

**Written Testimony of
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**Hearing on
Harmful Algal Blooms and Hypoxia: Formulating an Action Plan**

**Before the
Subcommittee on Energy and Environment
Committee on Science and Technology
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Introduction

Good morning Mr. Chairman and members of the Subcommittee. My name is Gregory Boyer, and I am a Professor of Biochemistry at the State University of New York's College of Environmental Science and Forestry (SUNY-ESF). I am also the Director of New York's Great Lakes Research Consortium (NY-GLRC), a Consortium of over 300 scientists located at 18 New York academic institutions and seven Canadian affiliate institutions with interests in all aspects of Great Lakes Science and Policy. However more importantly, I am a career scientist who has worked on the chemistry and ecology of harmful algal blooms (HABs) for more than 35 years, starting from my Ph.D. work on the chemistry of paralytic shellfish poisons, the neurotoxins produced by selected marine red tides, and continuing in my current work on the toxins produced by freshwater HABs. In 2002, I became the lead scientist for MERHAB-Lower Great Lakes, a NOAA-sponsored regional program to develop monitoring and event response protocols for harmful algal blooms in the lower Great Lakes. Working with MERHAB-LGL, NOAA's Oceans and Human Health Initiative (OHHI), and with the US-EPA Great Lakes National Program Office (GLNPO), I have spent many thousands of hours on our Great Lakes and inland waterways examining and responding to freshwater harmful algal blooms. I also operate a rapid response laboratory at SUNY-ESF for toxic HAB samples submitted from hospitals, health departments, state environmental conservation agencies, lake monitoring organizations and concerned citizens from across North America. These efforts give me a unique ground-up perspective from the needs of our national program to address both marine and freshwater HABs.

Today, my colleagues, Donald Anderson and Dan Ayers, will speak to you about marine harmful algal blooms so I would like to confine my comments to the issue with Freshwater HABs. This topic was first brought to your attention in a hearing last summer by Dr. Ken Hudnell. Here, I would like to summarize the types and impacts of freshwater harmful algal blooms in the United States, as well a comment specifically on the proposed legislation that is

being developed by Chairman Bart Gordon that directs the EPA to participate in freshwater HAB research and authorizes funds for freshwater research applications.

BACKGROUND

Blooms of freshwater algae occur across the United States and around the world. Under the proper conditions of light, temperature, and nutrients, these small aquatic plant-like organisms can grow to extremely dense concentrations, blocking out light from reaching the water below the surface, clogging the water intakes of our nation's power and industrial plants, and leading to taste and odor issues with our drinking water. Unsightly and smelly surface accumulations interfere with the use of local beaches and recreational parks, leading to a decrease in the recreational and tourism dollars flowing to small businesses and local municipalities. Upon their death, the decay of these blooms can consume the available oxygen in the water column, leading to fish kills and local hypoxia [low oxygen concentrations].

Freshwater HABs are not simply a nuisance issue. In addition to those generalized effects described above, there are a number of freshwater algal species that produce extremely potent toxins. When these occur in natural systems, bad things can happen, including illness and mortalities to domestic animals, widespread loss of fish and wildlife, and potential harm to humans using the waters for drinking or recreational purposes.

Toxins and their Health Effects

There are currently more than 300 different toxins reported to be produced by freshwater algae. These toxins vary widely in their chemistry, their effect on ecosystems, and in their potential effect on animals and humans who are exposed to them. Most toxins are produced by a group of 20 or more species of blue-green algae (a.k.a. cyanobacteria); however there are also species that are not blue-green algae that produce toxins that have dramatic impacts on aquaculture and fish communities. Some of the major toxins and species include:

Peptide Liver Toxins. The peptide liver or hepatotoxins called microcystins are probably the most common toxins produced by blue-green algae. Microcystins are named for the genus of cyanobacteria (*Microcystis*) from which they were first identified. Subsequent work has shown that they can be produced by a number of different genera and species. The peptide toxins also include a second, closely-related group of compounds (nodularins) that are usually associated with more saline environments such as marine systems, the Great Salt Lake in Utah, or the Salton Sea in California. *Microcystis* is an extremely common genus of cyanobacteria and it is likely this toxin will be found in every state of the United States. It has been associated with recent animal fatalities in the Midwest, Northeast, and Oregon, and is of major concern to drinking water suppliers that must use impacted waters (example: western basin of Lake Erie) as their source water. One of the first describe toxic events in the United States (1931) refers to an outbreak of *Microcystis* in the Ohio and Potomac Rivers that caused intestinal illness in an estimated 5,000 – 8,000 people. Nebraska (2004) recently experienced a similar event on a smaller scale with a number of dog, livestock, and wildlife fatalities, and more than 50 accounts of human skin rashes, lesions and flu-like gastrointestinal illness.

Microcystins have been linked with animal deaths and human illnesses all over the world. The acute human toxicity of these toxins was graphically observed in Brazil in 1996, when

naturally occurring concentrations of toxin in the water supply for a hemodialysis center led to the death of over 50 patients. There are also concerns over long-term sub-acute exposure. Microcystin-LR, the most studied member of the class, was recently reclassified by the International Agency for Research on Cancer into risk group 2B (*possibly carcinogenic to humans*) based on a review of existing scientific evidence. This raises further questions about the risk from chronic exposure to this group of toxins through drinking water supplies. To date, the United States does not have regulatory standards or guidelines for the concentrations of cyanobacteria toxins allowed in drinking water. However standards exist at both the international level (World Health Organization), national level (e.g. Australia Brazil, Canada, France, Japan, New Zealand, Norway, Poland, Spain), and exist or are under development at the State Level (CA, FL, IA, NE and OR). The US-EPA, as a first step for issuing such as standard, placed microcystins on its Critical Contaminant List or CCL as described under the Safe Drinking Water Act (SDWA) as amended in 1996. The drinking water CCL is a list of priority contaminants, known or anticipated to occur in public water systems, where further research may be necessary before US-EPA can decide if a regulatory ruling is needed under the SDWA. Microcystins have been on the US-EPA's CCL-1 (1998), CCL-2 (2005), and now CCL-3 (2008). They remain on the CCL, in part, because of missing information in terms of their health effects, routes of exposure and analytical methodology. The US-EPA, as part of their toxicological assessment associated with the CCL, has determined that microcystins, anatoxin-a and cylindrospermopsin (discussed below) are the cyanobacterial toxins of most concern (highest priority), followed by paralytic shellfish poisoning (PSP) toxins and anatoxin-a(S).

Freshwater Cytotoxins. The second group of cyanobacterial toxins is the cylindrospermopsin derivatives. This group is also produced by a number of different genera of cyanobacteria, including members of the genus *Cylindrospermopsis* for which they are named. The major toxin, cylindrospermopsin, results in generalized cell death (cytotoxin) but have also been linked to DNA damage as well as possible tumor initiation. However, in accordance with US EPA "Guidelines for Carcinogen Risk Assessment" (2005), this toxin should be listed as "*inadequate information to assess carcinogenic potential*" until further studies have been completed.

Cylindrospermopsin-producing species are generally associated with tropical or arid environments, and toxic *Cylindrospermopsis* blooms are common in the warmer drinking water reservoirs of Florida. Recently, the major potentially-toxic species (*C. raciborskii*) has been identified in temperate Europe and in several of the Great Lake States (MI, OH, WI), suggesting that its observed range has expanded from southern states (FL, NC) to more northern temperate climates. The factors responsible for this potential spread and the production of toxins in these more northern climates are an area of active investigation.

Freshwater Neurotoxins. A third class of toxins are the neurotoxic cyanobacterial toxins; anatoxin, anatoxin-a(S), and the PSP toxins discussed below. The most important member of the class, anatoxin-a, was originally reported from an *Anabaena* species (hence the name), but like the microcystins, this toxin can be produced by a number of different species and genera. Blooms containing anatoxin-a in many states across the United States. They have recently been associated with domestic animal (dog) and livestock (cattle) fatalities in NY, VT, OR, and in the mid-western states. A toxic bloom containing anatoxin-a occurred as recently as this last month in Elk Creek (Douglas County, OR). This bloom resulted in the deaths of several household pets and widespread media coverage of the event.

A second neurotoxin, anatoxin-a(S), is very distinct in both its chemistry and mode of action from anatoxin-a. Originally differentiated from anatoxin-a because the affected dogs showed extremely salivation (hence the S), this toxin is commonly reported in the prairie states. However anatoxin-a(S) symptoms are identical to organophosphate pesticide intoxication and hence its occurrence is likely under-reported in this and in other regions of the country.

Paralytic Shellfish Poisoning (PSP) Toxins. These freshwater toxins are produced by selected strains of blue-green algae and are very similar or identical to the PSP neurotoxins produced by marine red-tide dinoflagellate species. Blue-green algae produce a larger variety of PSP toxins than their marine counterparts, with almost twice as many different variations in chemical structures. Several toxins, including saxitoxin, are produced by both freshwater and marine algae. Saxitoxin is considered one of the most potent “non-protein” toxins known with a toxicity about 1000-times greater than cyanide. Saxitoxin is a regulated biological warfare agent listed in the Public Health Security and Bioterrorism Preparedness and Response Act of 2002 through the “Select Agents” program. Production of PSP toxins have been associated with domestic and wild-life fatalities in the United States, but a major bloom {to my knowledge} has never occurred in a drinking water supply reservoir. Such blooms have occurred in Australia, leading to the deaths of tens of thousands of livestock and forcing entire communities to shift to bottled or tanker water supplies.

Golden Algae Fish Toxins. Most toxic freshwater blooms are caused by cyanobacteria; however there are a number of non-cyanobacterial toxic species that are important in saline or aquaculture facilities. Perhaps the most problematic are the blooms of the fish-killing species *Prymnesium parvum*, which is also referred to as a “golden algae”. These blooms were first suspected of causing massive fish kills in Texas in the mid 1980’s. Since that time, the blooms of golden algae have expanded and been reported in nine southern states (TX, NM, CO, NC, SC, GA, AR, AL and OK) and are suspect in two others (NE, FL). These blooms have been estimated to kill more than 17 million fish worth over \$6.5 million dollars in Texas alone and now threaten the survival of several endangered or threatened fish species. They continue to result in the loss of millions of dollars to local economies due to a decrease in fish-related tourism each year.

In addition to these major categories described above, there have been a large number of different toxic events where either the bloom was not directly associated with the die-off of an easily observable species, or where a definitive cause and effect relationship has not be established. Harmful algal blooms can cause dramatic changes in ecosystems through the effect on the lower food web. They may also be responsible for many of the common ailments (e.g. swimmers itch) experienced by recreational users that come in contact with these blooms.

Occurrence and Causes of Freshwater HAB’s in the United States.

As evidenced from the discussion above, freshwater toxic algal blooms are not confined to any geopolitical boundaries. Blooms of toxic blue-green algae are widespread and have occurred in all 50 states of the United States (Figure 1). While not all blooms of cyanobacteria are toxic, many of the toxic species are cosmopolitan or widely distributed between ecosystems. Blooms are not confined to large lakes such as the Great Lakes, but occur in all sorts of water bodies ranging from smaller prairie potholes, rivers, reservoirs, impoundments to large lake ecosystems.



Figure 1. The approximate location of documented freshwater cyanobacterial harmful algal blooms in the United States through 2005. This map is not a complete representation of all toxic cyanobacterial outbreaks, but illustrates their widespread occurrence. (modified from “*Scientific Assessment of Freshwater Harmful Algal Blooms*”, Inter-agency working, IWG-4H)

A recent study by a NOAA-sponsored regional program to look at the occurrence of toxic algal blooms in the lower Great Lakes (MERHAB-LGL) has found that 50% of the samples collected from western Lake Erie over the last decade contained detectable levels of blue-green algal toxins. A significant fraction of these samples also exceeded the World Health Organization’s guidelines for safe drinking water. As the Great Lakes in total contain more than 84% of North America’s fresh surface waters, 22% of the world’s fresh surface waters, and currently provide drinking water for more than 40 million people, broad scale efforts to protect these essential resources from HABs are essential.

These studies in the Great Lakes are not an exception. Similar to the Great Lakes, there has been a rapid proliferation of toxic cyanobacteria blooms in other freshwater ecosystems, including those in the Northeast (VT, NY), Midwest (NE, IA), southern (FL), and western states (NM, CO, OR). Broad scale studies in Europe and the mid-western United States have shown a similar high percentage (~50%) of their blooms contain toxic species and/or toxins. Each year, new toxic blooms are reported in areas where they have not been previously reported. The increased number of scientific papers on freshwater harmful algal blooms over the last several decade, the increased numbers of reports in the popular press, and the increase in health advisories due to cyanobacterial toxins all suggest that, if anything, the occurrence of toxic freshwater blooms has increased over the past 30-40 years.

Causes and Costs of Freshwater Harmful Algal Blooms.

Blooms of freshwater algae, especially cyanobacteria, are triggered by a number of factors. These include, but is not limited to:

- Increases in nutrient loading from point and non-point sources. Like all plant species, freshwater algae must obtain the basic building blocks of nitrogen and phosphorus needed for growth. These are often obtained through runoff due to agricultural or land-use practices in the surrounding watershed. For example, the US-EPA has recently started extensive efforts to look at nutrient inputs from the watersheds surrounding the Maumee River region [near Toledo, OH] in western Lake Erie as a causative factor for the large blooms of toxic blue-green algae that occur in the western basin.

- Extended periods of high solar radiation that promote photosynthesis. This means bright, sunny days such as those that often come at the end of the summer season.
- Warm temperatures that can accelerate the growth of the organisms and lead to thermal stratification or separation of the water column into distinct layers.
- Calm wind conditions that also lead to a stable water column with little mixing. This can allow buoyant species to rise to the surface and shade competing species. Changes in hydrology, such as the formation of an impoundment in a normally flowing river may also increase the intensity and occurrence of bloom events.
- Changes in the ecosystem through the introduction of invasive species such as dreissenid mussels that selectively feed on non-cyanobacterial species. This can provide a selective pressure for the formation of selected species.

These general conditions lead to increased blooms of all algae, not specifically harmful or toxic algal species. We have a very poor understanding of those environmental factors that specifically lead to the formation of a toxic bloom over a non-toxic bloom. This lack of basic scientific research has hampered our efforts to design specific remediation techniques for freshwater HAB's, to forecast the occurrence of toxic blooms, and to predict the effects of large-scale ecosystem changes such as global climate change on freshwater harmful algal blooms.

For the same reason, it is difficult to provide an economic assessment for the costs associated with a toxic freshwater algal bloom in comparison to the costs associated with a non-toxic bloom. Dodds and coworkers from Kansas have calculated that the annual value of losses in recreational water usage, waterfront real estate, alteration of ecosystem structure, loss of endangered species, fish kills, and impacts on drinking water exceed \$2.2 billion dollars annually as a result of eutrophication in U.S. freshwaters due to increased nutrients and the resulting algal growth. Not all of these expenses are due to harmful algae, but anecdotal information provided by large water providers in states such as Florida suggest that their treatment costs needed to ensure water safety may increase more than a \$100,000 per week in response to a toxic cyanobacteria bloom. Added to this would be the millions of dollars in lost recreational activities, monitoring and response expenses, health care costs, and damage to the aquaculture/fishing industry. In addition, there are also costs where it is difficult to assign a monetary value, e.g. what is the value for the loss of an endangered or threatened species or permanent changes to an ecosystem? Our nation's freshwaters have faced increasing stress due to rising population pressure, land use changes, and the increased demand for freshwater resources. Once a harmful algal bloom becomes established in a given ecosystem, it is very difficult and costly to reverse the situation. Research funds spent understanding the basic science surrounding a toxic bloom, followed by translation of that knowledge into specific prevention, control, and mitigation technologies are funds well spent in the long run.

NEED FOR A NATIONAL PROGRAM FOR FRESHWATER HABs.

Congress passed the Harmful Algal Bloom and Hypoxia Research and Control Act in 1998 to authorize funds for research on marine HABs and hypoxia. This act was expanded in the 2004 Reauthorization Act to include all freshwater bodies. This latter act also calls for a series of reports to clearly assess the status and outline our research needs. Members of the scientific

community and the Interagency Working Group on Harmful Algal Blooms, Hypoxia and Human Health (IWG-4H) have prepared a number of reports, including the “*Scientific Assessment of Freshwater Harmful Algal Blooms*”, “*Cyanobacterial Harmful Algal Blooms, State of the Science and Research Needs*”, and the “*Harmful Algal Blooms Research Development Demonstration and Technology Transfer*” (HAB RDDTT) report. These reports clearly document an increased awareness on a national scale of impacts such as toxin-contaminated drinking water or seafood, mortality of fish and wildlife, damages to aquaculture enterprises, economic losses in coastal and lakeside communities from HABs and the impacts on Public Health. They also clearly identify the research needs and limitations to progress, as well as provide a path forward to protect against long-term ecosystem change.

The marine HAB community has benefited tremendously from the initial 1993 national plan for harmful algal blooms and the subsequent formation of the competitive, peer-reviewed, merit-based interagency research program in Ecology and Oceanography of Harmful Algal Blooms (ECOHAB) and NOAA’s Monitoring and Event Response of Harmful Algal Blooms (MERHAB), as called for in the Harmful Algal Bloom and Hypoxia Amendment Act of 2004. These programs have led to dramatic increase in our understanding of marine bloom events, increases in detection technology, improvements in event response, a better understanding of the societal aspects of harmful algal blooms, and an overall improvement in coordination between agencies.

Freshwater HABs and their root causes do not respect geopolitical and agency boundaries. Thus a regional and multiagency approach is again required. We need a similar emphasis on freshwater ecosystems, including, but not limited to, the Great Lakes ecosystems. As called for in the *Scientific Assessment of Freshwater Harmful Algal Blooms*, prepared by the IWG-4H, a successful freshwater HAB program must foster collaboration between agencies, minimize unnecessary duplication, and provide the essential resources for those agencies to carry out their mission. Furthermore, Congress must all authorize sufficient funding levels for each of these programs (Freshwater HABs, ECOHAB, MERHAB and Prevention Control and Mitigation) if they are collectively to have a chance for success, not simply shift funds from one to another.

Of key importance is the question of which agency should direct this important endeavor for freshwater systems. The Department of Commerce through NOAA has a mandated requirement to protect our marine environments, the Great Lakes and estuaries. However, the Clean Water Act of 1968 and the Safe Drinking Water Act of 1974 (and amendments) provide for US-EPA oversight of our nation’s freshwater resources that we use for drinking, swimming and recreational purposes. Previously, EPA, NOAA and other agencies joined together in soliciting research proposals for funding from the ECOHAB competitive research-grant program. Each agency reviewed and selected research proposals for funding by their agency that were appropriate for the agency’s mission. Unfortunately, due to a lack of clear authority from Congress and limited funding resources to research, monitor, control and prevent freshwater HABs, the EPA has withdrawn or limited its support for HAB research grant programs over the past several years. EPA participation in HAB-related programs and funding HAB research is essential and EPA needs to reestablish their participation in those grant programs. Critical research is needed to assess the frequency and concentrations with which cyanobacteria and cyanotoxins occur in recreational and finished drinking waters. Health research is needed to obtain the dose-response data needed to set limits for safe exposure to cyanobacterial toxins, and for determining cancer assessments. Alternative routes of exposure such as fish consumption

need to be carefully evaluated in these risk assessments. Risk management research is needed to assess the efficacy and sustainability of ecological and chemical approaches to freshwater HAB control, to develop improved and less expensive control technologies, and to devise enhanced mitigation strategies. New techniques in molecular biology, biochemistry and chemistry need to be applied to this problem as we constantly challenge the classical definitions of what is a “toxic” or “non-toxic” bloom. Thus, all of these recommendations and technologies need to be based on the best available science in this rapidly changing field.

The organisms and causes of freshwater HABs are very different from those that cause marine HABs, and therefore potential control and remediation technologies are also likely to be very different between marine and freshwater systems. A freshwater HAB program that specifically addresses those differences is needed. These freshwater locations need to extend beyond the Great Lakes into other impacted large water bodies such as Lake Champlain (VT) and Lake Mead (NV, AZ) and even to smaller freshwater ecosystems such as the Klamath River (CA, OR) or Elk Creek (OR) which suffer from freshwater HAB's. Congress needs to provide the US-EPA with a clear statutory mandate to participate in freshwater HAB research, and authorize funding for that research. The EPA, working with other affected agencies, needs to develop a comprehensive National Freshwater-HAB Research and Control Program, just as NOAA has done for HABs in oceans, estuaries, and the Great Lakes, and the US-EPA needs to work with NOAA in administering this program for the betterment of all. Congressman Baird's and this committee's legislation accomplishes all of these goals related to freshwater HAB programs and I commend you for recognizing this deficiency.

Smaller freshwater lakes and rivers are very different from larger freshwater systems such as the Great Lakes, which are in turn very different from our estuaries and coastal systems. In spite of these differences between freshwater, estuarine, and marine HABs, it is essential to realize that these water-body types are intimately interconnected; nutrients that enter waterways through their upland watersheds continually stimulate HABs as they flow from the smaller streams, to the larger freshwater lakes, to estuaries and finally to our coasts. Holistic legislation that addresses both marine and freshwater HABs is needed if we are to understand, control and remediate the problem of harmful algal blooms that occur within all our nation's waters.

Thank you for the opportunity to express my viewpoint.