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09-JULY-08

Science Office

Representative Nick Lampson
Chairman, Subcommittee on Energy and Environment
U.S. House of Representatives
Suite 2320 Rayburn House Office Building
Washington, DC 20515-6301

Dear Representative Lampson:

Thank you for inviting me to testify at the Subcommittee on Energy and Environment's hearing on Harmful Algal Blooms: The Challenges on the Nation's Coastlines. I understand that the hearing will be held on Thursday, July 10, 2008 at 10:00 a.m. in room 2318 of the Rayburn House Office Building. I am pleased to accept your invitation to provide testimony at the hearing.

My written testimony is attached below.

Sincerely,

H Kenneth Hudnell, PhD

Hilton Kenneth (Ken) Hudnell, PhD
Vice President & Director of Science
SolarBee Inc.

Congressional Testimony
H Kenneth Hudnell, PhD
09-JUL-08

Good morning to all in attendance today. Chairman Lampson and Ranking Member Inglis, thank you for inviting me to testify before the House Energy & Environment Subcommittee today concerning harmful algal blooms (HABs) in our Nation's freshwater bodies.

I am Dr. Hilton Kenneth Hudnell. I served as a neurotoxicologist in the US Environmental Protection Agency's (EPA) National Health and Environmental Effects Research Laboratory for 23 years. I focused on the human health effects of biotoxins, toxins produced by single cell organisms, for the last dozen years. I led an interagency effort to provide the scientific basis for developing a National Research Plan to address the risks of freshwater HABs - http://www.epa.gov/cyano_habs_symposium/, as mandated by the Harmful Algal Bloom and Hypoxia Research and Control Act (HABHRCA, as reauthorized in 2004). That effort culminated this year in the publication of a book entitled, *Cyanobacterial Harmful Algal Blooms: State of the Science and Research Needs* (1) - http://www.springer.com/biomed/neuroscience/book/978-0-387-75864-0, and the Congressionally mandated report, *Scientific Assessment of Freshwater Harmful Algal Blooms* (2). I am currently Vice President and Director of Science for SolarBee, Inc. - http://www.SolarBee.com/, a company that makes solar powered water circulators to solve water quality problems such as HABs, and an adjunct professor in the University of North Carolina at Chapel Hill's Institute for the Environment - http://www.ie.unc.edu/content/about/people/listing.cfm. Recently I was elected to the National HAB Committee, headquartered at Woods Hole, Massachusetts.

Whereas Drs. Anderson, Ayres and Magnien's testimony primarily concerns HABs in our oceans, estuaries and the Great Lakes, I will talk with you about HABs in our Nation's inland lakes, ponds, reservoirs, streams and rivers. Just as salt levels differ between freshwater, estuaries and oceans, so do their ecosystems and the organisms that cause HABs in those water bodies. Some of the causes of HABs in those environments are the same, such as over enrichment with nutrients. But it is important to understand the differences if we are to successfully develop strategies for controlling the increasing risks of freshwater HABs to human health, the sustainability of aquatic ecosystems and our Nations economy. Today I will discuss:

- *Freshwater HAB cells and their toxins*
- *Freshwater HAB risks for human health and ecosystem sustainability*
- *Occurrence, causes and costs of freshwater HABs*
- *Approaches to freshwater HAB control*
- *The need for improved legislation to comprehensively address HABs from freshwater (EPA jurisdiction) to oceans (National Oceanic and Atmospheric Administration (NOAA) jurisdiction)*

Freshwater HAB cells and their toxins

Freshwater HABs are primarily caused by cyanobacteria, although similar organisms such as golden algae also cause some of the freshwater blooms. Cyanobacteria (a.k.a. blue-green algae) are single-cell organisms that appear in the fossil record from about three billion years ago. This was a time when there was no oxygen in our atmosphere. They were the first organisms to use the photosynthetic process. They filled our atmosphere with oxygen, enabling the existence of life forms such as our own. Cyanobacteria have proven to be highly resilient organisms, surviving and even thriving over the eons as dramatic shifts occurred in the physical and chemical characteristics of our air, water and land. For example, some are able to "fix" nitrogen; they can take unusable forms of nitrogen from the air or water and change it to forms they can use for nourishment. Some are able to regulate their position in the water column through buoyancy control so they can make maximum use of sunlight or nutrients at optimal times. Now they are found in virtually all ecosystems, but are primarily a problem in our fresh-to-brackish waters. The first problem is that cyanobacteria "bloom" when conditions are right. They rapidly expand their population from a few cells per milliliter of water to dense mats of organic material floating on the water's surface or suspended in the water column. These huge masses of organic material create serious problems for humans and aquatic ecosystems, as explained below. The second and more serious problem is that cyanobacteria often produce cyanotoxins, some of the most potent toxins known. It's as if a single cobra could become a hoard of cobras overnight, injecting their toxic venom into the environment of all living things.

Cyanobacteria genera are known by tongue-twisting names such as *Microcystis, Aphanizomenon, Planktothrix, Anabaena, Cylindorspermopsis* and *Lyngbya.* Not all types of cyanobacteria are thought to make toxins, although the ones I named can make multiple toxins. We don't know what triggers their production of toxin, or what causes toxin production to stop. We don't even know why they produce toxins; the toxins are not essential for the cells to live. However, it is generally thought that the toxins provide some survival advantage. For example, the toxins kill some organisms with which cyanobacteria compete for space to grow and multiply. The toxins also inhibit grazing by some organisms that otherwise would be their predators. Zooplankton that graze the good, "edible" green algae, the base of the aquatic food chain, often avoid grazing the "inedible" blue-greens. Some filter feeders such as the zebra mussel seem to selectively "spit out" toxic cyanobacteria cells. The arrival of zebra mussels in the Great Lakes coincided with the resurgence of HABs in the Great Lakes in recent years, and some scientists postulate this to be a cause and effect relationship. We do know that many genera of cyanobacteria make not only one type of toxin, but multiple types of toxins. We also know that many genera make the same toxins. Other types of plankton also make some of the toxins made by cyanobacteria. An example is highly potent saxitoxin, the cause of Paralytic Shellfish Poisoning, made by both marine dinoflagellates and cyanobacteria. The genes responsible for toxin production are distributed widely within the planktonic world.

The cyanotoxins are often named after the organism first discovered to produce the toxin, such as microcystins, cylindrospermopsins and anatoxins. These are thought to be

the priority toxins in the U.S. because of their high potency and frequent occurrence. Not enough is known about saxitoxin occurrence in U.S. freshwaters to determine if it should be a priority cyanotoxin.

Cyanotoxins are among the most potent toxins known, far more potent than industrial chemicals. They cause death at dosage levels in the low parts per billion range. For example, the toxins named above are more potent than strychnine, curare (the poison dart toxin) and sarin (a nerve gas). One of the anatoxins is equivalent in potency to cobra venom. Only a few toxins are more potent than cyanotoxins, such as botulinum toxin (botulism) and ricin (derived from the castor bean). As little as a mouthful of lake water containing cyanotoxins can have immediate lethal and sub-lethal health effects.

The toxins are usually placed into one of three categories: 1) liver or hepatotoxins, such as the microcystins; 2) neurotoxins, such as the anatoxins and saxitoxins, and; 3) non-specific toxins, such as the cylindrospermopsins. The classification is based on the organ system in which failure is the cause of death at higher doses. However, it is a mistake to think that any of these toxins affect only one organ system. Lower dose exposures to many cyanotoxins result in multiple-system symptoms, gastro-intestinal distress and flu-like illness.

Freshwater HAB risks assessment: human health and ecosystem sustainability

HAB risk assessment. Whereas NOAA led the development of a National Research Plan for addressing HABs in oceans, estuaries and the Great Lakes, described in HARRNESS, 2005, *Harmful Algal Research and Response: A National Environmental Science Strategy* (3), there is no National Research Plan for addressing HABs in our rivers, streams, ponds, reservoirs and other lakes. Although many risks of freshwater HABs and their toxins for human health and aquatic ecosystem sustainability are well known, there are no Federal guidelines or regulations concerning HAB cells or toxins in U.S. drinking or recreational waters. The National EPA placed cyanobacteria, other algae and their toxins on their first Contaminant Candidate List (CCL) for drinking water toxins in 1998. Regulatory determinations concerning contaminants on the CCL, and revised lists, are to be made within each 5-year period. However, no determinations have been made for cyanobacteria and cyanotoxins. They are currently on the draft CCL3 list. The National offices of the EPA have made no regulatory determinations concerning HABs in recreational waters.

The World Health Organization (WHO) and a number of countries have developed guidelines or regulations for a few genera of cyanobacteria and their toxins. For example, the WHO developed guidelines for *Microcystis* and microcystins. Numerous mortalities in Brazil led to the first regulations on microcystins in drinking water. In the U.S., states and localities confronted by HAB risks are increasingly relying on the WHO guidelines to develop strategies for protecting human health. States developing guidelines for cyanobacteria include California, Florida, Iowa, Nebraska and Oregon. This year the Regional EPA office in Sacramento ordered that California develop the first ever Total Maximum Daily Load (TMDL) for a cyanobacterium and cyanotoxins in the U.S. The TMDL order requires California to develop a plan to prevent dangerous levels of *Microcystis* and microcystins in portions of the Klamath River. The Klamath regularly

experiences some of the highest levels of these cells and toxins seen anywhere in the world. Contentions have developed between some state and local agencies as localities hurry to develop regulations concerning fertilizer usage before state legislation preempts such actions. Federal leadership is badly needed to assist states and localities in meeting the challenges HABs pose for human health, ecosystem sustainability and economic vitality.

Human health effects. Exposures to cyanotoxins occur in recreational and finished drinking waters. High-level exposures generally occur through ingestion of recreational waters. Lower-level exposures occur through inhalation and dermal contact. Data from Florida indicate that toxin levels in finished drinking water often are higher than in source waters. HAB cells are lysed or split open when filtered during water processing. The lysed cells release their toxin load into the water. Normal drinking water processing often does not remove the toxins. There is no known and affordable method to remove all cyanotoxins from drinking water. Few, if any, water utilities systematically monitor for HAB toxins. However, high-level exposures through drinking water are probably rare. Cyanobacteria often make non-toxic compounds that cause noxious tastes and odors. Water utilities become aware of the taste and odor problems. They either implement supplemental treatment processes at high cost, or discontinue drawing water from the contaminated source until the tastes and odors dissipate. The additional processing to remove taste and odor compounds may reduce toxin levels sufficiently to prevent the most serious, acute health effects. However, humans are repeatedly exposed to lower levels of cyanotoxins in tap water. There is potential for higher-level exposures because many HABs go undetected; many do not produce taste and odor compounds or form surface scums. The potential for cyanotoxin exposure through drinking water is high because 2/3s of the U.S. population's tap water now comes from surface-water sources. Cyanotoxins in potable and recreational waters have caused acute human-health effects in the U.S. and many other countries.

HAB toxins pose serious risks for human health, as well as the health of domestic and wild animals. The health effects are generally placed in one of three categories.

Acute health effects - Swallowing a mouth full of contaminated water could cause serious injury or death due to respiratory arrest or organ failure. Lower level exposures cause a multi-system, flu-like illness. Every year there are multiple reports of animal deaths in the U.S. due to cyanotoxin exposure. Some states have HAB surveillance systems based on telephone hotlines for reporting animal deaths after water body contact. Occasionally there are reports of human deaths. For example, boys from a high school soccer team swam in a golf course pond after practice in Wisconsin during the summer of 2002. Two of the boys were horsing around, dunking each other under the water. They soon developed gastro-intestinal distress and then seizures. One boy died from respiratory arrest. Luckily, the other boy survived. *Anabaena* were found in stool samples taken from both boys. The coroner attributed the cause of death to anatoxins. The boys swallowed the "cobra venom".

Our book (1), mentioned earlier, has a chapter describing the Nebraska experience with HABs. State officials first noticed HABs in their surface waters during the summer of 2004. They determined that the HABs were predominated by *Microcystis* species. The

state implemented a monitoring program for microcystins in surface waters, and developed action levels based on WHO guidelines for increased monitoring and closure. Over 700 samples were taken from 111 different surface water bodies during 2004. Sixty-nine health advisories (increased monitoring) and 26 health alerts (lake closures) were issued in 2004. Some closures lasted for more than 3 months. The great benefit to public health provided by the Nebraska HAB action plan became evident when a mistake was made in 2004. Toxin levels exceeding the health alert level for lake closure were observed in the popular recreational lake, Pawnee Lake, on a Friday. Officials were instructed to post signs at lake beaches notifying the public that the lake was closed due to cyanotoxins. However, only one beach was posted. The public used other beaches and the rest of the lake that weekend. The state received over 50 reports the following week of severe gastro-intestinal and flu-like illness in people that recreated on Pawnee Lake the previous weekend. The actual number of poisonings may have been much higher. It is believed that most physicians do not recognize illnesses as being caused by cyanotoxins.

Chronic health effects - Most non-lethal cases of acute cyanotoxin poisoning recover within days or weeks. However, an unknown percentage of susceptible individuals continue to suffer neurological and other symptoms for many months or years. Although few studies have investigated chronic illness caused by algal toxins, the phenomenon is best described in the literature on chronic *Ciguatera*-seafood poisoning. It is estimated that a 1,000,000 people worldwide may contract *Ciguatera*-seafood poisoning yearly due to the consumption of reef fish contaminated with ciguatoxins. The U.S. Centers for Disease Control and Prevention (CDC) estimates that only 2-10% of *Ciguatera*-seafood poisoning cases in the U.S. are recognized or reported. Approximately 20% of acute illness cases are thought to develop a chronic condition characterized by extreme fatigue, weakness, muscle pain, sensory abnormalities, and cognitive deficits. The scientific literature contains reports of chronic illness following acute exposure or repeated, low-level exposure to cyanotoxins, although scientific studies of the condition are lacking.

Last month I reported a study of chronic illness in cyanotoxin-exposed patients at a medical conference in Costa Rica. My research colleague, Ritchie Shoemaker, MD, who specializes in biotoxin-associated illness, collected the data. The 17 patients had residential and/or recreational exposure to freshwater bodies that regularly experienced HABs predominated by *Microcystis.* The average duration of illness was about 2 years. Most patients previously consulted numerous physicians and received numerous diagnoses, none of which involved toxins. The patients displayed statistically significant and severe deficits in vision, multiple-system symptoms, and biochemical abnormalities, relative to control study participants. The biochemical abnormalities indicated exposure triggered an inflammatory process. Illness resolved and symptoms dissipated during cholestyramine therapy. Cholestyramine is a non-absorbable polymer that binds many toxins, cholesterol, and salts from bile in the intestines, causing them to be eliminated rather than re-absorbed during enterohepatic recirculation. These and other data indicate that there may be many unrecognized cases of chronic illness in the U.S. and world wide that are caused by algal and other biotoxins.

Delayed health effects - Little is known about the effects of repeated, low-level exposures, but cancer & neurodegeneration are outcomes implicated in the scientific literature. For example, laboratory studies indicate that microcystins are a cause and

promoter of liver, colon and other cancers. Microcystin levels in drinking water were associated with liver cancer incidence in Chinese epidemiological studies. Other studies indicate that cylindrospermopsin and other cyanotoxins also may be carcinogenic.

The results from decades of studying a neurodegenerative complex common among natives of Guam recently spurred research on Alzheimer's disease and the cyanotoxin, β-Methyl Amino Alanine (BMAA). Scientists reported a high incidence of a neurologic condition with aspects of Parkinsonism, Alzheimer's disease and Amyotrophic Lateral Sclerosis among the Chamorro population of Guam in the 1940s. The leading causative agent is now thought to be BMAA. The cyanobacteria genus *Nostoc* grows on the roots of the cycad plant and produces BMAA. BMAA accumulates in the seeds of the cycad. A species of flying fox feeds on the seeds and accumulates high levels of BMAA in its tissues. The flying fox was a traditional food of the Chamorro. Autopsy studies showed BMAA in case, but not control, brains. As the flying fox population decreased to near-extinction levels, the incidence of the Guam dementia complex decreased dramatically. Recent research produced two important results. First, BMAA is produced by most or all genera of cyanobacteria, and is often present in surface waters. Second, BMAA was observed in Canadian Alzheimer brains, but not in control brains. Evidence to date for a causative relationship between BMAA and Alzheimer's is far from conclusive, but the potential ramifications are enormous. Current studies continue to investigate the Alzheimer's-BMAA relationship, while others investigate cancer and cyanotoxin linkages.

Ecosystem effects - HABs adversely impact many trophic levels of aquatic environments through a variety of mechanisms during bloom formation and collapse. As blooms form, the increased biomass of the cells reduces water transparency. Resulting light limitations inhibit the growth of plants, epiphyton, benthic algae and other phytoplankton. Water alkalinity increases as the expanding biomass consumes carbon dioxide, altering phytoplanktonic interactions and causing lethal and sub-lethal impacts on fish populations. Cyanotoxins augment and expand these effects as fish, zooplankton, macro-invertebrates, wading birds and aquatic vertebrates suffer further lethal and sub-lethal effects. For example, data from Florida show strong correlations between *Cylindrospermopsis* and cylindrospermopsin concentrations and alligator death rates. Another example is a new syndrome among wading birds such as coots. The new syndrome, termed avian vacuole myelopathy, was first discovered in the U.S. southeast during the mid-1990s. After feeding on plants such as hydrilla in lakes, birds were observed to swim and fly erratically before dieing. Autopsies revealed vacuoles or holes in brain and spinal cord nervous tissues. The cause of death is believed to be an as yet unidentified toxin produced by a newly discovered cyanobacterium in the order of stigonematales that colonized aquatic plants. The lethality extended to predatory birds such as bald eagles as they easily captured and consumed the impaired wading birds.

Bloom collapses often are associated with massive fish mortality. HABs can completely infest smaller lakes, reservoirs, ponds and long stretches of slow moving rivers. Cell densities can soar, creating think mats of organic material that completely block out light. Eventually, cold weather or other natural causes lead to a gradual collapse of the blooms. The cells are lysed, release all of their toxins into the water column, and sink to the bottom. Bacterial and other aerobic processes deplete oxygen in the water column as the cells are decomposed. At first bottom waters, and then upper levels of the water

column, become hypoxic (no oxygen) or anoxic (low oxygen). Fish that are unable to escape to oxygenated waters die, often in massive quantities. The decomposition of cells and fish trigger a vicious cycle. The lack of oxygen at the sediment-water interface causes chemical bonds to be broken, releasing nutrients (e.g., phosphorus) and toxic, noxious gasses (e.g., hydrogen sulfide) from the sediment to the water column. Because phosphorus is often a limiting agent for bloom formation, the release of this and other nutrients sets the stage for new bloom formations. Repeated bloom cycles may irrevocably alter aquatic ecosystems, extinguishing biota that contribute to healthy ecosystems, while creating conditions for continued bloom dominance.

Occurrence, causes and costs of freshwater HABs

HAB occurrence. There is widespread agreement among scientists, water managers, local officials, and much of the general public that the occurrence of freshwater HABs is rapidly increasing in the U.S. and worldwide. Every year freshwater HABs occur where they previously have not been observed. HABs are lasting longer than before. Freshwater HABs occur in all parts of North America, and durations range from the summer months in more northern areas to year round in more southern areas. HABs may be readily visible due to the presence of surface scums, or difficult to detect because some types bloom only at mid-level depths. Although there are no National databases on freshwater HAB occurrence, and only a few state or local databases, the evidence for increasing spatial and temporal occurrence of freshwater HABs is undeniable.

HAB causes. Freshwater HAB incidence and duration is increasing because of increasing nutrient input into our water bodies, and rising temperatures. Climate change is driving much of the increase. Average temperatures on land and in water are increasing, an advantage for HAB organisms over many types of beneficial algae. The frequency of storms, heavy rainfalls and flooding is increasing, causing more nutrients to be washed into our water bodies. Somewhat ironically, the frequency of droughts is increasing at the same time. Slow-moving or stagnant waters favor HABs over beneficial algae. Warm, quiescent, and nutrient enriched waters provide the ideal setting for freshwater HABs.

There are four primary requirements for HAB occurrence - nutrients, warmth, sunlight and calm water. HAB cells thrive and multiply only when sufficient nutrients are available. Cyanobacteria and other algae require carbon, nitrogen, phosphorus and some trace elements to grow. Carbon is not a limiting factor; there is plenty of carbon in the air and water for algal growth. In fact, some strategies for promoting the expansion of beneficial algae have been discussed as a means of removing carbon dioxide, a greenhouse gas, from the air. Nitrogen is a limiting factor for only some types of HAB cells. When usable forms of nitrogen are low, types of cyanobacteria that can “fix” nitrogen into usable forms dominate HABs. Phosphorus is a limiting factor for all types of HAB cells. Phosphorus enrichment of our water bodies is driving much of the increase in HAB occurrence. The ratio of nitrogen to phosphorus concentrations often determines the types of cells that dominate HABs.

Types of cyanobacteria previously seen only in tropical areas have become common in much of the U.S. in recent years. This pattern is expected to continue as

average temperatures increase. Sunlight is required by cyanobacteria to produce energy through photosynthetic processes. Some of the recently invasive types of cyanobacteria such as *Cylindrospermopsis*, and others like *Planktothrix,* efficiently produce energy under low light conditions. These types of cyanobacteria often bloom deep in the water column, making them difficult to detect from the surface.

Freshwater HABs occur almost exclusively in quiescent, stagnant waters. Water flow rates decrease as an expanding population, agriculture, and industry withdraw larger quantities for use. Aquifer depletion forces increased withdrawal of surface waters and damming to create new reservoirs. These factors and droughts are decreasing flow rates and increasing the incidence of freshwater HABs.

HAB costs. Although a formal analysis of the total costs of HABs to our economy has not been conducted, it is known that freshwater HABs account for many millions of dollars in lost recreational revenue, water treatment expenditures, monitoring and response activities, health care and aquaculture losses. The development of control and mitigation technologies and processes offers an opportunity for the U.S. to avoid these losses, and create a world-leading industry. World leadership in HAB control and mitigation is needed to sustain the Earth's aquatic ecosystems, protect human health and vitalize the U.S. economy.

Approaches to freshwater HAB control

Of the four causative factors for freshwater HABs discussed above, only two can reasonably be targeted for HAB control - nutrients and calm water.

Nutrient input control. Nutrient inputs enter freshwater from both point and non-point sources. Point sources include outlets from wastewater treatment plants, urban storm-water collection systems, industries, aquacultures and concentrated animal-feeding operations. Strategies are needed to reduce the amount of nutrients entering these systems and exiting these systems. Representatives Stupak and Miller recently introduced a House bill that would require the EPA to order a reduction of phosphorus in detergents to help control HABs in the Great Lakes. Senator Levin introduced a similar bill.

An ultimate goal should be to recapture and reuse the nutrients in these systems. For example, phosphorus is not only essential for HABs, but is essential for all living organisms and required for agricultural production. There are no synthetic alternatives for phosphorus in fertilizer. Scientists around the world warn that there is a looming shortage of phosphorus on the horizon. Phosphorus production is predicted to peak within 30 years, and reserve depletion is predicted within 50-100 years. Countries such as India already face phosphorus shortages. Ultimately, there will be no alternative to recapturing and reusing phosphorus.

Non-point source inputs of nutrients to freshwater are much more difficult to control than point source inputs. Nutrients enter ditches, streams, rivers, reservoirs, ponds and lakes when rainwater washes them off of lawns, roads, highways, fields, pastures and forests. Development and expansion of watershed management plans and best

management practices for agriculture, industry and residential property are needed to reduce nutrient usage and enable the recapture of nutrients.

There is no question that reduction of nutrient inputs to freshwater benefits water quality in many ways. Additionally, systems developed to reduce nutrient inputs will likely reduce inputs of other pollutants such as pesticides, metals and pharmaceutical products. However, HAB control through nutrient-input reduction alone is a very long-term process. Many years of excessive nutrient input to freshwater bodies has resulted in high concentrations of nutrients in sediments. Nutrients in sediment are released to the water column under hypoxic and anoxic conditions, and whenever storms or other events stir up sediments. Nutrient resuspension often triggers new HABs. To my knowledge, there is no instance of sustained HAB elimination in a freshwater body of more that 100 acres in size through nutrient-input reduction alone.

Other approaches to nutrient reduction have proven to be cost prohibitive, ineffective over the long term, detrimental to the environment or a combination of these factors. For example, alum (aluminum sulfate) and other substances have been used to precipitate phosphorus from the water column to the sediment. However, this approach has the disadvantages of being effective at HAB prevention only in the short term, detrimental to much of the biota in aquatic ecosystems, cost prohibitive over the long term, and applicable only to smaller water bodies. Other approaches to nutrient reduction, such as hypolimnetic oxygenation, hypolimnetic withdrawal, dredging and biological manipulations, also have some combination of these drawbacks.

Calm water control. The calm water requirement for HABs can be targeted through hydrologic manipulations. Although excess water capacity is not usually available, increasing flow rates and decreasing water residence time eliminates HABs even in nutrient-rich freshwaters. However, the overall outcome of increased flow sometimes creates problems downstream. Nutrients in freshwater are transported to coastal environments where they stimulate HABs in estuarine and marine environments. Another approach is to destratify or artificially mix the entire water column. Diffused air system installed in ponds and smaller water bodies frequently provide good HAB control. Disadvantages include a small area of influence for each air diffuser, the continual need for electric-grid power, applicability limited to smaller water bodies due to cost and the vertical transport of nutrients sometimes stimulates HABs. The installation of artificial waterfalls or fountains in smaller water bodies often provides good HAB control with the only drawback being the continual need for grid power.

I joined SolarBee, Inc., because I believe that they developed the best technological solution to freshwater HABs in water bodies of all sizes. Two engineers in North Dakota, Joel Bleth and Willard Tormaschy, developed solar powered long-distance circulation (LDC) technology as a cost-effective alternative to aeration in wastewater lagoons. They fortuitously found that LDC not only provided the benefits of aeration at a lower cost, but also prevented the occurrence of HABs in these nutrient rich waters. LDC application for HAB control in 250 U.S. freshwater bodies to date has a success rate of about 95%.

LDC is created by floating platforms equipped with high-efficiency pumps powered by solar panels and a battery. The circulators operate 24 hours a day, seven days a week,

and are designed for a 25-year lifetime with little maintenance. The largest circulator transports 10,000 gal/min of water from the bottom of the photic zone to the surface, creating LDC sufficient for HAB control over an area of about 35 acres. LDC deployment for HAB control is unlike other "artificial circulation" approaches to HAB control in that it does not destratify the water column or aerate the hypolimnion. The circulator's intake hose is set at the base of the photic zone for HAB control, usually just above the thermocline. A plate suspended below the bottom of the intake hose causes near laminar-flow intake of water radially from long distances. The water smoothly departs from the unit radially, both above and below a disk positioned just under the surface. Only the epilimnetic water is circulated, the upper portion of the water column in which HABs occur. The thermocline or density-change barrier between the epilimnion and lower, nutrient rich hypolimnion remains intact, thereby preventing those nutrients from entering the photic zone and further promoting HABs.

Unfortunately, a chemical approach to HAB control is commonly used today. Algaecides such as copper sulfate are used to terminate blooms after they form. This reactive, as opposed to preventive, approach is dangerous for humans and has serious detrimental impacts in aquatic ecosystems. Copper sulfate lyses HAB cells, causing the release of all cyanotoxins to water instantaneously. These extreme levels of cyanotoxins in water threaten humans even if they are not directly in or on the water. Recent CDC and other evidence indicate that HAB toxins become airborne due to wind and wave action. Humans miles away from the affected water bodies inhale the toxins. The inhaled toxins cause respiratory distress in asthmatic and other susceptible populations, and may contribute to the chronic and delayed health effects discussed earlier. Copper sulfate itself is toxic to many plants and animals living in water. Furthermore, the copper binds with many pollutants such as pesticides, making them more bioavailable and damaging to aquatic organisms. Copper accumulates to high levels in sediment with continued use. As with bacteria resistant to antibiotics, there is growing evidence that some strains of cyanobacteria are becoming resistant to copper sulfate toxicity. Aquatic ecosystems will not survive repeated applications of algaecides over the long term.

HAB control summary. I believe that the combination of nutrient-input reduction and long-distance circulation provides the best approach to near- and long-term HAB control. This dual approach is sustainable, has no adverse impacts on aquatic ecosystems, provides many environmental benefits in addition to HAB control, and is cost effective over the long term.

However, research is needed to develop more efficient and effective strategies for controlling HABs in all water bodies. Specific research needs are detailed in the book (1), the mandated Freshwater report (2), HARRNESS (3), and the draft Management and Response report (4). A research plan that comprehensively addresses HABs in all of our Nation's water bodies, coordinates agency efforts and prevents duplication of effort can only be established through appropriate Federal legislation.

The need for improved legislation to comprehensively address HABs from freshwater (EPA jurisdiction) to oceans (National Oceanic and Atmospheric Administration (NOAA) jurisdiction)

Congress originally passed HABHRCA in 1998 to authorize funds for research on HABs and hypoxia. This authority through the Department of Commerce directed NOAA to conduct research and seek control of HABs and hypoxia in U.S. oceans, estuaries and the Great Lakes.

The 2004 reauthorization of HABHRCA expanded the Act to include all freshwater bodies. The reauthorization incorporated a reporting requirement by an interagency task force on freshwater blooms. The book (1) I mentioned earlier provided the scientific basis for that report. The report, *Scientific Assessment of Freshwater Harmful Algal Blooms, Interagency Working Group on Harmful Algal Blooms, Hypoxia, and Human Health* (2), describes the environmental, health and economic consequences of freshwater HABs. HABHRCA also mandated that the task force develop and submit to Congress a plan providing for a comprehensive and coordinated National Research Program to develop and demonstrate prevention, control, and mitigation methods to reduce the impacts of harmful algae. That report, *Harmful Algal Bloom Management and Response: Assessment and Plan,* is in draft form (4). It recommends the creation of a new interagency competitive-grant program, the Mitigation, Control and Prevention of Harmful Algal Blooms program (MACHAB). Implementation of MACHAB is critical for our Nation to develop cost-effective strategies for preventing HABs and mitigating their consequences. My belief in the need for a HAB control strategy is evidenced by my decision to leave the EPA and shift my research from human-health effects to HAB control technology. I believe it is much better to prevent HABs and biotoxin-associated illness than to have people in need of diagnosis and therapy due to HAB toxin exposures.

I fully support the existing HABHRCA reauthorization bills, including the “clean” reauthorization bill offered by Congressman Connie Mack, and the legislation being developed by Senator Bill Nelson that addresses some of the shortcomings of the 2004 legislation. I also support the bills to lower phosphorus levels in detergents for the Great Lakes area. However, these bills do not address the fundamental obstacle preventing the development of a coordinated National Research Plan for HABs in all of our Nations waters. Current and proposed legislation does not authorize funding for the EPA or direct the Agency to “take ownership” of the freshwater HAB problem. The current legislation authorizes funding only for NOAA through the Department of Commerce. That Department does not fund the EPA. It is the EPA that has purview over water quality in inland water bodies through the Clean Water Act and the Safe Drinking Water Act. As the lead agency with oversight over freshwater quality, the EPA must ensure the protection of “aquatic ecosystems to protect human health, support economic and recreational activities, and provide healthy habitat for fish, plants, and wildlife.” I believe that the development of a National Research Plan for all freshwater HABs is dependent on Congress passing a freshwater act that parallels HABHRCA but is specific for the EPA and all freshwater bodies.

Convincing the EPA to accept oversight responsibility for the freshwater HAB problem may not be an easy task. Since completion of the Freshwater (2) and Management and Response (4) reports, the EPA unilaterally determined that its statutory requirements regarding freshwater HABs were completed. There is no Agency effort to development and implement a National Research Plan for freshwater HABs. The Agency

virtually ceased all participation in freshwater HAB research and mitigation activities. Prior to that decision, the EPA annually contributed funds to one of the two interagency, competitive research grant programs for HAB research, the Ecology and Oceanography of Harmful Algal Blooms (ECOHAB). The EPA ceased funding that program this year. Scientists at the EPA's National Health and Environmental Effects Research Laboratory were ordered to cease all research on HABs. Staff were ordered to decline requests from the EPA regional offices and many state, local and tribal organizations seeking information on the risks and management of freshwater HABs. In taking this position, the EPA has failed to recognize the urgency of the freshwater-HAB problem, and that freshwater HAB cells differ from those that cause marine HABs, just as fresh and salt water and their ecosystems differ. Further, some of the causes of HABs and potential control technologies likely differ between freshwater and saltwater bodies. The EPA's shortsightedness can substantially harm human health, the environment and the economy. The EPA's decision to halt HAB research was likely influenced by unclear Congressional directives, a lack of budgetary authority and lower overall Agency funding. It is up to Congress to work with the EPA to correct this situation for the good of our Nation.

All Agency officials did not fail to adequately recognize the importance of freshwater HABs. The Agency's National Center for Environmental Research issued a competitive-grant request for proposals in 2007 on research to develop sensors for HAB cells and toxins. The EPA's National Center for Environmental Assessment is attempting to draft toxicological reviews for a few cyanotoxins.

My recommendation, and I believe I am joined by the vast majority of scientists in this view, is that statutory requirements are needed to direct the EPA to develop and implement a National Research Plan for freshwater HABs. Freshwater-HABHRCA legislation that parallels the current and proposed reauthorizations for HABHRCA can accomplish this goal. Congress should pass Freshwater-HABHRCA legislation that authorizes funding for, and directs the EPA to develop and implement, a comprehensive freshwater-HAB research program. This Act will create a unified approach toward protection our Nation from the risks of inland HABs, just as HABHRCA and NOAA have done for HABs in oceans, estuaries and the Great Lakes. The research should be conducted through a strong extramural, peer-reviewed, competitive-grant program and supplemented through intramural research. The Agency should be directed to fund the existing interagency grant programs, ECOHAB and the Monitoring and Event Response for Harmful Algal Blooms (MERHAB). The EPA should further be directed to help institute and fund the newly proposed MACHAB interagency grant program. The extramural grant programs will form partnerships critical to developing a successful National Program for Preventing HABs. These partnerships should include public, private, for-profit and non-profit institutions and organizations, including states, local governments, tribes, appropriate industries (including aquatic technology, fisheries, agriculture, and fertilizer), academic institutions, and nongovernmental organizations with expertise in water-quality science and management. Further, Congress must specifically authorize and appropriate funds for these freshwater-HAB research programs.

Legislation will provide clarity to the EPA that freshwater HAB research is authorized, and that the Agency must contribute to HAB research programs in order to develop solutions to the freshwater HAB problem through partnerships. The House

Science and Technology Committee is an appropriate legislative body to develop a new bill for establishing a National Research Program for Freshwater HABs because of its responsibility for the environment and jurisdiction over the EPA. I urge the members of the Energy and Environment Subcommittee to address this issue.

I am pleased to offer my expertise to help develop authority for the EPA consistent with NOAA's existing research and response programs. We must act now as a unified country to develop policy and interagency coordination to mitigate and control HABs in all of our Nation's waters. HAB toxins are far more potent than industrial chemicals, and the environmental load of HAB toxins is increasing at an alarming rate. The potential consequences of increasing HABs for human health, aquatic ecosystem sustainability and our economy are too great to ignore. Inaction is not an option that we cannot afford.

I thank the Subcommittee for allowing me to express my views today.

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