known or anticipated to occur in public water systems". This list is used to prioritize research and evaluate data to aid in possible regulatory determination. The US EPA listed "cyanobacteria (blue-green algae), other freshwater algae, and their toxins" on the first drinking water CCL (1998) and the second CCL in 2005 as priority chemical contaminants. "Cyanotoxins" was listed on the final CCL 3 in 2009 as a priority chemical contaminant, noting that various reports suggest three cyanotoxins for consideration (Anatoxin-a, Microcystin-LR, and Cylindrospermopsin).

Table 4. Cyanotoxins and their associated short and long-term health effects (from Lopez *et al*., 2008)

Toxin	Genera	Short term health effects	Long term health effects
Microcystins	*Anabaena*, *Aphanocapsa*, *Hapalosphon*, *Microcystis*, *Nostoc*, *Oscillatoria*, *Planktothrix*	Gastrointestinal, liver inflammation, and hemorrhage and liver failure leading to death, pneumona, dermatitis	Tumor promoter, liver failure leading to death
Nodularins	*Nodularia spumigena*	Similar to Microcystins	Similar to Microcystins
Saxitoxins	*Anabaena*, *Aphanizomenon*, *Cylindrospermopsis*, *Lyngbya*	Tingling, burning, numbness, drowsiness, incoherent speech, respiratory paralysis leading to death	Unknown
Anatoxins	*Anabaena*, *Aphanizomenon*, *Oscillatoria*, *Planktothrix*	Tingling, burning, numbness, drowsiness, incoherent speech, respiratory paralysis leading to death	Cardiac arrhythmia leading to death
Cylindrospermopsin	*Aphanizomenon*, *Cylindrospermopsis*, *Umezakia*	Gastrointestinal, liver inflammation and hemorrhage, pneumonia, dermatitis	Malaise, anorexia, liver failure leading to death
Lipopolysaccharide	*Aphanizomenon*, *Oscillatoria*	Gastrointestinal, dermatitis	Unknown
Lyngbyatoxins	*Lyngbya*	Dermatitis	Skin tumors (Fujiki *et al*., 1990), unknown
BMAA	*Anabaena*, *Cylindrospermopsin*, *Microcystis*, *Nostoc*, *Planktothrix*		Potential link to neurodegenerative diseases

Table 5. Examples of documented outbreaks associated with CHAB toxins according to exposure source (from WHO, 2003; Lopez *et al.*, 2008)

	Year	Country	Outbreak
Recreational Water	1959	Canada	Illness (headache, muscular pains, gastrointestinal) of 13 people after recreational exposure to cyanobacterial bloom (Dillenerg and Dehnel, 1960)
	1995	Australia	Human illness (gastrointestinal) associated with recreational water contact in waters with cyanobacteria (Pilotto *et al.*, 1997)
	1989	England	Illness in soldiers training in water with cyanobacterial bloom; 2 developed serious pneumonia (Turner *et al.*, 1990)
	2004	USA	GI illness & dermal irritation associated with recreational exposure to a CHAB event in Nebraska (Walker *et al.*, 2008)
Drinking Water	1931	USA	Illness of around 8,000 people whose drinking water came from tributaries of Ohio River, where a large cyanobacteria bloom had occurred (Miller and Tisdale, 1931)
	1968	USA	GI Illnesses were documented to occur in association with massive blooms of cyanobacteria (cases compiled by Schwimmer and Schwimmer, 1968)
	1979	Australia	Serious illness & hospitalization of 141 people associated with toxic bloom in drinking water reservoir, which had been treated with copper sulfate (Falconer, 1993)
	1988	Brazil	Death of 88 and illness of around 2,000 people associated with toxic cyanobacteria in drinking water reservoir after flood (Teixera *et al.*, 1993)
	1993	China	Liver cancer incidence found higher for populations using surface waters where cyanobacteria occurred in drinking water rather than groundwater (Yu, 1995); although, other factors, including hepatitis and exposures to aflatoxin, may have contributed
	1994	Sweden	Illness (gastrointestinal and muscle cramps) of 121 (out of 304) inhabitants of a village whose drinking water supply was accidentally cross-connected with cyanobacterial contaminated untreated river water (Anadotter *et al.*, 2001)
Water used for Hemodialysis	1974	USA	Chills, fever, hypotension in 23 dialysis patients in Washington, D.C. associated with cyanobacteria in local water source (Hindman *et al.*, 1975)
	1996	Brazil	Death of 52 dialysis patients & illness of 64 others associated with microcystin toxins in water used for dialysis (Jochimsen *et al.*, 1998; Carmichael *et al.*, 2001)

No policy or regulatory determinations have been made as of yet. (U.S. EPA, 2011)

Human exposure to cyanotoxins

The main routes of exposure to cyanotoxins are ingestion, inhalation, and dermal contact. The most common route is ingestion. In recreational water, exposure to toxins can occur through accidental ingestion of water containing cyanobacteria cells and dermal contact (Funari and Testai, 2008). Inhalation of toxin-containing aerosols during recreational activities such as swimming or boating has been shown to be another potential route of exposure (Backer *et al.*, 2010).

Exposure may also occur via ingestion of drinking water or food (e.g. shellfish, nutrient supplements) containing cyanotoxins. Cyanotoxins may occur in drinking water, depending on the concentration in the surface water and the efficiency of the treatment technology used. In areas without adequate water treatment, people may be exposed to intracellular toxins and dissolved cyanotoxins (Funari and Testai, 2008). There may be both short-term and chronic effects of continued ingestion.

Exposure to cyanotoxins can also occur through hemodialysis if untreated or under-treated surface water containing cyanobacteria toxin is used for the treatment. Although rare, this route of exposure can have devastating effects on human health, as the toxin is delivered directly into the bloodstream (Funari and Testai, 2008). One of the most severe outbreaks of CHAB poisoning occurred in Brazil in 1996 when 56 out of 131 patients died after receiving hemodialysis treatment in which the water was contaminated with microcystin (WHO, 2003).

Potential health effects

Health effects and symptoms associated with CHAB exposure are dependent on the level of toxin, route of exposure, and the type of toxin encountered. There may also be both short and long-term effects that result from exposure. For example, short-term health effects associated with exposure to micro-

Table 6. Efficiency of various treatment processes for removal of intact algal cells (OEPA, 2011a)

	Intact algal cells
Treatment process	Treatment efficiency
Coagulation/ sedimentation	Very effective for the removal of intracellular toxins provided cells accumulated in sludge are isolated from the plant
Rapid filtration	Very effective for the removal of intracellular toxins provided cells are not allowed to accumulate on filter for prolonged periods
Slow sand filtration	As for rapid sand filtration, with the additional possibility of biological degradation of dissolved toxins
Combined coagulation/ sedimentation/filtration	Extremely effective for the removal of intracellular toxins provided cells accumulated in sludge are isolated from the plant cells and any free cells are not allowed to accumulate on filter for prolonged periods
Membrane processes	Very effective for the removal of intracellular toxins provided cells are not allowed to accumulate on membrane for prolonged periods
Dissolved Air Flotation	Same as coagulation/sedimentation

cystins include gastrointestinal symptoms, liver inflammation, liver failure leading to death, pneumonia, dermatitis, etc. (Lopez *et al*., 2008). Chronic and long-term exposure to the toxin has been associated with liver and colorectal cancers (Davis *et al*., 2009). Table 4 contains a list of several select toxins, their associated cyanobacteria genera, and their potential short and long-term health effects.

In addition to the outbreaks mentioned above as examples, there have been a number of documented cases of human illness and even deaths associated with cyanotoxins in water worldwide (Lopez *et al*., 2008). A list of select documented outbreaks associated with CHAB toxins and their routes of exposure in several countries is shown in Table 5 (WHO, 2003; Lopez *et al*., 2008).

Drinking water issues

CHAB toxins in drinking water can cause a variety of adverse health effects and pose a risk to public water supplies. Severe CHABs can cause drinking water crises if affecting a population's only or main water supply. For example, in May 2007, a massive *Microcystis* bloom resulted in a major drinking water crisis in Wuxi, Jiangsu Province, China. The bloom affected Lake Tai, which served as the city's only water supply, affecting approximately two million people for at least a week (Qin *et al*., 2010).

CHABs can also cause a variety of problems for public water supplies. Additional monitoring and technology may be required if the toxins are not effectively removed by the current water treatment process. Cyanobacteria and their toxins can also increase treatment chemical demand, microbial growth, and disinfection by-product (DBP) formation within the system (Westrick *et al*., 2010). Additionally, blooms can also cause taste and odor issues in drinking water. However, although undesirable, taste and odor can serve as indicators for presence of toxin in drinking water (Graham *et al*., 2008).

The effectiveness of various water treatment processes on removing intact cyanobacteria cells and dissolved toxins has been relatively well-studied. CHAB toxins are most effectively removed while still encased in cells since "approximately 95% of anatoxin-a, saxitoxin variants, and the microcystin variants are found intracellularly during the growth stage of the bloom" (Westrick *et al*., 2010). Once cyanotoxins are released from cells via excretion, lysis, or cell damage and dissolved in water, they are more difficult to remove. Thus, optimization of the conventional water treatment process for intact cyanobacteria cell removal is an important aim for removing CHAB toxins from drinking water supplies (Westrick *et al*., 2010; Ohio Environmental Protection Agency [OEPA], 2011a). Ohio EPA has developed a guidance document for public water systems regarding CHAB toxin treatment (OEPA, 2011a). Current monitoring in Ohio focuses on the most prevalent cyanotoxins including microcystins, cylindrospermopsin, saxitoxin, and anatoxin-a.

Removal of Cyanobacteria cells and intracellular toxins

The conventional drinking water treatment process generally includes a sequence of coagulation, flocculation, sedimentation, filtration, and disinfection. Usually, proper coagulation, flocculation, sedimentation, and filtration are very effective for removing cyanobacteria cells. Care should be taken, however, to optimize the processes to avoid toxin release from physical damage of cells or decay of cells (OEPA, 2012). For example, in the filtration process, backwash frequency can be increased to avoid the accumulation of cells for long periods of time. The efficiency of various treatments for removal of intact algal cells and intracellular toxins is summarized in Table 6.

Precautions for adding oxidants at intake or pre-oxidation

Oxidants are often added at water intake to reduce taste and odor compounds, discourage biological growth on intake pipes, reduce DBP production, stimulate coagulation, and for the removal of manganese (Westrick *et al*., 2010). This practice, however, can increase the concentration of extracellular toxin by causing lysis of cyanobacteria cells and the release of toxins. For example, chlorination or ozonation have been shown to cause cell lysis and release of toxins (Westrick *et al*., 2010). In the case that pre-oxidation is necessary, a weaker oxidant such as potassium permanganate (KMnO4) can be used, to avoid toxin release (Westrick *et al*., 2010; OEPA, 2011a).

Removal of extracellular toxins

Bypassing the filtration system, extracellular or dissolved toxin may not be completely removed by disinfection, the next step in the treatment process. Treatment efficiency is dependent on a number of factors, including source water quality, disinfectant dose, toxin type and concentration, etc. (OEPA, 2011a). For example, chlorine dioxide and chloramines are not effective in removing toxins from drinking water under practical conditions. Table 7 provides a summary of effectiveness of specific oxidants for four cyanotoxins.

While disinfectants such as chlorine can be effective for some cyanotoxins, additional or advanced treatment may be needed to remove the toxins to safe drinking levels. Current research and water management has focused on the use of the multi-barrier approach and combined treatments (Merel *et al*.,

Table 7. Various oxidants and their effectiveness for removal of anatoxin-a, cylindrospermopsin, microcystin, and saxitoxin (OEPA, 2011a)

	Anatoxin-a	Cylindrospermopsin	Microcystin	Saxitoxin
Chlorine	Not Effective	Effective (at low pH)	Effective	Somewhat Effective
Chloramine	Not Effective	Not Effective	Not effective at normal levels	Inadequate Information
Chlorine Dioxide	Not effective at normal levels	Not Effective	Not effective at normal levels	Inadequate Information
Potassium Permanganate	Effective	Data ranges from Not Effective to Possibly Effective	Effective	Not Effective
Ozone	Effective	Effective	Effective	Not Effective
UV/Advanced Oxidation	Effective	Effective	Not Effective	Inadequate Information

Table 8. Various treatment processes and their effectiveness against extracellular or dissolved toxins (OEPA, 2011a)

Extracellular Toxins		
Treatment process	Toxin	Treatment efficiency
PAC (dose required varies with water quality)	Microcystins (except m-LA)	Wood-based, chemically activated carbon is the most effective, or similar, 60 min contact time recommended
	Microcystin LA	High doses recommended
	Cylindrospermopsin	Wood-based, chemically activated carbon is the most effective, or similar, 60 min contact time recommended
	Saxitoxins	A microporous carbon (coconut or coal based, steam activated wood) 60 min contact time recommended effective for the most toxic of the variants
GAC	All dissolved toxins	GAC adsorption displays a limited lifetime for all toxins. This can vary between 2 months to more than 1 year depending on the type of toxin and water quality
Biological filtration	All dissolved toxins	When functioning at the optimum this process can be very effective for the removal of most toxins. However, factors affecting removal such as biofilm mass and composition, acclimation periods, temperature and water quality cannot be easily controlled.
Membrane processes	All dissolved toxins	Depends on membrane pore size distribution

2010). For example, chlorine may be combined with adsorption with activated carbon, a well-established technique for removing cyanotoxins and a variety of organic compounds. Table 8 lists a number of additional treatment processes that may be used in conjunction with disinfectants and their relative efficiencies with respect to removing certain cyanotoxins.

Advanced treatment for cyanotoxin removal can be costly for public water systems and may even cause operational and maintenance issues (OEPA, 2011b). For larger Ohio community water systems, costs for powdered activated carbon alone can exceed $200,000/month (OEPA, 2012, Personal Communication). Routine monitoring of raw water and optimizing the water treatment process if cyanotoxins are detected may be a less costly solution. It is important to note that water sources and systems are unique. Factors such as source water quality, environmental conditions, dominant cyanobacteria species, water treatment technology and others may differ between areas and should be considered when developing a strategy to reduce public health risk.

Disinfection by-products (DBPs) in drinking water

DBP formation occurs during the water treatment process when disinfectants used to reduce pathogenic microorganisms react with organic matter, bromides, and iodides in the water (Richardson and Postigo, 2011). As briefly mentioned above, in addition to the threat posed to drinking water due to toxins, the increased biomass of the cyanobacterial cells may also result in an increased exposure to DBPs. A number of DBPs, including trihalomethanes (THMs) and haloacetic acids (HAAs), are regulated in the U.S. and other countries, because of their potential reproductive, developmental, and carcinogenic effects (Richardson, 2003). There is much ongoing research with regards to other DBPs and alternative disinfectants used and there are several DBPs on the US EPA's latest CCL list as priority chemical contaminants for regulatory review.

In addition to the organic matter of the cyanobacterial cells, cyanotoxins can also react with disinfectants and form DBPs. Currently, six chlorination byproducts of microcystin-LR and two products of cylindrospermopsin have been characterized (Merel *et al.*, 2010). Although chlorination seems to reduce the toxicity in comparison to the original toxins, there is concern over possible toxicity through a new pathway. Thus, additional research is needed regarding the formation of toxin-related DBPs and their toxicity as well as the overall contribution of harmful algal blooms (HABs) to DBPs in treated water.

Case study: Harmful algal blooms in Ohio

Harmful Algal Blooms have gained increasing attention in state and local agencies as well as in the media. CHABs have been prominent recently in western Lake Erie and several inland lakes, including Grand Lake St. Marys. The CHAB-forming cyanobacteria commonly found in Ohio include *Microcystis*, *Anabaena*, *Aphanizomenon*, *Cylindrospermopsis*, *Lyngbya*, *Nostoc*, and *Planktothrix* (also known as *Oscillatoria*) (Ohio Sea Grant, 2010).

Ohio HAB response strategy

In 2007, Ohio EPA participated in the National Lake Survey program. Of the 19 randomly-selected lakes in Ohio, 36% had detectable microcystin concentrations, which was higher than the national average. These results were released in 2009 and sampling continued in a number of Ohio lakes. Several inland lakes including Grand Lake St. Marys and Maumee Bay State Park had high levels of microcystins during the summer. In response to the growing concern over CHABs and their potential effects on human, animal, and ecosystem health, a committee of representatives from Ohio EPA, Ohio

Table 9. Ohio toxin thresholds for microcystin, anatoxin-a, cylindrospermopsin, and saxitoxin (ODH *et al.*, 2012)

Threshold (µg/L)	Microcystin*	Anatoxin-a	Cylindrospermopsin	Saxitoxin**
Recreational Public Health Advisory	6	80	5	0.8
Recreation - No Contact Advisory	20	300	20	3
Drinking - Do Not Drink	1	20	1	0.2

* * Microcystin and Saxitoxin thresholds are intended to be applied to total concentrations of all reported congeners of those toxins

Department of Health (ODH), and Ohio Department of Natural Resources (ODNR) collaborated and released the State of Ohio's Harmful Algal Bloom Response Strategy in June 2011 and updated in 2012 (ODH, OEPA, ODNR, 2012). The document details guidelines for screening, monitoring, toxin thresholds, advisory postings, sampling techniques, outreach protocols, illness reporting, etc.

Toxicity thresholds for recreational and drinking water

Using toxicity values and exposure assumptions from literature, the committee established recreational and drinking water thresholds for four cyanotoxins: microcystin, anatoxin-a, cylindrospermopsin, and saxitoxin (Table 9). These recommended thresholds are conservative. Ohio's recreational advisory thresholds were designed to be protective of children (lower body weight was used to calculate threshold). For drinking water, OEPA decided to base drinking water thresholds on adult body weight and be consistent with the WHO microcystin guidelines (OEPA, 2012, Personal Communication). In Minnesota, a new drinking water microcystin guideline of 0.04 μg/L was provided and it was developed to be protective of infants and is more conversative than Ohio's threshold of 1.0 μg/L (http://www.health.state.mn.us/divs/eh/risk/guidance/gw/table.html).

Ohio bloom advisory postings

The 2012 Ohio HAB Response Strategy established two levels of algal bloom advisories and the specific language to be posted on signs at State Park beaches (ODH *et al*., 2012). These advisories are used to inform the public of health risks associated with exposure to cyanotoxin-containing water: Recreational Public Health Advisory, and No Contact Advisory. A Recreational Public Health Advisory will be posted when there are toxin levels equal to or greater than the Public Health Advisory thresholds listed in the previous table. During this advisory, the public is advised that swimming and wading are not recommended for the very old, the very young or those with compromised immune systems. The highest advisory level, the No Contact Advisory, is issued when toxin levels meet or exceed the No Contact thresholds, and one or more probable cases of human illness or pet deaths are reported. The public is then advised to avoid all contact with the water. Advisory sign postings are the responsibility of the agency managing the public lake. Posting removals must be based on having two consecutive samples taken at least one week apart with toxin levels below thresholds (ODH *et al*., 2012).

In 2010, when there was a different advisory posting protocol and different thresholds, there were three inland lakes with No Contact advisories including Grand Lake St. Marys, Blue Rock, and Wingfoot Lake. Toxin advisories were posted for eight Ohio lakes. Eleven lakes had posted Bloom advisories. The map (Fig. 2) published by the Ohio EPA depicts the bloom advisories by lake in Ohio in 2010 (OEPA, 2010). For those state park lakes that are not monitored for HABs,

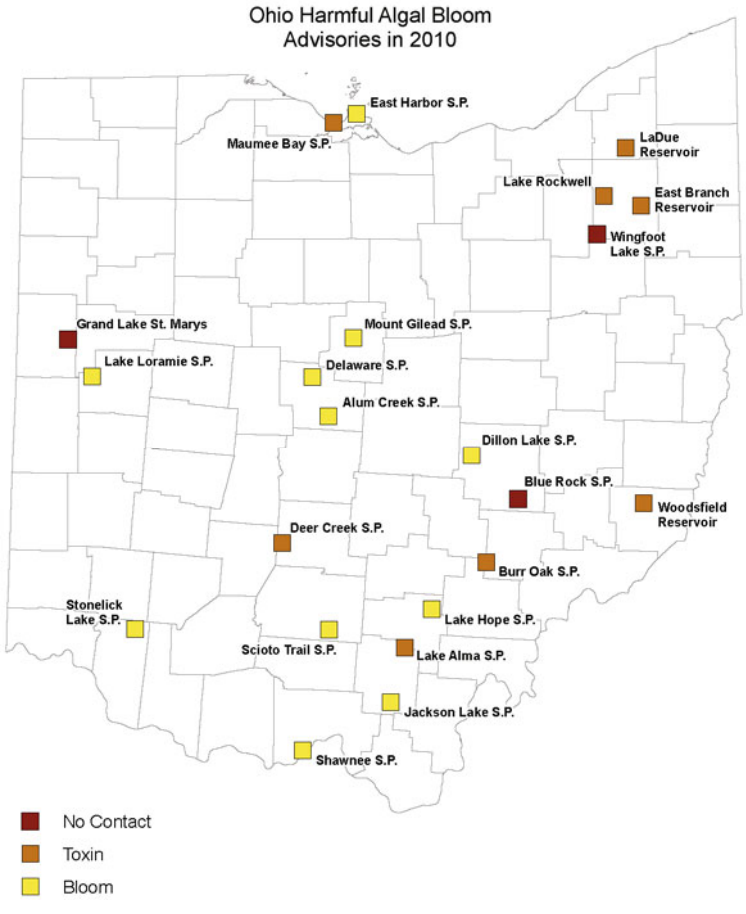

Fig. 2. Harmful algal advisory posted in Ohio lakes during 2010 (OEPA, 2010).

a general signage is posted at lake access locations to alert the public to avoid potential exposure to harmful toxins in the water. The sign includes the pictures showing colors and forms of CHABs (ODH *et al.*, 2012). The response strategy of Ohio continues to evolve.

Cyanotoxin measurements in Ohio public water supplies

In response to CHABs from 2010 to 2012, Ohio EPA collected 487 water samples for cyanotoxin analysis at public water supplies. Public water supplies with source waters chronically affected by CHABs voluntarily collected an additional 463 samples for cyanotoxin analysis of their raw and finished drinking water, and shared data with Ohio EPA. Cyanotoxins were detected in the majority of source waters sampled. In Lake Erie, all the 11 westernmost public water systems had cyanotoxins detected in their raw water. There was only one finished water microcystin detection, but it was below Ohio EPA's drinking water threshold, so a drinking water use advisory was not issued. The absence of cyanotoxins in finished drinking water is largely due to the use of advanced water treatment technologies, including powdered activated carbon, granular activated carbon, and ozone treatment. The one cyanotoxin detection from finished water occurred when advanced treatment was not being used. Source water microcystin concentrations were 1.02 μg/L at that time and the finished water had a microcystin concentration of 0.6 μg/L. For comparison purposes, the City of Celina has had source water microcytin concentrations above 100 μg/L, but cyanotoxins have never been detected in their finished drinking water due to their advanced treatment. The use of advanced drinking water treatment technologies is often at great expense to the local community.

Observational and anecdotal evidence from the Ohio EPA's cyanotoxin sampling suggest that when visible scums appear, the concentration of toxins at a water supply intake may be less than when blooms are not yet visible and the cyanobacteria are mixed in the water column. It seems reasonable that when the cyanobacteria and their toxins are concentrated at the surface (in a visible bloom), they are less prevalent lower in the water column, at the intake depths. For example, 2011 sampling of Lake Erie found that the highest concentrations of cyantoxins at water systems intakes occurred prior to the large scums forming across the lake and the only time microcystin was detected at Lake Erie island intakes was prior to scum formation (OEPA, 2012). Similarly, although there was visible CHAB scum formation over the City of Akron's Lake Rockwell intake in June, no cyanotoxins were detected at the intake. In September, however, low concentrations of saxitoxin were detected at the intake despite the absence of visible bloom formation. Thus, the public health risks associated with CHAB toxins in drinking water supplies and recreational water may differ in timing and require separate consideration and strategies (OEPA, 2012).

Severe CHABs in Grand Lake St. Marys and Mitigation efforts

The most well-known CHAB case in Ohio, aside from blooms in western Lake Erie, is that of Grand Lake St. Marys, Ohio's largest inland lake. The lake is a popular recreational site for fishing, boating, swimming, and other activities and also serves as a drinking water supply for the city of Celina. Over the years, nutrient enrichment has resulted in the degradation of water quality and severe HABs in the lake. In the past few years, CHAB blooms have resulted in EPA advisories to limit contact with water and have had steep economic consequences for the region.

In response to the severe blooms at Grand Lake St. Marys and their impacts, various state agencies including OEPA, ODNR, ODA, and ODH have worked together to develop both short and long-term strategies. Proposed state actions include strategies to address internal phosphorous loading and external nutrient enrichment. The action plans recognize the importance of cooperation between various agencies, landowners, industries, and the community in efforts to restore the lake.

In 2009 and 2010, approximately $6.8 million in local, state, and federal funding was committed addressing water quality issues in the watershed and lake (OEPA, 2010). Examples of grants awarded by the OEPA in 2010 include pilot projects for aluminum sulfate (alum) application, developing wetlands and treatment trains, installation of an aeration device/system, installation of a bed load sediment collector upstream from the lake, education and outreach programs concerning home sewage treatment systems, etc.

Conclusion

Exposure to cyanotoxins can cause serious health effects in both humans and animals and major economic losses in local communities. Experts agree that the incidence of HABs have been increasing worldwide and with warming climates and continued nutrient loading from human activities, it is very possible that the number and severity of toxic blooms may continue to increase. This would further threaten drinking and recreational water sources in Ohio and around the world.

Currently, it seems that conventional and advanced water treatment processes are effective for removing toxins. Water treatment processes are complex, however, and strategies that work in one area may not be as effective in others. Water systems are unique and CHAB toxin risk to public health may depend upon a number of factors such as the quality of the area's source water and the water treatment technology used. Thus, it is vital that public water system managers and local heath agencies investigate and determine the best strategy to protect public health for each system. Apart from the toxins directly produced by the blooms, disinfection byproducts resulting from the high organic matter in the raw water may pose an additional risk to public health. Future research should include characterization and continued monitoring of DBPs and their potential health effects.

The development and implementation of strategies to address CHAB and nutrient issues is also vital. Public outreach and awareness of the issue is crucial in both reducing the amounts of nutrients released into water sources and minimizing human exposure in the event of a toxic cyanobacteria bloom. With the great variety of of cyanobacteria, toxins, and nutrient sources, understanding and managing CHABs can be difficult, but given the public health risk and

environmental and economic impacts, it is certainly a worthwhile endeavor.

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