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Public health responses to toxic cyanobacterial blooms: perspectives from the 2016 Florida event

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Abstract

In June 2016, massive cyanobacterial blooms occurred in the St. Lucie River in Florida, caused by nutrient and cyanobacterial-laden water releases from Lake Okeechobee. We independently collected and analyzed bloom material for cyanotoxin diversity and concentrations. The concentrations of microcystins, potent hepatotoxins, present in the bloom material greatly exceeded World Health Organization Guideline Values for drinking and recreational water. We also detected the neurotoxins anatoxin-a(S) and *β-N*-methylamino-L-alanine (BMAA). The Florida State Governor declared a state of emergency, but many affected aquatic recreational areas in St. Lucie County remained open during the bloom event without adequate hazard notification to citizens. During the bloom event, issues with preparedness, communication, sampling, analysis, closures and contingencies were observed. We suggest better ways that cyanobacterial bloom events can be predicted, managed, and mitigated in the future throughout the world. As similar problems with cyanobacterial bloom frequency and occurrence present worldwide, understanding governmental responses to the 2016 Florida incident can help in the development of effective mitigation and management strategies for future bloom events.

Keywords: Contingency; Cyanobacteria; Eutrophication; Mitigation; Response; Toxins

Introduction

Cyanobacteria produce an array of toxic compounds (cyanotoxins), including potent hepatotoxins, neurotoxins, gastrointestinal irritants, and contact dermatogens (Metcalf & Codd, 2012). When conditions are

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favorable, cyanobacteria in waterbodies can produce blooms occupying thousands of square kilometres, some of which are detectable from space (Hunter *et al*., 2010). The increasing frequency and duration of cyanobacterial blooms with their associated cyanotoxins is an international public health issue. Cyanotoxins have been detected in more than 50 countries and in nearly every US state (Loftin *et al*., 2016). While water discolorations, mats or scums along the shores, and unpleasant odors have long been salient, only relatively recently – over the last century or so – have the links between cyanobacterial blooms and human health been made clear (Gibble *et al*., 2016; Wood, 2016). Since the finding in Australia that cyanobacteria of the genus *Nodularia* were toxic to sheep (Francis, 1878), periodic intoxications of fish, birds, livestock and pets have been reported and attributed to cyanotoxins (Metcalf & Codd, 2012). Humans have also succumbed to the effects of cyanobacterial toxins, including mortalities, most notably via haemodialysis of patients in Caruaru, Brazil, where deaths were attributed to microcystins (Pouria *et al*., 1998; Carmichael *et al*., 2001) as well as the mass illness of aboriginal people on Palm Island, Australia, later associated with cylindrospermopsin in the drinking water supply (Griffiths & Saker, 2003).

Consequently, reducing the risks of human exposure to cyanobacterial blooms and toxins has emerged as a key strategy in minimizing adverse health consequences. Of the known cyanotoxins, the World Health Organization (WHO) has accepted Guideline Values (GVs) for microcystin-LR of 1 μg/L in drinking water (WHO, 1998) and guidelines for the amount of cyanobacteria present in recreational waters, which are often different between countries have also been developed (WHO, 2003; Ibelings *et al*., 2014). Other cyanobacterial toxins are yet to have guidelines established for human exposures, although guidelines for cyanobacterial toxins such as cylindrospermopsin have been proposed (Humpage & Falconer, 2003). Even though the WHO GVs are being adopted by countries around the world, not all countries have legislation concerning the permissible concentration of cyanotoxins. Some countries have guidelines and legislation for cyanobacteria and their toxins, including Argentina, Australia, Brazil, Canada and the United States as examples (Chorus, 2012; Ibelings *et al*., 2014). As further toxins are isolated and links established with human health, then there will likely be increasing pressure to include these cyanotoxins in testing with protective legislation to follow.

Of increasing interest is chronic toxicity from exposure to cyanobacterial toxins. Long-term exposure to microcystins has been implicated in the development of primary liver cancer in Japan and Europe (Ueno *et al*., 1996; Svircev *et al*., 2013). Additionally, nodularin-R may be a carcinogen (Ohta *et al*., 1994). Chronic dietary exposure to the cyanotoxin *β-N*-methylamino-L-alanine (BMAA) produces neuropathologies in laboratory animals consistent with progressive human neurodegenerative illness (Cox *et al*., 2016).

In 2015, the United States Government passed an amendment to the 'Drinking Water Protection Act' (P.L. 114-45) which legislates the establishment of a strategic plan using the best available science to (a) evaluate risks to human health due to public water contaminated with algal toxins; (b) establish a comprehensive list of algal toxins which have an adverse effect on human health; (c) provide summaries of the known adverse human health effects of cyanotoxins in public water; (d) consider public advisories, feasible analytical methods, and frequency of monitoring; (e) recommend treatment options; and (f) enter into cooperative agreements and provide technical assistance to State agencies. Although they do not establish guideline or regulatory values for cyanotoxins in drinking water, they do indicate a need to coordinate public protection at a Federal level. At the State level, three US states (Ohio, Oregon and Minnesota) have published guidance values for cyanotoxin concentrations in drinking water and others are considering or developing action plans (Ibelings *et al*., 2014; Henrie *et al*., 2017).

Critical to reducing cyanobacterial toxin exposures is the ability of public health authorities to accurately assess toxin concentrations and then provide rapid warnings to the public. Similar alerts have long

been provided to protect the public from exposure to paralytic shellfish poisoning (saxitoxins) and brevetoxin associated with red tides. Brevetoxin and ciguatoxin exposures occur in warm waters following dinoflagellate blooms where these toxins accumulate in shellfish and pelagic fish as well as those that dwell in coral reefs (Naar *et al*., 2007). Similar to saxitoxins, both toxins affect voltage-dependent sodium channels disrupting nerve conduction and, in human exposure, lead to symptoms of paresthesias, reversed sensations of hot and cold, myalgia, ataxia, and sometimes respiratory, gastrointestinal, and cardiovascular distress (Lombet *et al*., 1987; Farstad & Chow, 2001).

While satellite imaging and geographic information system (GIS) techniques can provide some indication of existing cyanobacterial blooms (Hunter *et al*., 2010), predicting toxic cyanobacterial blooms is still an uncertain endeavor. Since approximately half of all reported human exposure incidents are a result of recreational activities (Wood, 2016), rapid notification could have a tremendous positive impact in protecting public health. Therefore, when cyanobacterial blooms occur in areas frequented by the public, rapid efforts should be made to analyze the blooms for known toxins and to provide the public with information and warnings, so people can minimize their exposure.

Although the 2016 cyanobacterial blooms that occurred in Martin and St. Lucie counties in Florida were prominently reported in national print and broadcast outlets, public information on toxicological risks was not immediately forthcoming from public health authorities. With global warming and increased nutrient effluents into lakes, estuaries, and other near shore environments, cyanobacterial blooms seem to be increasing both in frequency and extent (Paerl & Huisman, 2009). Understanding the governmental response to the 2016 Florida cyanobacterial bloom incident can help in the development of effective mitigation and management strategies in future cyanobacterial bloom events worldwide.

Background to the 2016 Florida bloom

Lake Okeechobee. Lake Okeechobee is the seventh largest freshwater lake in the USA, with a mean depth of 2.7 m and a water volume of approximately 5.2 cubic kilometres. The lake covers an area of 1900 square kilometres and is fed from lakes, canals and rivers to the north, many of which are contaminated with agricultural runoff containing high phosphorus and nitrogen concentrations. In order to protect the Everglades from flooding downstream and to increase suitable agricultural acreage for sugar cane cultivation, the US Army Corps of Engineers constructed the Herbert Hoover Dike surrounding the lake along with a navigation channel. Two additional canals were constructed to allow flow from Lake Okeechobee. To the west of the lake is the Caloosahatchee River, which flows to the Gulf of Mexico near Ft. Myers and Sanibel Island. To the east, the St. Lucie Canal flows toward Stuart, southern Indian River Lagoon and into the Atlantic Ocean. Both of these waterways divert nutrient-rich freshwater from the lake into the ocean, preventing flow into the protected Everglades and flooding of agricultural communities south of the lake.

Due to the inflow of large amounts of water into the lake and issues concerning the structural integrity of the Herbert Hoover Dike, the US Army Corps of Engineers release water intermittently from the lake so as to maintain the water level at a suitable height (Gunter & Hall, 1963). Prior to such comprehensive management, in 1926 a two-metre tall earthen dike was breached by a storm surge resulting in approximately 300 deaths to the south of the lake. Two years later during the 1928 San Felipe Segundo hurricane, floodwaters in excess of six metres caused thousands of fatalities downstream (Blake *et al*., 2011).

During the period before the 2016 cyanobacterial bloom incident in Lake Okeechobee, large amounts of rainfall resulted in nutrient runoff into the lake, as well as a significant increase in water height. Warm, sunny, still weather, in addition to the nutrients, resulted in the formation of a large cyanobacterial bloom in the lake. In order to control water levels, nutrient and cyanobacteria-laden water were released into the St. Lucie River in May-July 2016 (Lantigua, 2017).

The objective of our study was to determine the potential toxicity of the cyanobacterial bloom in the 2016 Florida incident (Florida, 2016) by collecting and analyzing cyanobacteria and fish for the presence of known cyanotoxins, as well as evaluating the adequacy of the response of Florida State officials to the potential public health consequences of the cyanobacterial bloom. These data and our analysis of the government response will hopefully be useful to inform future government responses and best practices for lake water management worldwide.

Materials and methods

Field collections and microscopy

Cyanobacteria were collected at sites on Lake Okeechobee and the St. Lucie Canal (Figure 1). Scums and mats were collected in 60 ml containers at each site, taken at shorelines of publicly accessible sites on 7 and 8 July 2016, stored on ice and then transported to the laboratory. Samples were taken where people were likely exposed to cyanobacteria, assuming a worst-case scenario, by collection of shoreline scums likely to contain the highest concentrations of cyanotoxins. Once received, the samples underwent

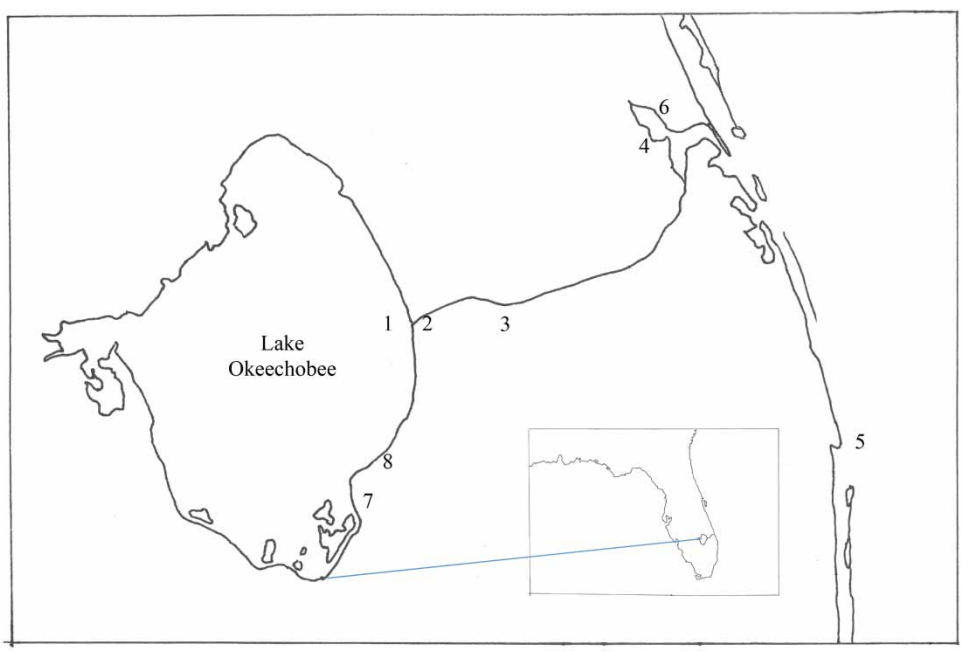

Fig. 1. Location of sampling sites on Lake Okeechobee and along the St. Lucie River.

qualitative microscopic analysis at magnification of × 200 (Zeiss Axioplan 2, San Diego, USA) according to Whitton (2002) and the remainder of each sample was lyophilized. Once lyophilized, the entire 60 ml collection was weighed and subsamples removed for extraction and cyanotoxin analysis. Extracts were sonicated in either 70% (v/v) methanol (microcystins, anatoxins) or with DirectQ water (Millipore; anatoxins, cylindrospermopsin) at a concentration of 50 mg dry wt. cells per ml for analysis.

Three fish of each species were caught by hook and line on July 13, 2016 with catfish caught at Roosevelt Bridge over St. Lucie River (27°12'12''N, 80°15'29''W) and mojarra at Palm City Bridge over St. Lucie River (27°10'25''N, 80°15'40''W) before transportation to the laboratory on ice where they were frozen. The fish samples represented the types of fish that could be caught by members of the public at these sites. The brain and muscle were dissected from each frozen fish and the sample freeze dried for analysis of BMAA and neurotoxic isomers according to Chatziefthimiou *et al.* (2018). All biological samples were treated as individual replicates with each sample analyzed in triplicate.

Anatoxin-a(S)

Aqueous and methanolic extracts were assessed for the presence of anatoxin-a(S) using an acetylcholine esterase inhibition assay (Mahmood & Carmichael, 1986). Acetylcholinesterase (AChE) from electric eel, type V-S was obtained from Millipore-Sigma (St. Louis, MO, USA). A stock solution of the enzyme was prepared in 0.1 M KH_2PO_4 buffer (pH 8.0) and kept frozen. For each assay, 0.25 U of enzyme were used. Solutions of acetylthiocholine (ATCh, Millipore-Sigma, St. Louis, MO) were prepared with 0.1 M KH_2PO_4 buffer and kept frozen. A 0.01 M solution of dithiobisnitrobenzoate (DTNB, Millipore-Sigma, St. Louis, MO) was prepared in 0.1 M KH_2PO_4 buffer and 15 mg of $NaHCO_3$ was added to the final 10 ml volume and kept frozen. Into each microtiter plate well, 300 µl of 0.1 M KH_2PO_4 buffer, 20 µl of DTNB, 0.3 µl of sample (or neostigmine standard), 2 µl ATCh and 10 µl AChE were added in order, with the last reagent added in rapid succession via a multi-channel pipette. The plate was maintained at 37 °C with continuous shaking, and read every minute at 412 nm for 10 minutes with a Powerwave HT Microplate Spectrophotometer (Biotek, Winooski, VT, USA). The concentration of anatoxin-a(S) was based on an equivalent concentration of neostigmine in the enzyme inhibition assay.

Microcystin

Cyanobacterial material extracted in 70% (v/v) methanol was analyzed with ultra-high performance liquid chromatography with photodiode array detection (UPLC-PDA, Waters Acquity Sample Manager, Binary Solvent Manager and PDA detector, Waters, Milford, MA, USA) following separation with a Waters Acquity Ultra 2.1 × 100 mm C18 column heated to 55 °C, using solvents of purified water +0.1% (v/v) trifluoroacetic acid (TFA, A) and acetonitrile +0.1% (v/v) TFA (B), using a gradient of 25 to 75% B over 5 minutes, and monitoring at 238 nm. Spectra were compared with a microcystin-LR standard (Millipore-Sigma) and quantified as microcystin-LR equivalents.

Anatoxin-a

Aqueous extracts of cyanobacteria were analyzed by Waters Acquity UPLC-PDA using a Waters Acquity Ultra 2.1 × 100 mm column maintained at 40 °C using DirectQ (DQ) water +0.1% TFA (A)

and acetonitrile +0.1% TFA (B) from 0% to 0.5% B over 5 minutes in comparison with an anatoxin-a standard (Millipore-Sigma), monitoring the PDA chromatogram at 227 nm and with spectral matching.

Cylindrospermopsin

Aqueous extracts of cyanobacteria were analyzed by Waters Acquity UPLC-PDA using a Waters Acquity Ultra 2.1 × 100 mm column maintained at 40 °C using DQ water +0.1% TFA (A) and acetonitrile +0.1% TFA (B) from 0% to 0.5% B over 5 minutes in comparison with cylindrospermopsin purified from *C. raciborskii* CR3 (Metcalf *et al*., 2002) through monitoring of the PDA at 262 nm with spectral matching.

BMAA and isomers

Samples were extracted using 6.0 M HCl following previously validated protocols (Glover *et al*., 2015). 6-aminoquinolyl-N-hydroxysuccinimidyl carbamate (AQC)-derivatized amino acids were separated by reverse phase chromatography (Glover *et al*., 2015) and analyzed on two instruments; a Waters Xevo TQ-S Triple Quadrupole mass spectrometer (Waters, Milford, MA, USA) and a Thermo Scientific TSQ Quantiva (Thermo-Fisher Scientific, Waltham, MA, USA) triple quadrupole mass spectrometer in comparison with AQC-derivatized BMAA and isomers (*N*-2(aminoethyl) glycine (AEG); 2,4-diaminobutyric acid (DAB); *β*-amino-*N*-methyl-alanine (BAMA)).

For fish tissues, dried tissue was extracted with 10% (w/v) trichloroacetic acid (TCA) using an internal standard of *β*-*N*-methyl-$^{2}H_3$-amino-DL-alanine-$^{15}N_2$. Tissue was sonicated on ice (Fisher Scientific Sonic Dismembrator, model 100, Waltham, MA; 2 Watts, 2 × 30 s) and extracted at 3 °C overnight, followed by centrifugation (14,000 × *g*, 3 min) and removal of the supernatant. The pellet was resuspended in 10% TCA, sonicated, and extracted at room temperature for 2 h. Supernatants were pooled, centrifuge filtered (0.2 µm, Millipore Ultrafree-MC, Merck Millipore, Cork, Eire) and analyzed for free BMAA and isomers using AQC derivatization (2 µl sample +78 µl borate buffer +20 µl AQC) following manufacturer's recommendations (Waters AccQTag reagent, PN WAT052880, Waters, Milford, MA). The remaining pellet was hydrolyzed in 6M HCl (17 h, 110 °C) with a second addition of internal standard at this time. Hydrolyzed samples were diluted 1/50 with purified water (Millipore Direct Q-3uv, 18 MΩ) and derivatized with AQC (20 µl sample +60 µl borate buffer +20 µl AQC). Analysis was conducted on a TSQ Quantiva (Thermo-Fisher Scientific, Waltham, MA) triple quadrupole mass spectrometer equipped with a Waters Ultra High Pressure Liquid Chromatography instrument using a validated method, as previously described (Cox *et al*., 2016). Resulting peaks were compared with authenticated standards of BMAA (Millipore-Sigma B-107 St. Louis, MO) and L-2,4-diaminobutryic acid dihydrochloride (Millipore-Sigma, DAB-32830) with method detection limit (3.2 ng/ml BMAA; 0.61 ng/ml DAB) and limits of quantification (9.6 ng/ml BMAA; 1.8 ng/ml) calculated according to Environmental Protection Agency (EPA) guidelines.

Results

Microscopic analysis of the cyanobacterial bloom material from Lake Okeechobee and the St. Lucie Canal showed the presence of *Microcystis aeruginosa* as the dominant cyanobacterium present. In

addition to *Microcystis aeruginosa*, *Dolichospermum flos-aquae* (formerly *Anabaena flos-aquae*) and *Planktothrix* sp. were also present within the bloom material.

Samples collected from six out of eight sites contained microcystin(s) with concentrations up to 1.56 μg/mg which equated to around 200 mg/l (Table 1). The data concerning the presence of microcystins show that on a gravimetric basis, the concentration was fairly constant, with a factor of 3 variation between sampling sites (Table 1). However, when calculated on a per volume basis, differences between the amounts of microcystin were variable, largely due to the differences in cyanobacterial density, indicative of the pulse-like nature of the release of the cyanobacterial bloom material from Lake Okeechobee (Table 1).

In addition, two samples from Hobe Sound and Lake Okeechobee contained anatoxin-a(S), a potent acetylcholinesterase inhibitor, with concentrations at 6.71 and 12.67 ng/mg neostigmine equivalents, respectively, most likely due to the presence of *Dolichospermum flos-aquae*. Neither anatoxin-a nor cylindrospermopsin were found in samples.

The chronic neurotoxin, *β-N*-methylamino-L-alanine (BMAA) was found in three out of six samples hydrolyzed and tested on a Waters Xevo TQ-S Triple Quadrupole mass spectrometer using positive electrospray ionization (ESI) between 4 and 12 ng/g (Table 1). We further note that, in these samples, BMAA co-occurred with three BMAA isomers (*N*-(2-aminoethyl)glycine (AEG); 2,4-diaminobutyric acid (DAB); and *β*-amino-*N*-methyl-alanine (BAMA); Table 1). BMAA was detected as the monoisotopic ion, a calcium adduct and a sodium adduct in the three samples. BMAA isomers but not BMAA itself were detected in a second analysis using a Thermo Scientific TSQ Quantiva triple quadrupole mass spectrometer following an aqueous extraction for free amino acids only. Brain samples from the fish samples contained both BMAA and DAB at 4 ng/g and 90 ng/g for catfish and mojarra, respectively (Table 2).

Discussion

Toxicological potential

Exposure to cyanobacteria has the potential to cause adverse human health effects from toxins contained within the bloom material. As the 2016 Florida bloom (Florida, 2016) was comprised of cyanobacteria that

Table 1. Cyanotoxin analysis of cyanobacterial samples collected from Lake Okeechobee and the St. Lucie Canal, July 7–8, 2016 ($n = 3$).

Site	ATX-a(S) ng/mg	MC			BMAA ng/g	DAB ng/g	AEG ng/g	BAMA ng/g
		No. Var.	μg/mg	mg/l				
1	ND	2	0.92	0.31	NT	NT	NT	NT
2	ND	5	1.18	6.47	6	4	3	10
3	ND	9	0.54	216	4	180	ND	10
4	ND	4	1.56	194	ND	ND	ND	ND
5	6.71	ND	ND	ND	12	90	ND	3
6	ND	ND	ND	ND	ND	40	0.6	3
7	12.67	3	0.65	0.27	NT	NT	NT	NT
8	ND	3	1.25	5.75	ND	ND	ND	ND

NT, not tested; ND, not detected; MC, microcystin; ATX-a(*S*), anatoxin-a(*S*); Sites, see Figure 1; No. Var., number of microcystin variants.

Table 2. Selected cyanotoxin analyses of fish samples collected from Florida (2016) ($n = 3$).

Sample/location	Free BMAA ng/g	Protein BMAA ng/g	Free DAB µg/g
Catfish brain	4	ND	2.1
Catfish muscle	ND	ND	5.2
Mojarra brain	90	ND	0.5
Mojarra muscle	ND	ND	9.5

ND, not detected.

were well known for their ability to produce microcystins (MC), the bloom material was assessed for the presence of this class of hepatotoxins. The concentration of microcystins contained within this material was extremely high, orders of magnitude greater than the WHO Guideline Value for MC-LR in drinking water of 1 µg/l (WHO, 1998) and 2,000 times higher than German recreational Guideline Values (Ibelings *et al.*, 2014). As a result of the MC concentration contained within the bloom material, closure or restriction of access to the waters should have occurred rapidly, if not immediately (Figure 2), with continuous monitoring to determine potential adverse health effects. Given the likelihood of long-term hepatotoxic or carcinogenic consequences from the microcystin content, it may have been prudent for public health officials to offer assistance to households living on the banks or near the St. Lucie River until the cyanobacterial bloom subsided.

Although there was the potential for adverse short-term human health impacts, there is also the possibility that exposure to such bloom material may have long-term health impacts. Certainly, in the case of microcystins, the 1 µg/l GV is for daily consumption over a lifetime. Long-term exposure to relatively low MC concentrations is considered to be a risk factor for primary liver cancer, as indicated from studies in China (Ueno *et al.*, 1996) and Serbia (Svircev *et al.*, 2013). Chronic exposure to BMAA and isomers may also have long-term health implications as evidenced by neuropathologies in vervets consistent with human neurodegenerative disease (Cox *et al.*, 2016). Consequently, although the short-term toxicological implications of the Florida (2016) cyanobacterial bloom are significant, understanding the long-term implications requires further research, longitudinal epidemiological surveys of individuals exposed to the Florida (2016) cyanobacterial bloom, and ultimately protection through guidance, legislation, and warnings.

Legislation concerning microcystins has been introduced in a number of countries around the world (Ibelings *et al.*, 2014). This is largely based on the WHO GV for microcystin-LR of 1 µg/l and has led to systems designed ultimately to protect the public. Exposure can occur through drinking and recreational waters and alert systems are largely in place, based upon microscopy of water samples and the number of cells contained within which trigger various alert levels, depending on the country (Chorus, 2012). Certainly microscopy is a useful tool for determining the potential risk and is used in a number of countries, including the UK as a basis for alerting to potential issues associated with the bloom or water (Turner *et al.*, 2018). Other methods, such as polymerase chain reaction (PCR) can be used to detect genes involved with cyanotoxin production. Although the presence of genes can infer the potential for toxin production, unless toxin analysis has been carried out it may be difficult to determine the potential risk of exposure. Even when legislation is not present, other regulations such as the Drinking Water Directive and the Bathing Water Directive of the European Union and the Clean Water Act in the USA (Ibelings *et al.*, 2014) allow for protection of people and animals from a wide range of toxicants, including cyanobacterial toxins.

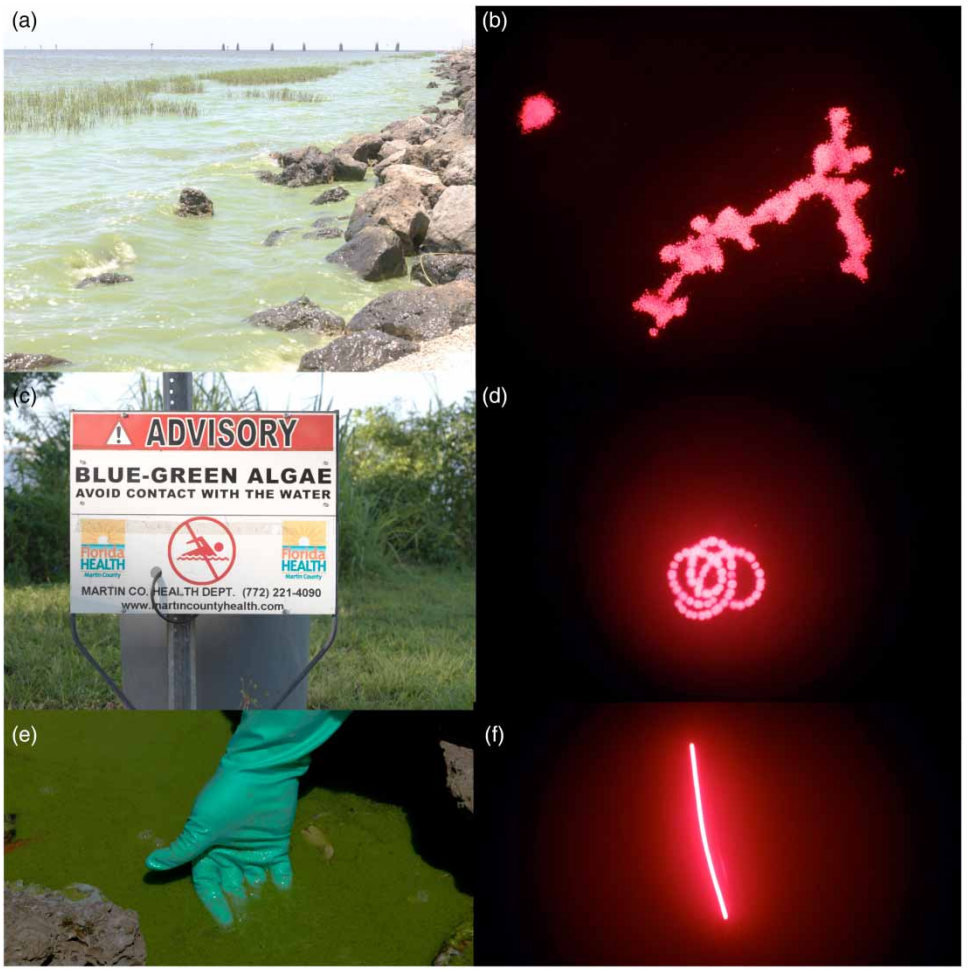

Fig. 2. Example photographs of the cyanobacterial bloom and fluorescence microscopy of representative organisms in the St. Lucie Canal. A, *Microcystis* bloom; B, *Microcystis aeruginosa* colony; C, example warning notice; D, *Dolichospermum flos-aquae*; E, protective clothing with *Microcystis* scum; F, *Planktothrix* sp.

Florida has had a harmful algal bloom (HAB) task force, dedicated to assessment, communication, and human protection, in place since 1998 (Florida, 1998). This task force is intended to produce a united response from five agencies: Department of Environmental Protection, five water management districts (WMDs), Florida Department of Health (DOH), Florida Fish and Wildlife Conservation Commission (FWC), and the Florida Department of Agriculture and Consumer Services (DACS).

Neither the State of Florida, nor the US Government, currently has water quality criteria or alert levels for cyanotoxins, although they are listed in the Drinking-Water Contaminant Candidate List with health advisory drinking water regulations in three US states (Hudnell *et al*., 2014; Henrie *et al*., 2017). However, during the release of the bloom down the St. Lucie Canal, samples were collected for cyanobacterial and cyanotoxin analysis by government workers who were likely concerned about potential human health consequences of exposure to the cyanobacterial bloom. Ultimately, low microcystin

concentrations were detected in bloom material collected by agencies working in Florida (Florida, 2016). With respect to sampling, based upon the precautionary principle, samples of concentrated scums should be collected in order to provide information for the potential dose of toxin which may be consumed as a worst-case scenario. If open waters are sampled, containing little or no cyanobacteria, rather than scums or thick bloom material on shorelines or in embayments, then if present, lower toxin concentrations will be obtained which may not trigger alert levels. Based on microcystin content alone reported here, it is a reasonable prediction that the cohort of Florida State citizens exposed to the 2016 Florida cyanobacteria bloom incident, including children of underprivileged families that we witnessed picnicking, fishing, and swimming in cyanobacterially contaminated waters, may experience an increased lifetime risk of liver cancer and/or hepatic dysfunction requiring hospitalization or transplantation.

Implications for best practices elsewhere

Over the last few years a number of high profile cyanobacterial bloom incidents, some of which contained toxin concentrations of concern to the general public, have occurred in the USA. In addition to the microcystin-containing bloom in Florida, a large cyanobacterial bloom caused closures in Utah Lake, Provo, Utah (Brooks *et al.*, 2017) and the occurrence of a large bloom of *Microcystis* on Lake Erie shut down a water treatment plant and resulted in the provision of bottled water to the residents of Toledo, Ohio (Pelley, 2016). The government of Ohio had a HAB management document in place with threshold toxin values for microcystins, cylindrospermopsin, anatoxin-a, and saxitoxins specified within the document (Ohio EPA, 2016). Exceeding specified toxin concentrations effectively triggered a 'harmful algae blooms' advisory system with proscribed response and counter-response when toxin concentrations later dropped. In the case of such blooms with public health implications, speed of response is essential. Furthermore, awareness and education of the risks posed by cyanobacteria and their toxins is also necessary. In order to allay public concerns, communication between agencies involved and to the public is necessary. Coupled with warning notices and social media, this will help prevent misinformation and panic. By doing this, with closures and warnings where necessary, financial losses will ultimately be minimized, whether those losses occur through reduced recreation, tourism or agriculture, as waters can be reopened when risks of exposure to cyanobacterial toxins have passed. In order to accomplish this, planning and contingencies are essential and the following should be considered.

Analytical plan

As cyanobacterial blooms can occur at any time with the potential for acute or chronic toxicity, plans concerning the analysis of cyanobacterial blooms are necessary. As with any analysis, an understanding of the cyanobacterial taxa that are present can aid in the analytical methods that follow (Metcalf & Codd, 2012). This is largely based upon the analyses of many strains of cyanobacteria, comprising many genera and species. Using microscopy as a principal tool can help predict what toxins may be present in the bloom and is often quicker and easier than genetic methods such as PCR. Rapid collection and transportation of samples should also be performed with samples stored cold to prevent degradation. If analysis cannot be carried out quickly, then the samples should be frozen. Analytical methods are best carried out 'in house', and screening methods are commercially available for rapid and simple testing of cyanobacterial samples for known cyanotoxins, with verification by other methods after the initial

screen. Such methods will allow the triggering of alert levels and US states, including Florida, should consider their implementation. In addition, dead or sick animals present at the site of the bloom – such as the 11 dead manatees present in the St. Lucie River during the 2016 Florida incident reported by the press – may provide indicators as to the nature of the toxicant. In the case of decaying cyanobacterial blooms, aquatic animal deaths may result from the release of toxins from cells or a reduction in the amount of dissolved oxygen due to the bloom decay process (Small *et al*., 2014).

Communications plan

Although accurate information can be rapidly obtained and assessed by scientists, as with many environmental issues there are stakeholders and agencies, both national and international that may require access to the information, in addition to providing valuable input. Such information could be used to assess needed actions which should be taken, as well as informing the general public regarding closures, water restrictions and general medical advice. Central to this approach is rapid and accurate communication. Some governments such as the Scottish Government (2012) have documentation in place which is revised periodically providing information and advice in dealing with such blooms and their effects. A communications plan should be developed and in place prior to development of blooms.

Precautionary principle

Often, when a cyanobacterial bloom is identified or observed, the precautionary principle is invoked. Closure of waterways, and restrictions on public and animal access to such waters can often offer the greatest level of protection for the public. However, this may not always be possible as some waters may be used for the preparation of drinking water and for agriculture. Certainly, in some cases with regard to drinking water, alternative water sources or changes to the extraction point or depth can be made. However, as in the case of Toledo, OH, this was not possible and bottled drinking water was provided to people who were unable to consume tap-based drinking water. In addition, another preparatory activity with regard to drinking water provision is adaptability in the drinking water treatment process, with the potential to add further treatment measures to try and remove cyanobacterial components from water.

It may also be necessary to stop water-based recreational activities such as water skiing, jet skiing and fishing if a lake or waterway is contaminated with a cyanobacterial bloom. In the case of fishing, it will also be important to prevent consumption of any fish or shellfish from the waterbody, unless tested and found to be safe, as cooking processes will not destroy toxins present in the food.

Waterbody plan

Plans should be drawn up, maintained and revised where necessary, to allow closures to be introduced efficiently, and with communication to all relevant parties as to the nature of the closure and any restrictions. This may include posting of warning notices (Figure 2), press releases and social media releases to effectively communicate when and what is happening and to prevent panic. During such times, it is also important to have regular monitoring and testing of the waterbodies to determine the extent of the risk(s) posed and to be able to re-open waters once the incident has passed.

Media plan

As cyanobacterial bloom events, such as in Florida (2016), can be significant in their duration and geographical range, public information is an essential element that requires good stewardship and may actually serve to reduce public anxiety. Through social media, press releases and television and radio interviews, information concerning the extent and health effects of cyanobacterial blooms can be safely and rapidly released. With additional elements such as warning signs and testing, toxic cyanobacterial blooms can be managed effectively. Key to this is the planning and preparation of what needs to be done and advanced determination of who will carry this out before a cyanobacterial bloom incident occurs.

Adverse effects

Cyanobacterial blooms have the potential to cause economic losses from adverse impacts on tourism, agriculture, and fisheries. If preparations are in place and can be implemented quickly, then along with frequent analysis and situation assessment, closures and negative economic impacts can be minimized. Depending on the country, litigation and compensation may also occur. Ultimately, the problems associated with cyanobacterial blooms can be alleviated or mitigated through multi-agency efforts, largely through catchment management to prevent the introduction of nutrients such as nitrogen and phosphorous which are often the drivers for cyanobacterial blooms.

Conclusion

Cyanobacterial blooms have the potential to cause adverse human and animal health effects, as well as significant economic losses through closures of waters, fisheries and agricultural facilities. In order to minimize such losses, essential planning and communication are required to inform stakeholders and the public to allow rapid analysis and risk assessment and the closure of affected waters for the minimum period necessary to protect human and animal health. This can include the use of social media to inform the public, provision of protective clothing and bottled water and sampling from scums in order to obtain worst-case scenarios for cyanotoxin presence. Furthermore, once a cyanobacterial bloom is observed then the agencies responsible should follow the precautionary principle and close waterbodies if necessary to protect water users.

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