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Cyanobacterial Harmful Algal Blooms: Chapter 31: Ecosystem Effects Workgroup Report

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Chapter 31: Ecosystem Effects Workgroup Report

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Introduction

Harmful cyanobacterial blooms represent one of the most serious ecological stressors in lakes, rivers, estuaries and marine environments. When there are persistent or frequent blooms with high biomass of cyanobacterial cells, colonies or filaments in the water, a wide range of impacts on the ecosystem may occur. These are well established in the scientific literature and are summarized in Paerl et al. (2001). Blooms may shade the water and thereby inhibit growth of other primary producers including phytoplankton, benthic algae and vascular plants and may elevate pH, particularly in poorly buffered waters. High population densities of large cyanobacteria interfere with food collection by filter–feeding zooplankton. The senescence and subsequent microbial decomposition of blooms may impact benthic macro–invertebrate community structure, as well as fish and other biota, due to increased organic loading and resulting anoxia of sediments, accumulation of NH4 in the water and accompanying increases in pH. Blooms of toxic cyanobacteria have been implicated in mass mortalities of birds and fish (e.g., Matsunaga et al. 1999; Rodger et al. 1994), but

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the importance of cyanotoxins relative to the other stressors that accompany blooms remains unknown. With persistent blooms, there are substantial declines in biodiversity at all levels ranging from phytoplankton and zooplankton to birds. Changes in nutrient cycling and disruptions of carbon and energy flow in pelagic and benthic food webs are observed (Paerl et al 1998). Where blooms become severe in shallow lakes, a positive feedback loop develops through various biological mechanisms related to the presence of cyanobacteria and fish that maintains a turbid water state (Scheffer and Carpenter 2003).

A major uncertainty regarding the effects of cyanobacterial blooms is the role that cyanotoxins play in contributing to the various biological responses listed above. There are three reasons for this uncertainty: (a) most research to examine cyanobacterial bloom effects at the ecosystem level has focused on factors not associated with toxins but with the mere presence of cyanobacteria; (b) no experimental studies have been done at the whole community level to examine effects of blooms in the presence of vs. absence of cyanotoxins; and (c) experimental studies dealing with cyanotoxins have largely involved exposure of a single species to a single toxin under ideal conditions in the laboratory. Studies have not examined synergistic effects with other natural stressors, nor have they adequately investigated how multiple toxins of natural and anthropogenic origin might affect the biota. Thus, laboratory results are not readily transferable to the field.

The objective of this report is to identify major knowledge gaps regarding the impacts of cyanobacterial blooms on biota in lakes, rivers and estuaries from the individual to ecosystem level. The text is organized around six charges given to the Ecologic Effects Working Group. All of the identified research components are considered by the Working Group to be a high priority. Careful consideration was given to information already available in the primary literature in determining research needs to avoid duplicity of effort. A simple conceptual model illustrates the interrelationship among the research and modeling work discussed in the subsequent sections of this paper (Fig. 1).

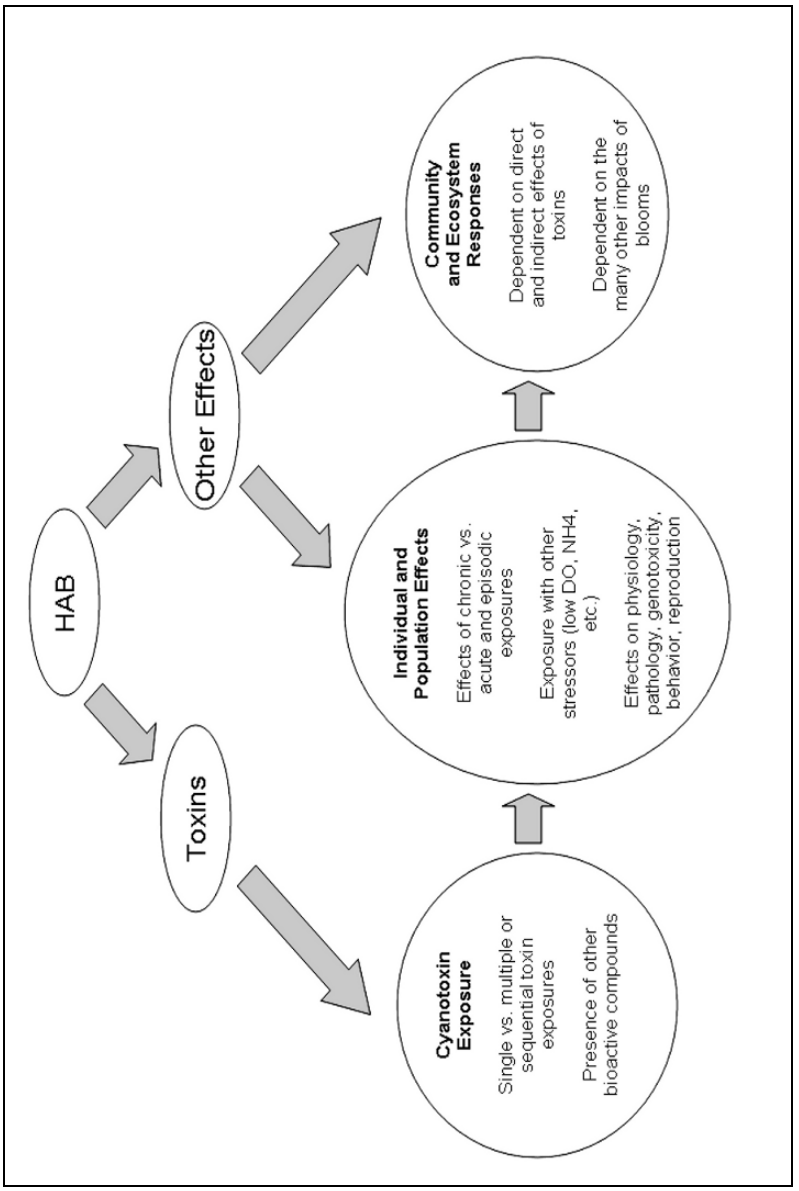

Fig. 1. Conceptual model of the ecosystem effects of cyanobacteria and cyanotoxins

There is a logical order in which the research topics noted here might be addressed, starting with the species level work and then scaling up to the community level with environmentally–relevant experiments based on findings from previous work. Development of community bioaccumulation models may occur in concert with controlled and observational research, so that at any given time, modeling tools may become available for application with clearly identified levels of uncertainty and defined boundaries of applicability.

Charge 1

Identify research needed to quantify effects of cyanotoxins under environmentally relevant conditions.

To understand the effects of cyanotoxins on aquatic biota and ecosystems, it is critical that environmentally–relevant exposure conditions be identified and evaluated. Almost all experimental studies of exposure have been of single species exposed to individual cyanotoxins under optimal conditions (see Ibelings and Havens, this issue for an overview). In addition, there are a limited number of studies that have assessed the distribution of cyanotoxins in lake food webs (Kotak et al. 1996; Ibelings et al. 2005). These studies have furthered our understanding of potential ecological effects, but field studies alone are insufficient to identify associations between exposures and ecological effects during periods when cyanobacterial blooms predominate in aquatic communities.

It is well established in the toxicological literature that stressors may have antagonistic, additive or synergistic effects (Taylor et al. 2005). Hence there is a need for studies that determine how exposure to cyanotoxins alone or in combination with other physiologically stressful conditions (e.g., low dissolved oxygen, high ammonia (NH_4), high pH, poor food quality, high and low temperature, salinity, etc.) affect the fitness of aquatic biota. Often these sub–optimal or stressful environmental conditions coincide with the presence of cyanotoxins in the water, and the relative contribution of each exposure is poorly understood. Bury et al. (1995) demonstrated that NH_4, like dissolved microcystin–LR, impeded fish growth. Additionally, interactions may occur between different classes of the cyanotoxins themselves. Indeed, lipopolysaccharide endotoxins can inhibit glutathione *S*–transferases *in vivo*, thereby reducing the capacity of glutathione *S*–transferases to detoxify microcystins (Best et al. 2002).

Most controlled experiments have examined the biotic effects of microcystin–LR, and to a lesser extent nodularin, with relatively few studies looking at the effects of other cyanotoxins (Table 1). Although some cyanobacteria produce saxitoxins, the ecological effects of these toxins have been studied primarily in marine systems where they are produced by dinoflagellates (Landsberg 2002). Toxins that have been less frequently studied are cylindrospermopsin and its derivatives, and lyngbyatoxins. Given the likelihood that these toxins are present in US waters (Carmichael et al. 1997; Burgess 2001), their effects must be quantified if we are to make predictions about the ecological effects of toxic cyanobacterial blooms with a reasonable level of certainty.

Table 1. Number of peer reviewed papers on ecological effects of cyanotoxins by class of toxins and group of aquatic organisms. Results from October 2005 search (key words: toxin plus organism as listed in the column head) of ISI Web of Knowledge (Thomson Scientific and Healthcare, Stamford, Connecticut).

Class of toxins	Zooplankton	Bivalves	Fish	Waterfowl
Microcystin	73	38	87	13
Nodularin	17	17	16	2
Anatoxin–a and a(s)	11	0	7	6
Cylindrospermopsin	5	2	7	0
Lyngbyatoxin	5	1	9	0
Microviridin	4	0	0	0
Saxitoxin (freshwater)	2	2	6	0

It is becoming increasingly clear that the numerous studies of microcystin–LR may not be representative of the complexities of the interactions between other cyanotoxins, cyanobacterial blooms, and other biota. Complexity arises because: (a) other microcystin variants can be abundant and may be ecologically more relevant, such as the less toxic microcystin–RR which may be taken up preferentially into biota (Xie et al. 2005); (b) cyanotoxins other than microcystins occur widely and have documented adverse ecological effects, such as the association between anatoxin–a(s), anatoxin–a and mass avian mortality events (Henriksen et al. 1997; Krienitz et al. 2003); (c) there is an array of potentially harmful bioactive compounds produced by cyanobacteria which have not been well–studied. For example, microviridin–J has detrimental effects on molting in *Daphnia*, but its effects on other biota are not well understood (Rohrlack et al. 2004). There have been some examples of the toxicity of crude cell–extracts exceeding the expected toxicity of the component cyanotoxins, suggesting that unidentified compounds or synergistic effects are associated with observed toxicity (Lürling 2003).

It also is critical that exposure studies use relevant organisms where possible. For example, there is considerable variation in intraspecies susceptibility to cyanotoxins among fish and birds, but only a relatively small number of species have been studied (Carmichael and Biggs 1978; Fischer and Dietrich 2000). Few experimental studies have been done using waterfowl (Carmichael and Biggs 1978). This has largely necessitated the use of oral toxicity data obtained from the study of other animal groups to estimate the risk of avian toxicity (Krienitz et al. 2003).

Charge 1: Identify research needed to quantify effects of cyanotoxins under environmentally relevant conditions

Near-term Research Priorities

- Laboratory studies exposing key aquatic biota to cyanotoxins under simulated natural conditions, including low dissolved oxygen, elevated pH, elevated ammonia, and other stressors associated with cyanobacteria growth and senescence.
- Field and/or mesocosm studies of the ecologic effects of cyanotoxins under varying environmental conditions.

Charge 2

Identify research needed to quantify the physiological, pathological and behavioral effects of acute, chronic, and episodic exposures to cyanotoxins.

The duration of exposure to cyanobacterial blooms and toxins can range from days to years, yet most studies have investigated the effects of short–term exposures. Research is needed on the effects of long–term exposure and adaptive responses of populations that may employ both existing phenotypic plasticity and adaptive evolution. Although mortality has been examined as a common endpoint, sub–lethal effects require further study. With the exception of *Daphnia*, few studies have examined behavioral responses and their significance to the affected species. A small number of studies have demonstrated that fish behavior is affected by exposure to cyanotoxins in water (Best et al. 2003; Baganz et al. 1998). Further research needs include controlled experiments to examine effects of

cyanotoxins and toxin mixtures on: (a) behavior, especially as it relates to escape from predators and ability to acquire resources; (b) reproduction; (c) neurologic function, and (d) genotoxicity. Some work has been performed to evaluate the role of enzyme inhibition and oxidative stress on genotoxicity, however, the relevance of oxidative stress under field conditions is unknown. Inhibition of protein phosphatases is the classic mode of action of microcystins and nodularins, resulting in hyper–phosphorylation of cytoskeletal proteins and the disruption of numerous other phosphorylation–regulated cell processes. Oxidative stress occurs during the detoxification process. Detoxification produces glutathione–microcystin conjugates, a depletion of the cellular glutathione pool and an imbalance in reactive oxygen species (Pflugmacher 2004).

The effects of chronic exposure to cyanotoxins are rarely investigated in aquatic animals, despite their widespread geographic distribution and potential lifelong exposure to toxic cyanobacterial blooms. Some cyanotoxins have been reported to be tumor promoters, and the risk of tumorigenesis increases with chronic exposure. Microcystins, nodularins, cylindrospermopsins, aplysiatoxins, debromoaplysiatoxin, and lyngbyatoxin–a have all been demonstrated to be tumorigenic, but these properties have only been experimentally demonstrated in small mammals or cell assays (Fujiki et al. 1984; Falconer and Humpage 1996). Recent studies have investigated the association between tumor–promoting cyanotoxins and an increased prevalence of fibropapillomas in seaturtles (Landsberg 2002; Arthur et al. 2005).

It is important to consider individual susceptibility as influenced by factors including age, disease, nutrition, and gender. Species–specific susceptibilities include those related to differences in detoxification and metabolism of cyanotoxins. More detailed knowledge is needed, both in the field of toxicodynamics and toxicokinetics among species. There have been a few sub–chronic exposure studies on the accumulation and depuration of cyanotoxins in a limited number of animals. This type of research seems to have focused on bivalves and, to a lesser extent, fish. The bivalve studies showed a biphasic depuration of microcystin. Fluctuating microcystin concentrations during depuration were speculated to be the result of an ongoing process of covalent binding and release of microcystins (Amorim and Vasconcelos 1999). Overall much is unknown about the fate of cyanobacterial cells and cyanotoxins after ingestion. Reports suggest that only a small percentage of the toxins that are ingested with the food end up in the blood and organs of the organisms: 2.7 % in *Daphnia*, and 1.7 % in rainbow trout (Rohrlack et al. 2005; Tencalla and Dietrich 1997). There are barriers to microcystin uptake at various levels. Even if taken up, aquatic organisms have the capacity for detoxification, which in fish is followed by rapid excretion via the biliary excretion system. Studies that examine

chronic exposure of biota over ecologically relevant time scales of months – years have, to our knowledge, not been conducted. In addition, very little is known about patterns in the accumulation and depuration of cyanotoxins other than microcystin. Ultimately, good quantitative data is required for a number of well defined endpoints for a range of toxin classes and aquatic biota (Table 2).

Table 2. List of Definitions

Term	Definition
Bioaccumulation:	The process which causes an increased chemical concentration in an aquatic organism compared to the water, due to uptake by all exposure routes (Gray 2002).
Bioconcentration:	Uptake directly from the water, and results in the chemical concentration being greater in an aquatic organism than in the water (Gray 2002).
Biomagnification:	Transfer of a chemical from food to an organism, resulting in a higher concentration in the organism than in its diet. The result may be a concentration of the chemical as it moves up the food chain (Gray 2002).
Biodilution:	Decreased toxin levels are observed at each increase in trophic level in the food web.
Endpoint:	An observable or measurable biological event or chemical concentration (e.g., metabolite concentration in a target tissue) used as an index of an effect of a chemical exposure.
Exposure	
Acute:	Resulting in adverse effects from a single dose or exposure to a substance
Chronic:	Continuous or repeated exposure to a substance over a long period of time, typically the greater part of the total life–span in animals or plants
Subchronic:	Exposure for period typically involving a time period in between acute and chronic
No–Observed–Adverse–Effect Level (NOAEL):	The highest dose at which there are no biologically significant increases in the frequency or severity of adverse effects between the exposed population and its appropriate control; some effects may be produced at this level, but they are not considered adverse or precursors of adverse effects. In comparison, see LOEL.
Lowest Observable Effect Level (LOEL):	The lowest dose which produces an observable effect.
Toxicodynamics:	The determination and quantification of the sequence of events at the cellular and molecular levels leading to a toxic response to an environmental agent.
Toxicokinetics:	The determination and quantification of the time course of absorption, distribution, biotransformation, and excretion of chemicals.

Charge 2: Identify research needed to quantify the physiological, pathological and behavioral effects of acute, chronic, and episodic exposures to cyanotoxins

Near-term Research Priorities

- Investigate the behavioral effects of cyanotoxin exposures
- Investigate sub–lethal effects of chronic exposures of key taxa of aquatic biota including invertebrates, fish, bivalves and others.

Charge 3

Identify research needed to quantify biological effects of exposure to multiple toxicants

An increasing number of reports describe the co–occurrence of different cyanotoxins in aquatic systems, and there is an emerging catalogue of bio–active materials associated with cyanobacterial blooms. For example, co–occurrence has been observed for microcystin and anatoxin–a, microcystin and cylindrospermopsin, and there is an assumed universal co–occurrence of lipopolysaccharides with all other known cyanotoxins (Codd et al. 2005). Research on the death of lesser flamingos in Kenya's Rift Valley lakes demonstrated that both microcystin and anatoxin–a were present in the cyanobacteria on which the birds were feeding (Krienitz et al. 2003). The relative contribution of these cyanotoxins, which differ greatly in their mode of action, to the mass bird mortalities remains unclear, as is the role of co–occurring anthropogenic pollutants like heavy metals and organic pesticides. Thus, research is needed to examine effects of simultaneous and sequential exposure to multiple toxins (Codd et al. 2005). The Working Group considers the highest priority for multiple–exposure studies of effects in US lakes to be the evaluation of the combination of microcystin and cylindrospermopsin. Research also is needed to examine effects of other bio–active compounds, including non–microcystin cyclic peptides and lipopeptides.

Charge 3: Identify research needed to quantify biological effects of exposure to multiple toxicants

Near-term Research Priority

- Investigate the effects of simultaneous and sequential exposure to multiple toxins, particularly the combination of microcystins and cylindrospermopsin, and microcystins and anatoxins

Charge 4

Identify research needed to quantify effects of cyanotoxins at whole community level

Community level effects are a function of: (a) the direct effects of cyanotoxins; (b) the direct effects of cyanobacteria blooms; (c) the indirect effects associated with altered competitive and predatory interactions; and (d) changes in nutrient cycling. Effects may occur in all biota from bacteria to birds and mammals and are integrally linked with a loss of biodiversity in aquatic systems. Key research needs include studies involving complete natural communities and studies with simple food chains to examine the effects of exposure to toxic vs. non–toxic strains of cyanobacteria.

At this time we do not adequately understand the relative importance of different uptake routes of toxins from the environment. Exposure to cyanotoxins can be through direct ingestion of cells, uptake of toxins that are present in the environment, or by the transfer of toxins through the food web. Vectorial transport of cyanotoxins has been demonstrated in a few experiments involving dissolved toxins. Fish may be negatively affected by dissolved toxin uptake through the gills, but other biota are not very sensitive to the toxins once they are extracellular (Lürling & Van der Grinten 2003; Zurawell et al. 1999). There is little information about the relative importance of this exposure route vs. exposure by direct ingestion of toxic cells. In one experiment, pike larvae were exposed to zooplankton that had accumulated dissolved nodularin. There was a strong inhibition of larval feeding rate despite the fact that only 0.03 % of the toxin that was present in the zooplankton was actually taken up by the larvae (Karjalainen et al. 2005). The remainder of the toxin was either metabolized or excreted. If this is a representative result, vectorial transport of only a small amount of the cyanotoxin that is produced at the base of the food web may have significant ecological effects at higher trophic levels. Substantiation of the relevance of vectorial transport and the effects of different classes of

cyanotoxins throughout the food web is of prime importance to understand ecological effects of these toxins.

Much research is needed to understand the degree of bioaccumulation that occurs in communities. Bioaccumulation may vary considerably between species, but this has been studied only with a small number of organisms. Most bioaccumulation studies have focused on microcystin. In future research and modeling, it is critical that we distinguish between bioaccumulation, bioconcentration and biomagnification (Table 2). In most studies, the term bioaccumulation is used in a loose way, simply meaning that toxins are present in biota. Ibelings et al. (2005) have argued that biomagnification is the most relevant process to study in food webs since most of the transfer and uptake of cyanotoxins appears to be via food. An increase in microcystin concentration, as it moves up the food chain was not found by these authors, and was not expected due to the low octanol–to–water partition coefficient of microcystin–LR (De Maagd et al. 1999). However it is known that the octanol–to–water coefficient varies widely according to the microcystin variant and correlates with in vivo toxicity to Tetrahymena (Ward and Codd 1999). Cyanobacterial toxins other than microcystin may behave very differently, as indicated by the distribution of the neurotoxin β–N–methylamino–L–alanine (BMAA) in the terrestrial food web on the island of Guam. Biomagnification of this toxin may have accounted for the exposure of the indigenous Chamorro people to high concentrations of BMAA via their consumption of flying foxes (Cox et al. 2003). More knowledge is required on the potential for bioaccumulation, bioconcentration and biomagnification of different cyanotoxins and other cyanobacterial bioactive compounds in the food web.

An important shortcoming of all but a few studies is the absence of data on covalently–bound microcystin in biota. Microcystins are routinely extracted using aqueous methanol, but this does not extract quantities of the methyldehydroalanine–containing microcystins which are covalently bound to protein phosphatases in the cell. Lemieux oxidation does extract these covalently bound forms. Studies that have compared standard aqueous methanol extraction to extraction after Lemieux oxidation have demonstrated that a large part of the total microcystin pool in biota is indeed covalently bound (Table 3). Most of the literature therefore severely underestimates the concentration of total microcystin (free and bound forms). Covalent binding of microcystins may reduce the transfer of free, unbound microcystin along the food chains, and potentially contribute to biodilution of microcystin (Karjalainen et al. 2005). A relevant, but as yet unanswered question, concerns the toxicity and bioavailability of the covalently–bound microcystins (Ibelings et al. 2005).

Table 3. Comparison of standard aqueous methanol extraction and Lemieux oxidation among organisms

Organism	MeOH extraction as % of Lemieux oxidation	Reference
Dungeness Crab (larvae)	0.01	Williams et al., 1997a
Salmon (liver)	24	Williams et al., 1997a
Blue mussel	0.1	Williams et al., 1997b
Zebra mussel	62	Dionisio Pires et al., 2004

Charge 4: Identify research needed to quantify effects of cyanotoxins at whole community level

Near-term Research Priorities

- Investigate the effects of exposure to toxic vs. non–toxic strains of cyanobacteria in natural communities and simple food chains.
- Examine the potential for bioaccumulation, bioconcentration and biomagnifications of different cyanotoxins and other cyanobacterial bioactive compounds in the food web.
- Determine the toxicity and bioavailability of covalently–bound microcystins.

Charge 5

Identify research needed to determine the relative importance of the effects of cyanotoxins vs. the effects of cyanobacteria at the ecosystem level

Cyanobacterial bloom development, maturation and senescence can all result in adverse environmental conditions that affect biota independent of the effects of cyanotoxins. Key research questions at the ecosystem level of inquiry include: (a) how important are the effects of cyanotoxins vs. the effects of cyanobacteria; (b) does the presence of high concentrations of cyanotoxins, for instance through interference with zooplankton grazing, contribute to the stability of the turbid water state in shallow eutrophic lakes; (c) does the increasing occurrence of cyanotoxins in shallow lakes undergoing eutrophication contribute to the shift from the clear to turbid

state? Effects of toxins on benthic communities and benthic processes are not well understood, yet those processes play a key role in aquatic food webs and nutrient cycling (Palmer et al. 2000). The relevance of these processes is demonstrated by the consequences of the invasion by zebra mussels (Dreissena polymorpha) in the Laurentian Great Lakes of North America. It has been hypothesized that selective filter feeding by these mussels has been instrumental in the return of Microcystis blooms to Lake Erie (Vanderploeg et al. 2001). However, in the Netherlands, Microcystis is efficiently grazed by Dreissena, resulting in a low concentration of cyanobacteria in areas where the mussels are abundant (Dionisio–Pires et al. 2004). This paradox is not fully understood, although emerging explanations include variation in cyanotoxin concentrations of the Microcystis strains involved and the relevance of nutrient recycling in lakes of widely varying trophic status (Raikow et al. 2004). The pseudofeces of Dreissena are rich in cyanobacteria and they may transfer toxins to the benthic food web, where benthic feeders are potentially exposed to the toxins (Babcock–Jackson et al. 2002). To further address these issues and gain a deeper understanding of interactions between toxins, cyanobacteria and other biota at the ecosystem level, we propose research at a high level of integration, including the use of static and flowing mesocosms under controlled conditions.

Charge 5: Identify research needed to determine the relative importance of the effects of cyanotoxins vs. the effects of cyanobacteria at the ecosystem level

Near-term Research Priorities

- Investigate the importance of the effects of cyanotoxins vs. cyanobacteria at the community level.
- Determine if the presence of high concentrations of cyanotoxins contributes to the stability of the turbid water state in shallow eutrophic lakes.
- Determine if the increasing occurrence of cyanotoxins in shallow lakes undergoing eutrophication contributes to the shift from the clear to turbid state.

Charge 6

Identify how modeling can contribute to a predictive understanding of HAB bloom and cyanotoxin effects.

Basic models relating cyanobacterial growth to nutrient inputs and other environmental conditions are readily available, and their development is not a priority research area. Various factors including phosphorous, nitrogen, iron and light have been studied in the laboratory and are known to have an effect on cyanotoxin concentrations (Wiedner et al. 2003).

However, there is a need for models that relate environmental conditions to cyanotoxin types, concentrations and compartmentation (soluble vs. particulate pools) in blooms and water bodies containing benthic cyanotoxins. Evidence is emerging that cyanotoxin concentrations increase in direct response to exposure to grazers like zooplankton and fish (Jang et al. 2003, Jang et al. 2004). This would strengthen the idea that cyanobacteria produce these energetically costly toxins as a grazer–deterrent, but whether this is the sole or primary purpose for toxin production is an important question that demands further research. While many of these factors impacting toxin production have been studied individually, modeling the interactive effects of both the bottom–up and top–down factors could provide further insight into the potential toxicity of a bloom given a set of environmental conditions.

Models are needed to describe the fate of cyanotoxins in water, sediment and food webs. This is not a trivial undertaking since toxins at every level in the food web are potentially subject to covalent binding, metabolism (detoxification) and excretion, and thus the amount of bioavailable cyanotoxin that is transferred to the next trophic level is a complex issue. As noted, modeling can be an ongoing activity, with predictive certainty and general applicability increasing as ongoing research at the population, community and ecosystem levels provides additional information for model parameterization, calibration and verification.

Conclusions

The authors of this report have identified near term priorities for research on ecological and ecosystem effects of harmful cyanobacterial blooms. Although the negative impact of cyanobacterial blooms on many ecosystems is well known, the specific contribution of cyanobacterial toxins to the harmful effects is hard to distinguish. Most research has involved exposure

of a single species to a single toxin under controlled laboratory conditions. More research is needed at the whole community level. The research priorities ordered from the species level and scaling up to the community and ecosystem level that have been identified are:

- To study the effects of cyanotoxins under environmentally relevant conditions, including other environmental stressors; additive or synergistic effects of combinations of cyanobacterial toxins or bioactive compounds produced by cyanobacteria.
- To use relevant, naturally co–occurring organisms in exposure studies; more knowledge is needed on species–specific toxicokinetics and toxicodynamics.
- To obtain good quantitative data for a number of well–defined endpoints for a range of toxin classes and biota.
- To study the effects of long term exposures and adaptive responses of aquatic organisms.
- To identify community level effects of cyanotoxins. Key research needs include studies of simple food chains and natural communities exposed to toxic cyanobacteria.
- To identify the relative importance of different uptake routes from the environment and the extent to which vectorial transport of toxins in the food web takes place.
- To understand the potential for bioaccumulation, bioconcentration and biomagnification of cyanobacterial toxins in aquatic food webs.
- To distinguish between the ecosystem effects of cyanobacterial toxins and the harmful effects of cyanobacterial blooms in general (toxic vs. non toxic blooms).
- To build and test models that relate environmental conditions to cyanotoxin types, concentrations and compartmentalization and models that describe the fate of cyanotoxins in water, sediment and food webs.

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