EARTHJUSTICE
COMMENTS ON PROPOSED RULE
PART II

The following section explains and illustrates the environmental and public health threats that may be caused by water transfers, including the transferring of exotic species and toxic algae.

IV. INTERBASIN TRANSFERS CAN ADD TOXIC BIOLOGICAL POLLUTANTS THAT CAN DEGRADE LARGE WATER BODIES AND DRINKING WATER RESERVOIRS AND POSE GRAVE PUBLIC HEALTH THREATS

A. Toxic cyanobacteria are the most likely biological pollutants to successfully invade a receiving water body as the result of an interbasin transfer.

Interbasin biota transfers occur when aquatic life forms such as fish, fish eggs, viruses, bacteria, vascular plants, and invertebrates are transferred from one watershed into another. Biota transfers are an inherent risk when water is transferred from one waterbody into another meaningfully distinct body of water. In a 2005 study of the risks of consequences of biota transfers potentially associated with surface water diversions between the Missouri River and Red River Basins, USGS and National Park Service scientists concluded:

> Interbasin transfers of untreated waters implemented via an open conveyance (e.g., canals) have a very high likelihood of establishing pathways to potentially promote biota transfers and subsequent biological invasions. While most of these invasions will fail in the absence of sustainable populations, such precursors to invasions will occur with near certainty. . . . [I]f interbasin transfers water transfers occur via such a mechanism, species invasions will occur and some species will establish populations in the receiving system despite any implementation practice adopted by Bureau of Reclamation or other government or nongovernment organization.[1]

Similar risks were presented by transferring water via pipeline.[2] The biota with the lowest risk of transfer was fish; that with the highest risk was toxin producing cyanobacteria (also known as blue-green algae), specifically *Anabaena flos-aquae, Microcystis aeruginosa,* and *Aphanizomenon flos-aquae.*[3] The report concluded that the greatest reduction in risk was achieved when source waters were treated within the exporting basin then transferred via closed conveyance to the importing basin.[4]

Toxic algae are biological pollutants that pose a serious and growing public health threat. At the same time that the EPA Office of Water was writing the agency memorandum that would exempt transfers of toxic biological pollutants, the USEPA, Office of Research and Development, National Health and Environmental Effects Laboratory, Neurotoxicology Division, was identifying cyanobacteria as “an increasing risk to human health and ecosystem sustainability.” In September 2005, EPA was responsible for organizing an interagency, international symposium on Cyanobacterial Harmful Algal Blooms.[5] That symposium was conducted in partial fulfillment of the mandates of the Harmful Algal Bloom and Hypoxia Research & Control Act which Congress amended in 2004 to require research into the causes, prevention, mitigation and treatment of cyanobacteria harmful algal blooms in freshwaters of the United States.[6]

In June of this year, the United States Geological Survey (the scientific research arm of the Department of the Interior), also became involved and issued a briefing sheet on cyanobacteria which announced that: “harmful algal blooms cause ecologic, economic, and public health concerns in both freshwater and marine ecosystems.” As the USGS explained:

Potential impairments include reduction in water quality, accumulation of maladorus scums in beach areas, production of toxins potent enough to cause illness or kill aquatic and terrestrial organisms, including humans, and the production of taste-and-odor compounds that cause unpalatable drinking water and fish.[7]

B. Cyanobacteria And Their Toxins

Cyanobacteria, also known as blue-green algae although they are not a true algae, are primitive life forms that typically (although not always) contain a soluble pigment that gives them their blue-green coloration.[8] In nutrient-enriched waterbodies, cyanobacteria can undergo periods of rapid and excessive growth called "algal blooms"[9] that can take over a waterbody in just a few days, often producing a film on the water that looks like green paint."[10] When these blooms begin to die and disintegrate, this pigment may color the water a distinctive blue color.[11] See Figure 2.[12]

Figure 2. Decaying Blue-Green Algae Bloom, St. Lucie River, Florida (August 13, 2005).

A "harmful algal bloom" ("HAB") is a concentration of algae that has an adverse impact on plants, animals, or humans, often due to the production of deadly liver and nerve toxins.[13] Of the approximately 60 genera of cyanobacteria worldwide, one-third produce toxins that are "harmful, and even deadly, to fish, animals, and humans."[14]

The most common toxic cyanobacteria are *Microcystis aeruginosa, Anabaena flos-aquae*, and *Aphanizomenon flos-aquae*.[15] Cyanobacteria produce: 1) "dermatoxins" that create severe dermatitis and are known tumor promoters; 2) "neurotoxins" which interfere with nerve cell function; and 3) "hepatotoxins" which attack the liver. Neurotoxins anatoxin-a and anatoxin-a(S) can cause spasms, convulsions, paralysis and death due to respiratory failure. Acute exposure to hepatotoxins, the most common of which are the toxins known as microcystins, can produce anorexia, vomiting, diarrhea, and death. Chronic exposure to microcystins is linked to gastrointestinal upset, vomiting, diarrhea, and liver cancer.

A fourth type of cyanobacteria, *Cylindrospermopsis* ("Cylindro"), produces the hepatoxin known as Cylindrospermopsin which has been responsible for outbreaks of acute hepato-enteritis and renal damage.[16] A 1999 survey that identified Cylindrospermopsin in Florida waters was the first time the toxin had been identified in North America; all samples that contained the organism tested positive for the toxin.[17] Cylindro, a "formerly tropical species," has now spread as far north as Wisconsin and Indiana.[18]

Another commonly occurring form of cyanobacteria is *Lyngbya*, known for producing a potent dermatoxin that can cause severe skin reactions along with gastrointestinal inflammation. See Figure 3.[19]

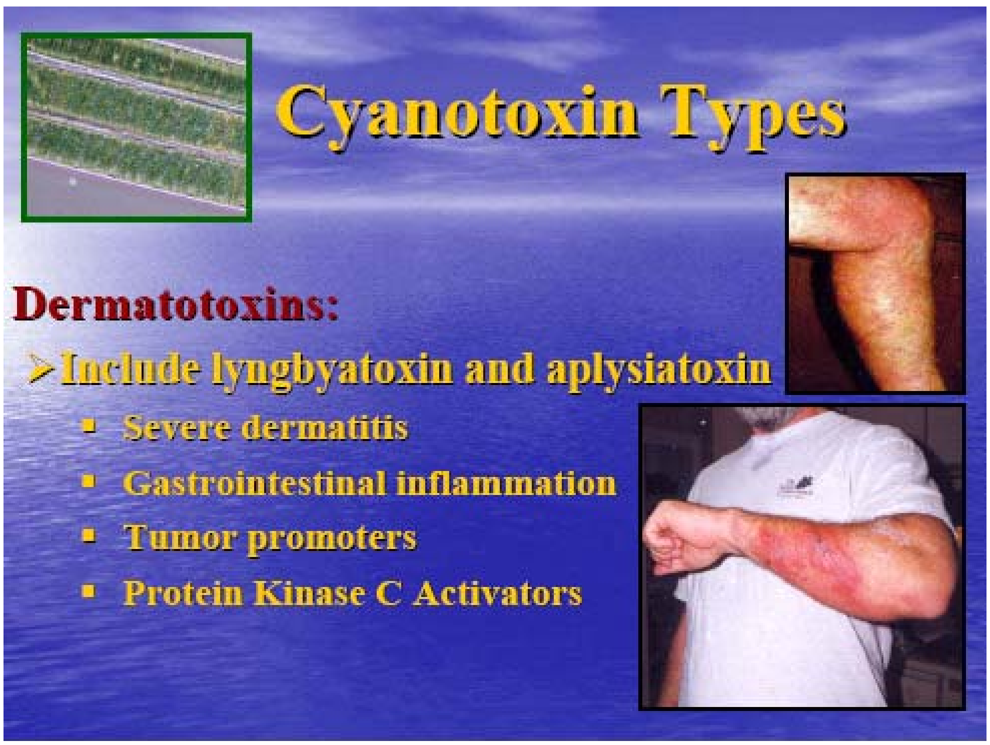

Figure 3. Photographs of severe dermatitis caused by dermatoxins.

The same toxins that affect humans and animals also affect plant tissues.[20] In a study of the effect of commonly occurring algae in Colorado reservoirs and ponds on higher plants, *Lyngbya* and *Anabaena* were both found to inhibit growth and *Microcystis* when combined with *Aphanizomenon* produced an "extreme inhibition."[21] In a more recent study it was "clearly shown" that microcystins "are inhibitors of growth and development in potato shoots and mustard seedlings under laboratory conditions" and suggested that "exposure to microcystins in irrigation water contaminated with toxic cyanobacteria poses a threat to the quality and yield of crop plants."[22] The same study found that the plants accumulated the toxins in their plant tissue, and concluded that "[t]he exposure of crop plants to microcystins via irrigation techniques has far reaching consequences for both economic and health reasons.[23] See Figure 4.[24]

Figure 4. Effect of increasing levels of microcystin toxin on the growth of mustard seedlings.

Cyanotoxins are comparable to cobra toxin in their potency; they are ten times more potent than curare, four hundred times more potent than strychnine.[25] The same cyanobacteria species can produce multiple toxins even during the same bloom.[26] While blue-green algae have "significant taste and odor constituents" (often described as a "moldy smell"), the toxins themselves have no taste, odor, or color.[27] The risk of exposure to algal toxins can come from drinking water, recreational water, residue on produce irrigated with contaminated water, and consumption of plant and animal tissue.[28]

C. Toxic algae blooms are a growing public health issue and demonstrate why EPA should not exempt water transfers from section 402 NPDES permitting requirements.

Recent awareness of public health issues related to cyanobacteria was triggered because "the number of toxic waterblooms, the economic losses from them, the health impacts and the number of toxins and toxic Cyanobacteria species have all increased dramatically in recent years in the United States."[29]

> [T]hese harmful algal blooms appear to be occurring with greater frequency in the United States as concentrated agricultural practices and population increases alter ecological cycles. . . . Increasingly in the United States, groundwater supplies aren't meeting drinking water needs and a greater reliance on surface water sources is widely predicted. An estimated 48 percent of lakes in North America are eutrophic, meaning they are high in nutrients and low in oxygen. Eutrophic conditions often coincide with the presence of cyanobacterial blooms and are considered an emerging water quality problem in the United States.[30]

For example, in Florida, although water treatment officials anticipate that groundwater demands will exceed supply by the year 2020,[31] a 1999 study of Florida waters found that: "[o]f 167 samples taken . . . 88 samples representing 75 individual waterbodies contained significant levels of toxic cyanobacterial species . . . 78% of the samples with measurable levels of microcystins and cylindospermopsin were lethal when injected into mice, and 80% of the samples showed potential tumor producing properties.[32]

As scientists have succinctly stated: "Cyanobacteria toxins have quickly risen in infamy as important water contaminants that threaten human health."[33]

D. Public Health Risks Associated with Cyanotoxins in Water Bodies Used as a Source of Drinking Water

The World Health Organization has set a drinking water guidance level for Microcystin-LR (the most common form of cyanotoxin) at 1 microgram per liter (1µg/L) which is equivalent to 1 part per billion (1 ppb).[34] Other countries' regulatory limits are shown in Figure 5.[35]

Figure 5. Microcystins Drinking Water Guidelines.

A study of finished water from 15 Florida water treatment plants found 3 plants producing drinking water that exceeded the WHO 1 ppb standard. One plant was producing water containing 8 times the recommended level of microcystin and another was producing water with 10 times the recommended level. See Figure 6.[36] The plants, however, could truthfully state that they meet all federal drinking water standards in the United States. Although USEPA officially recognized the public health risks associated with cyanotoxins in 1998 when it placed "Cyanobacteria (blue-green algae), other freshwater algae, and their toxins" on its Contaminant Candidate List (suspected drinking water contaminants that may be the subject of future regulation); no drinking water standards have ever been set.[37]

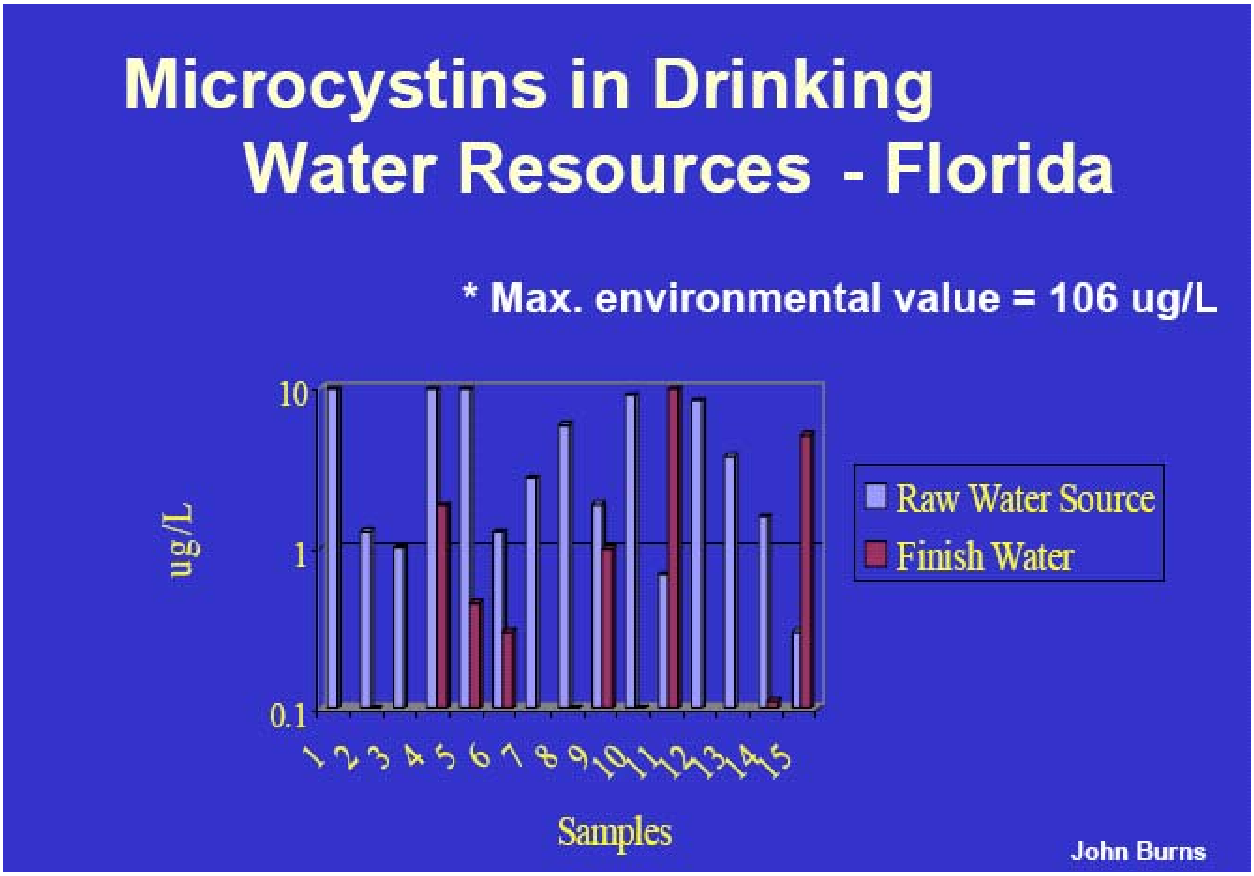

Figure 6. Microcystins in Drinking Water Resources – Florida (1 µ/L is the WHO guideline for microcystins in drinking water).

Admittedly part of the difficulty in setting a standard is identifying what needs to be done if the standard is exceeded. For example, microcystins are bound within the living cell; killing the cell simply releases the toxin into the environment. Therefore, killing the algae with disinfectants (such as chlorine) or common algaecides (such as copper sulfate) "can actually worsen the contamination problem, elevating the public health risk of toxin exposure."[38] That explains why, as shown in Figure 6, the Florida study found several plants with higher toxin levels in their finished water than in their raw water source. Higher levels of cylindrospermopsin (which were found at levels nearing 100 ppb) were also found in finished water. See Figure 7.[39]

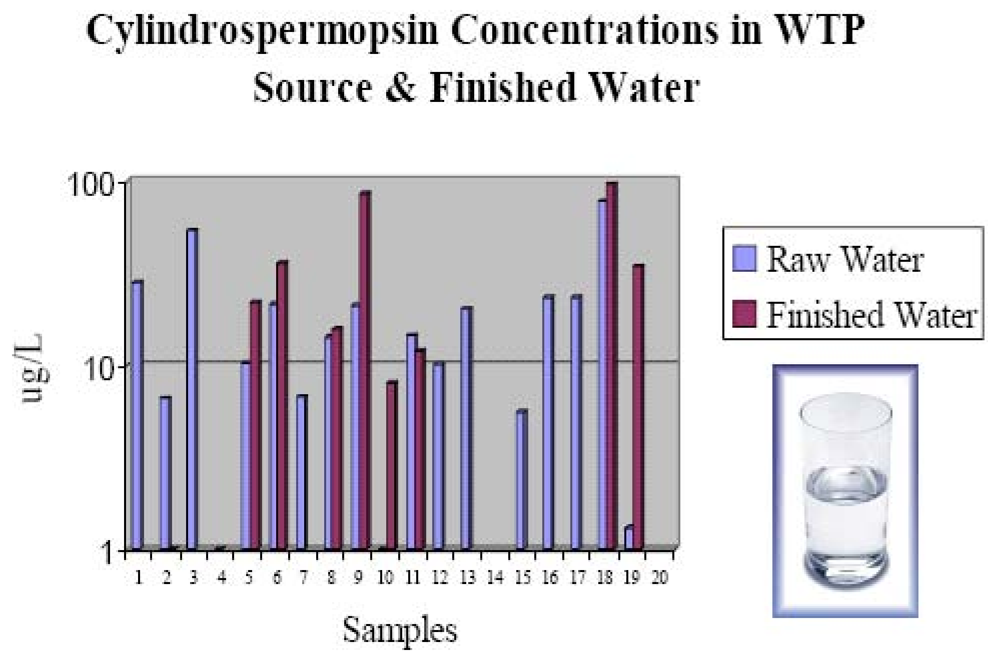

Figure 7. Results of Florida survey of drinking water. One recommended guideline value for cylindrospermopsin is 1 ppb. [40]

Acute exposure to drinking water contaminated with cyanotoxins has resulted in numerous recorded instances of kidney, liver and intestinal damage and has led to severe, gastroenteritis epidemics involving numerous deaths.[41] In 1988, drinking water from a reservoir contaminated with toxins produced by the *Anabaena* and *Microcystis* species of blue-green algae was responsible for a severe gastroenteritis epidemic in Brazil that caused 2000 cases of gastroenteritis and 88 deaths over a 42 day period.[42] In Australia, a city that drew its water from a eutrophic reservoir treated the reservoir with copper sulfate after people complained of bad taste and odor in the water.[43] Despite the fact that the city treated the water with pre-chlorination, alum flocculation, sedimentation, rapid sand filtration, and post chlorination and fluoridation, an epidemiological study of the local population found evidence of substantial liver damage occurring simultaneously with the termination of the bloom (and the subsequent release of toxins into the water from the dead algae cells).[44] *Cylindrospermopsis raciborskii* was identified as the causative agent in a similar incident where 140 children and 10 adults were hospitalized with kidney, liver, and intestinal damage within a week after authorities used copper sulfate to kill an algae bloom (and thus release toxins) in a drinking water reservoir.[45]

Low-level chronic exposure to microcystin is known to increase the risk of cancer in the liver.[46] A recent Florida study found increased risk of liver cancer associated with residence at the time of diagnosis in a surface treatment plant area of water distribution.[47]

Removal of cyanobacteria and their accompanying cyanotoxins requires, at a minimum, a multi-barrier, multiple strategy approach with advanced treatment options and frequent monitoring of treatment performance – an element crucial to safety particularly with respect to cyanotoxin removal.[48] Unfortunately initially successful treatment strategies can rapidly be compromised by source water with high loads of dissolved organic carbon (TOC) – a circumstance common in highly eutrophic, algae-filled waterbodies.[49]

E. Public Health Risks Associated with Cyanotoxins in Water Bodies Used for Recreation.

POINTS FROM HEALTH ADVISORY – LEMOLO LAKE, OREGON
JULY 12, 2006

- The public will be advised when high algae levels no longer exist.
- Until then, swallowing or inhaling water droplets from Lemolo Lake should be avoided, as well as skin contact with water by humans or animals.
- Toxins cannot be removed by boiling, filtering, or treating water.
- The Department of Human Services recommends that fish from Lemolo Lake should be cleaned of all fat, skin, and organs before cooking, since toxins are more likely to collect in these tissues.
- Major symptoms of toxin poison include numbness, tingling, dizziness and paralysis, which can lead to difficulty breathing or heart problems that require immediate medical attention.
- Minor symptoms include skin irritation, weakness, diarrhea, nausea, cramps and fainting, which should also receive medical attention if they persist or worsen.
- Children and pets are particularly susceptible to toxic poison.

Figure 8. Health advisory warnings for Lemolo Lake, Oregon (July 12, 2006).[50]

There have been “repeated descriptions of adverse health consequences for swimmers exposed to cyanobacterial blooms” and “even minor contact with cyanobacteria in bathing water can lead to skin irritation and an increased likelihood of gastrointestinal symptoms.”[51] Because cyanobacteria “have features in common with general airborne allergens” inhalation can cause allergic reactions.[52] In 1989, 20 British army recruits exhibited symptoms of “vomiting, diarrhea, central abdominal pain, blistering of the lips and sore throats” after swimming and canoeing training in water with a dense microcystis bloom.[53] “Two recruits developed severe pneumonia attributed to aspiration of a *Microcystin* toxin and needed hospitalization and intensive care.”[54]

In 2004, a similar incident occurred in Nebraska’s Pawnee Lake. A complaint concerning algae triggered sampling in the lake, the lake was found to contain *Microcystis* and *Aphanizomenon*, toxin levels of greater than 15 ppb (or 15 µ/L) of microcystin were recorded and a health alert was issued.[55] Unfortunately, only the beach on the east side of the lake was posted, and during the weekend of July 17-18, 2004, “more than 50 people complained about symptoms such as skin rashes, lesions, blisters, vomiting, headaches, and diarrhea after swimming or skiing in Pawnee Lake.”[56]

The World Health Organization and several countries (but not the United States) have recreational water guidelines. Figure 9.[57] Under the WHO guidelines, Level 1 (4 µ/L or 4 ppb) represents slight risk, Level 2 (20 µ/L or 20 ppb) represents moderate risk; and level 3 (visible surface scum) indicates that "immediate action to control scum contact should be taken."

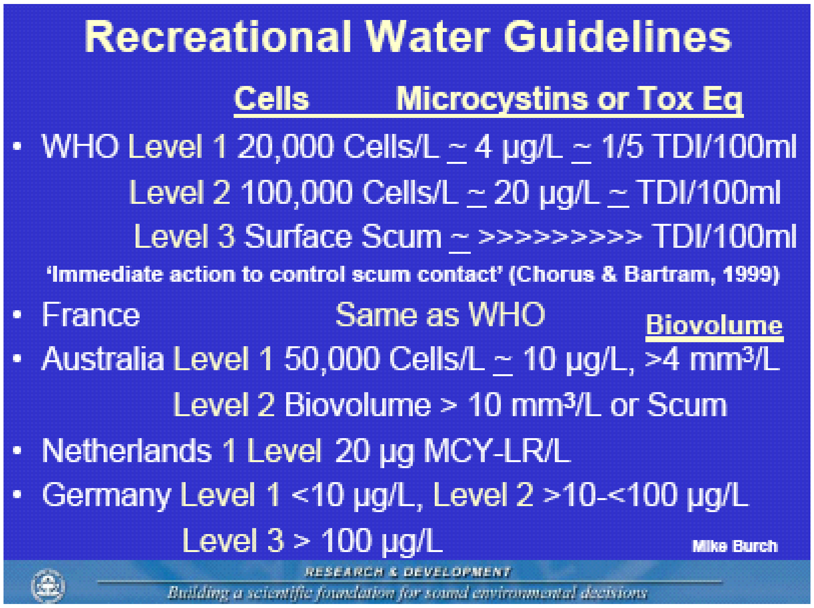

Figure 9. Recreational Water Guidelines for Microcystins.

"Numerous cases of animal poisonings (often lethal) substantiate the concern of health hazards for humans exposed to cyanobacteria."[58] Often, due to lack of public monitoring, officials first become aware of toxic algae problems when they receive reports of dogs, cows, or wildlife dying within minutes to days after exposure to cyanotoxins.[59]

Figure 10. Algal sample from a toxic HAB in Nebraska that killed pets and wildlife during Memorial Day weekend, 2006.[60]

Microcystins have also been linked to a chronic fatigue type illness. Based on an examination of seven people complaining of multiple symptoms following exposures to a

Microcystis bloom in Florida's Lake Griffin, researchers concluded that exposures to *Microcystis* blooms may cause a form of chronic, biotoxin-associated illness with symptoms similar to those experienced by people exposed to water damaged indoor environments.[61]

F. Public health risks associated with cyanotoxins in water bodies used for irrigation.

Other pathways for human exposure to cyanotoxins are created when cyanotoxin contaminated water is used for irrigation. An investigation into the toxicity of plant tissues following cultivation in cyanotoxin contaminated water found that "microcystin was detected in the tissues of exposed plants using a commercially available ELISA kit, suggesting that the uptake of these toxins by edible plants may have significant implications for human health."[62] Other studies have found that "colonies of *M. aeruginosa* and microcystins were retained on salad lettuce following spray irrigation with water that had experienced a cyanobacteria bloom."[63]

G. Environmental risks to fish and wildlife.

Cyanotoxins also take a toll on wildlife. In 1999, almost 400 alligator mortalities in Florida's St. John's Chain of Lakes were linked to levels of *Cylindrospermopsis* cells found in the water. See Figure 11.[64]

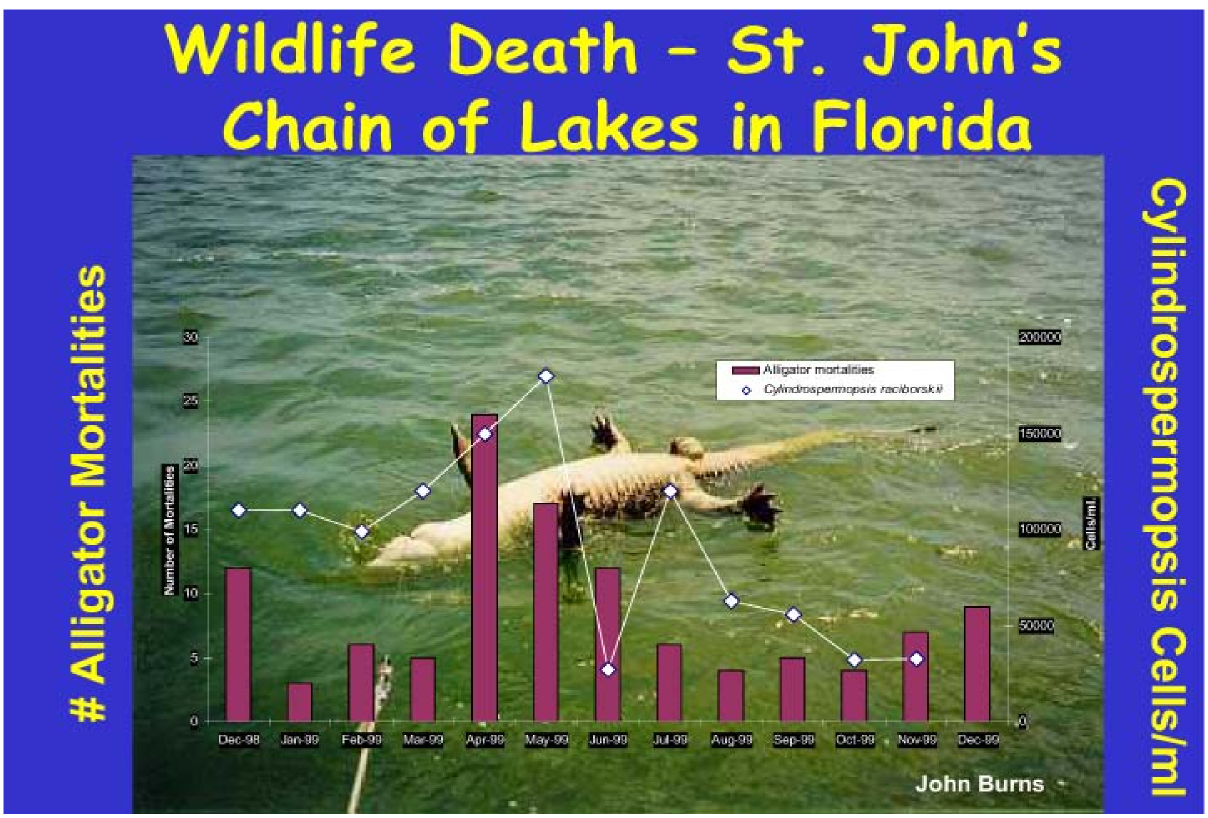

Figure 11. Graph showing relationship between *Cylindrospermopsis* and alligator mortalities.

In Texas, the problem is not a cyanobacteria but "golden algae," a type of algae which produces a toxin that does not appear to affect humans but is lethal to any fish with which it comes in contact. As explained on Texas' Harmful Algal Bloom website:

> Fish kills from the golden alga, *Prymnesium parvum*, have been documented in inland waters in Texas since 1985. While originally noted in the Pecos River in the Rio Grande Basin, the alga has also caused fish kills in four other river basins (Brazos, Canadian, Colorado, and Red River Basins) in Texas. This algal species is found worldwide in estuarine waters (estuaries are mixing zones between freshwater from rivers and seawater) and in some freshwater bodies that have relatively high salt content. Texas biologists were the first to note the occurrence of this alga in freshwater bodies in the Western Hemisphere. Subsequently, other states have reported its occurrence or possible occurrence. Fish kills caused by the alga can be significant, resulting in ecological and economic harm to the affected waterbodies.[65]

Figure 12 shows the occurrence of golden algae in Texas through 2001.[66]

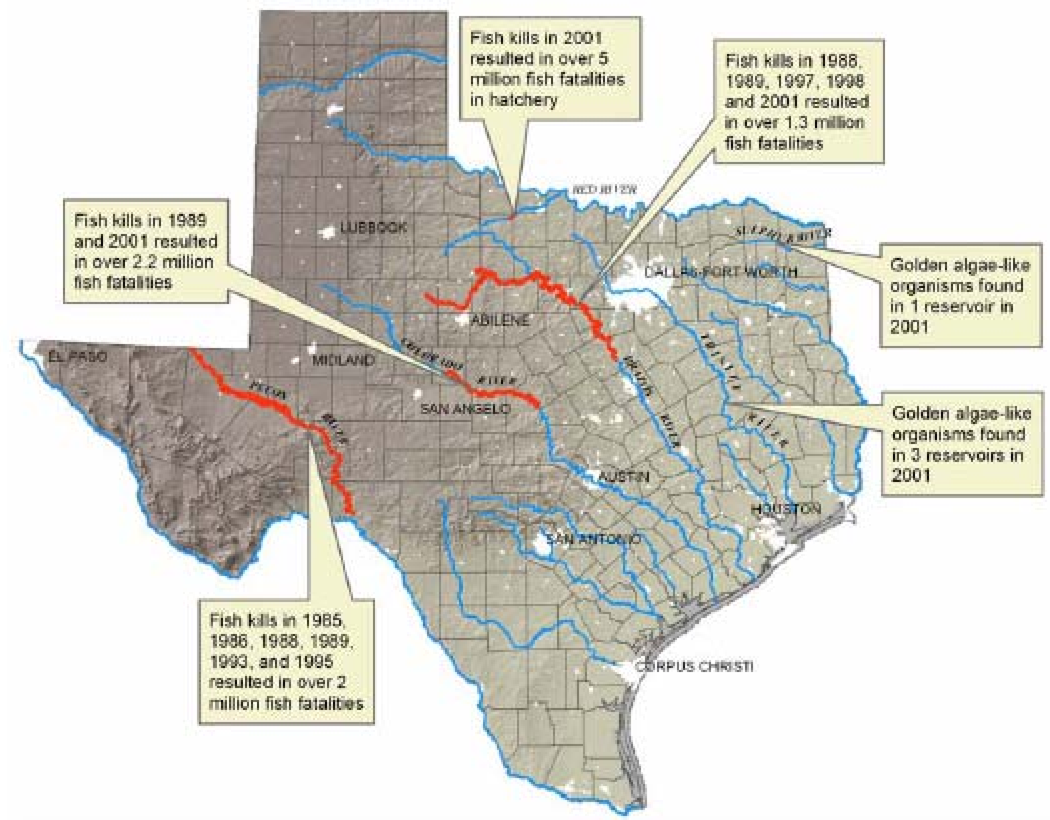

Figure 12. Golden Alga Occurrences Through 2001.

Through the mid-point of 2006 (five years later), the algae has appeared in most major river basins, it has caused more than 65 fish kills in more than 30 different locations, and has now killed more than 25 million fish worth more than $10 million.[67] The algae was discovered in Arizona in 2004, and the first fish kills were reported in 2005.[68] Arizona officials have noted that one of the means by which golden algae can be dispersed is by interbasin transfers of water.[69]

Figure 13. Photograph of golden algae fish kill at Jackson Bend, Lake Granbury, Texas (the white objects are all dead fish).[70]

H. Environmental Risks Associated With Bioaccumulation Of Cyanotoxins

Bioaccumulation is simply the accumulation of a substance, such as a toxic chemical, in the tissues and organs of a living organism in excess of what is normally expected. Biomagnification refers to an enhancement of toxicant concentrations in tissues and organs at each successive level in a food chain. Microcystins have been shown to bioaccumulate in aquatic plants and aquatic animals, including fish used for human consumption.[71] They have also been shown to retard the growth of ecologically important submerged aquatic plants.[72] Human and animal ingestion of toxins that have bioaccumulated in the food chain presents another risk of exposure to potentially toxic levels of cyanotoxins.

For example, research is currently underway to determine the cause of a newly identified bird disease, Avian Vacuolar Myelinopathy (“AVM”), responsible for the death of over a 100 eagles and thousands of American coots and other water birds in the Southern United States. The deaths are caused by an unidentified neurotoxin. Scientists have observed a “strong association” between the occurrence of AVM, hydrilla (an invasive aquatic weed), and a novel and previously unknown cyanobacteria that grows on the hydrilla. During 2001-2004, the cyanobacteria was present on hydrilla “at every site where AVM was diagnosed, but absent or scarcely found in areas were AVM was not observed.” The hypothesis is that the deaths are the result of a cyanotoxin that has bioaccumulated through the food chain as waterfowls ingested the cyanobacteria growing on the hydrilla.[73]

This discovery of a "novel" cyanobacteria found in connection with an invasive species (hydrilla) raises the issue of synergies created when invasive species begin to interact with cyanobacteria. A notable example is the zebra mussel/*Microcystis* bloom connection. Zebra mussels first colonized the Great Lakes around 1986; by 1989, large populations were found in Lake Erie.[74] At that time, the Great Lakes had not experienced cyanobacteria blooms for over a decade. Then, beginning around 1990, huge blooms began to occur; in 1995 a bloom covered the entire western basin of Lake Erie.[75] In August 2003 another massive *Microcystis aeruginosa* bloom formed in Lake Erie which persisted for nearly a month and was described as follows:

> Surface scums of *Microcystis* containing high concentrations of the toxin microcystin washed ashore in Michigan and Ohio, resulting in foul-smelling, rotting, algal mats. Beaches and recreational boating areas were rendered unusable and sport fishing was adversely affected.[76]

While the *Microcystis* bloom of 2003 was perhaps the most severe in Lake Erie's recent history, "it was only the latest in a trend towards increasing frequency of *Microcystis* blooms in the last decade."[77] Researchers have discovered that a synergy between zebra mussels and *Microcystis* has led to these blooms. Zebra mussels "selectively filter and reject phytoplankton so as to promote and maintain *Microcytis* blooms" as follows:

> Mussels filter the water whether or not *Microcystis* is present, but they spit *Microcystis* back into the water, while at the same time they eat other algae. Thus the competitors of *Microcystis* are removed. . . . At the same time this selective feeding process is occurring, the mussels are excreting nutrients (phosphate and ammonia) derived from the phytoplankton they eat as part of digestion and metabolic processes. These nutrients, in turn, serve to fertilize growth of *Microcystis*.[78]

As a result of this synergy between cyanobacteria and an invasive species, the entire ecology of the Great Lakes has undergone a rapid and profound change.[79] A "*Microcystis* Shake" of algae from the Great Lakes is shown in Figure 14.[80]

Figure 14. "Shake" of *Microcystis aeruginosa* from Lake Eire.

I. The toxic algae problem is growing rapidly throughout the United States.

Within just the past three years, cyanotoxins have been found in lakes, streams, and reservoirs at levels warranting public health advisories in Southern California,[81] Northern California,[82] Florida,[83] Oregon,[84] Washington,[85] the Great Lakes,[86] New Hampshire,[87] Michigan,[88] Nebraska,[89] Utah,[90] New York,[91] Minnesota,[92] Wisconsin,[93] Indiana,[94] and Kansas.[95]

In Kansas alone, harmful algal blooms have led to the shutdown of water treatment facilities and the trucking in of drinking water, expenditures of millions of dollars to update water treatment facilities plagued with problems caused by algae, and complete discontinuance of use of a drinking water reservoir after a USGS study determined that blue-green algae was the likely cause of taste and odor problems in a town's drinking water.[96]

In the summer and fall of 2005, the Klamath River in Northern California was the location of a massive toxic algae bloom that was detected by monitoring by the Karuk Tribe.[97] Toxin levels measured at the shore line and in open water exceeded the WHO standard for recreational use by 468 and 89 times, respectively. Microcystin toxin produced by the blooms exceeded the WHO Tolerable Daily Intake level by 217 times and 60.3 times, respectively.[98] These levels were among the highest ever recorded in the United States.[99] On September 30, 2005, a joint warning was issued by the Karuk Tribe, the North Coast Regional Water Board, and the United States Environmental Protection Agency.[100] A map of documented cyanobacteria harmful algal blooms is shown in Figure 15.[101]

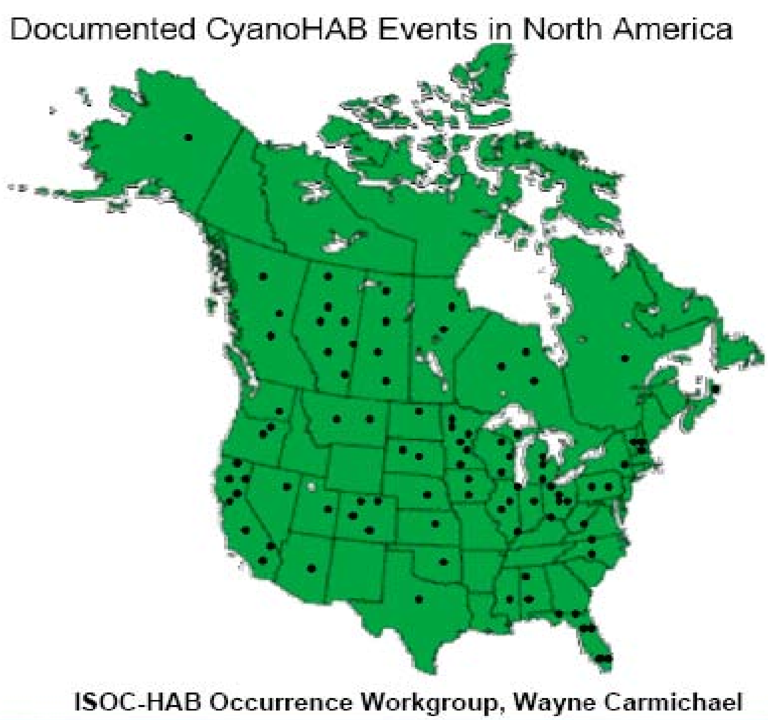

Figure 15. Documented blooms in North America.

[1] App. 18 (USGS and NPS: Interbasin Biota Transfers at p. 6-1)
[2] App. 18 (USGS and NPS: Interbasin Biota Transfers at p. 6-2)
[3] App. 18 (USGS and NPS: Interbasin Biota Transfers at pp. ES-3 – ES-5)
[4] App. 18 (USGS and NPS: Interbasin Biota Transfers at p. ES-5)
[5] App. 19 (USEPA: Hudnell Cyanobacteria PowerPoint)
[6] App. 19 (USEPA: Hudnell Cyanobacteria PowerPoint)
[7] App. 20 (USGS: HAB Briefing Sheet)
[8] App. 21 (Crayton: Toxic Cyanobacteria Blooms Field/Laboratory Guide)
[9] App. 22 (Reynolds: Cyanobacteria: Natural Organisms With Toxic Affects)
[10] App. 21 (Crayton: Toxic Cyanobacteria Blooms Field/Laboratory Guide)
[11] App. 21 (Crayton: Toxic Cyanobacteria Blooms Field/Laboratory Guide)
[12] App. 23 (Nuttle: Photographs of St. Lucie River Algae Bloom)
[13] App. 24 (FL DOH: Aquatic Toxins Program PowerPoint); App. 25 (Burgess: Wave of Momentum for Toxic Algae Study)
[14] App. 24 (FL DOH: Aquatic Toxins Program PowerPoint); App. 22 (Reynolds: Cyanobacteria: Natural Organisms With Toxic Affects at p. 78.)
[15] App. 24 (FL DOH: Aquatic Toxins Program PowerPoint); App. 22 (Reynolds: Cyanobacteria: Natural Organisms With Toxic Affects at p. 78)
[16] App. 26 (Burns: Cyanobacteria and Their Toxins in Florida Surface Waters at pp. 16-21)
[17] App. 26 (Burns: Cyanobacteria and Their Toxins in Florida Surface Waters at pp. 16-21).
[18] App. 27 (ORLANDO SENTINEL: Health Menace Lurks in Lakes); App. 54 (Indiana University: Distribution and Abundance of *Cylindrospermopsis raciborskii* in Indiana Lakes and Reservoirs)
[19] App. 24 (FL DOH: Aquatic Toxins Program PowerPoint) (slide on Cyanotoxin Types: Dermatoxins)
[20] App. 28 (Kugrens: Effect of Algal Inhibitors on Higher Plant Tissues)
[21] App. 28 (Kugrens: Effect of Algal Inhibitors on Higher Plant Tissues)
[22] App. 29 (McElhiney: Inhibitory Effects of Microcystins on Plant Growth and Toxicity of Plant Tissues Following Exposure)
[23] App. 29 (McElhiney: Inhibitory Effects of Microcystins on Plant Growth and Toxicity of Plant Tissues Following Exposure)
[24] App. 19 (USEPA: Hudnell Cyanobacteria PowerPoint)
[25] App. 19 (USEPA: Hudnell Cyanobacteria PowerPoint)
[26] App. 24 (FL DOH: Aquatic Toxins Program PowerPoint)
[27] App. 22 (Reynolds: Cyanobacteria: Natural Organisms With Toxic Affects)
[28] App. 22 (Reynolds: Cyanobacteria: Natural Organisms With Toxic Affects)
[29] App. 30 (Carmichael: Testimony for a Hearing on the Scientific Issues Related to Harmful Algae Blooms and Hypoxia)
[30] App. 22 (Reynolds: Cyanobacteria: Natural Organisms With Toxic Affects)
[31] App. 25 (Burgess: Wave of Momentum for Toxic Algae Study)
[32] App. 25 (Burgess: Wave of Momentum for Toxic Algae Study); App. 24 (FL DOH: Aquatic Toxins Program PowerPoint)
[33] App. 57 (Svrek and Smith: Cyanobacteria Toxins and the Current State of Knowledge on Water Treatment Options: A Review)
[34] App. 58 (WHO: Chemical Fact Sheet for Microcystin LR)
[35] App. 19 (USEPA: Hudnell Cyanobacteria PowerPoint)

[36] App. 24 (FL DOH: Aquatic Toxins Program PowerPoint)
[37] App. 59 (USEPA: Drinking Water Contaminant Candidate List 2); App. 60 (USEPA: Creating a Cyanotoxin Target List for the Unregulated Contaminate Monitoring Rule)
[38] App. 22 (Reynolds: Cyanobacteria: Natural Organisms With Toxic Affects)
[39] App. 24 (FL DOH: Aquatic Toxins Program PowerPoint)
[40] App. 24 (FL DOH: Aquatic Toxins Program PowerPoint)
[41] App. 24 (FL DOH: Aquatic Toxins Program PowerPoint) (slide titled "Drinking Water Exposure Reports").
[42] App. 61 (WHO: TOXIC CYANOBACTERIA IN WATER: A GUIDE TO THEIR PUBLIC HEALTH CONSEQUENCES, MONITORING, AND MANAGEMENT)
[43] App. 61 (WHO: TOXIC CYANOBACTERIA IN WATER: A GUIDE TO THEIR PUBLIC HEALTH CONSEQUENCES, MONITORING, AND MANAGEMENT)
[44] App. 61 (WHO: TOXIC CYANOBACTERIA IN WATER: A GUIDE TO THEIR PUBLIC HEALTH CONSEQUENCES, MONITORING, AND MANAGEMENT)
[45] App. 61 (WHO: TOXIC CYANOBACTERIA IN WATER: A GUIDE TO THEIR PUBLIC HEALTH CONSEQUENCES, MONITORING, AND MANAGEMENT)
[46] App. 60 (USEPA: Creating a Cyanotoxin Target List for the Unregulated Contaminate Monitoring Rule)
[47] App. 62 (Fleming: Blue Green Algal Exposure, Drinking Water and Primary Liver Cancer)
[48] App. 57 (Svrek and Smith: Cyanobacteria Toxins and the Current State of Knowledge on Water Treatment Options: A Review); App. 22 (Reynolds: Cyanobacteria: Natural Organisms With Toxic Affects); App. 25 (Burgess: Wave of Momentum for Toxic Algae Study); App. 24 (FL DOH: Aquatic Toxins Program PowerPoint); App. 61 (WHO: TOXIC CYANOBACTERIA IN WATER: A GUIDE TO THEIR PUBLIC HEALTH CONSEQUENCES, MONITORING, AND MANAGEMENT)
[49] App. 61 (WHO: TOXIC CYANOBACTERIA IN WATER: A GUIDE TO THEIR PUBLIC HEALTH CONSEQUENCES, MONITORING, AND MANAGEMENT)
[50] App. 39 (THE NEWS-REVIEW (OR): High algae levels detected in Lemolo Lake)
[51] App. 61 (WHO: TOXIC CYANOBACTERIA IN WATER: A GUIDE TO THEIR PUBLIC HEALTH CONSEQUENCES, MONITORING, AND MANAGEMENT) (Chapter 4. Human Health Aspects)
[52] App. 61 (WHO: TOXIC CYANOBACTERIA IN WATER: A GUIDE TO THEIR PUBLIC HEALTH CONSEQUENCES, MONITORING, AND MANAGEMENT)
[53] App. 61 (WHO: TOXIC CYANOBACTERIA IN WATER: A GUIDE TO THEIR PUBLIC HEALTH CONSEQUENCES, MONITORING, AND MANAGEMENT)
[54] App. 61 (WHO: TOXIC CYANOBACTERIA IN WATER: A GUIDE TO THEIR PUBLIC HEALTH CONSEQUENCES, MONITORING, AND MANAGEMENT)
[55] App. 48: (NE DEQ: Nebraska Cyanobacteria Experience; App. 19 (USEPA: Hudnell Cyanobacteria PowerPoint) (slides on Pawnee Lake)
[56] App. 48: (NE DEQ: Nebraska Cyanobacteria Experience; App. 19 (USEPA: Hudnell Cyanobacteria PowerPoint) (slides on Pawnee Lake)
[57] App. 19 (USEPA: Hudnell Cyanobacteria PowerPoint) (slide titled Recreational Water Guidelines)
[58] App. 61 (WHO: TOXIC CYANOBACTERIA IN WATER: A GUIDE TO THEIR PUBLIC HEALTH CONSEQUENCES, MONITORING, AND MANAGEMENT)
[59] App. 24 (FL DOH: Aquatic Toxins Program PowerPoint) (slide on Animal Deaths); App. 19 (USEPA: Hudnell Cyanobacteria PowerPoint) (slide titled "Bucaneer Bay, May 4, 2004); App.

20 (USGS: HAB Briefing Sheet); SUNY College of Environmental Science and Forestry, “New York’s Toxic Algae Blooms: ESF Leads New Study (August 13, 2005); App. 42 (PENINSULA DAILY NEWS (WA): Poisonous algae closes state park); App. 50 (UTAH AG NEWS: Blue-Green Algae Bloom Blamed for Cattle Deaths: Uintah County Health Advisory Issued); App. 48 (NE DEQ: Nebraska Cyanobacteria Experience; App. 38 (Hill: Dog Deaths in Humboldt and Mendocino County Water Bodies Possibly Related to Cyanobacterial Toxicity PowerPoint); App. 40 (Oregon DHS Powerpoints: Overview of Oregon Cyanobacteria Experience and Cyanobacteria & Public Health in Oregon)

[60] App. 20 (USGS: HAB Briefing Sheet)

[61] App. 63 (Shoemaker: Characterization of Chronic Human Illness Associated With Exposure to Cyanobacterial Harmful Algal Blooms Predominated by M*icrocystis*)

[62] App. 29 (McElhiney: Inhibitory Effects of Microcystins on Plant Growth and Toxicity of Plant Tissues Following Exposure)

[63] App. 29 (McElhiney: Inhibitory Effects of Microcystins on Plant Growth and Toxicity of Plant Tissues Following Exposure)

[64] App. 19 (USEPA: Hudnell Cyanobacteria PowerPoint) (slide labeled Wildlife Death – St. John’s Chain of Lakes in Florida)

[65] App. 64 (TPW: Golden Algae)

[66] App. 65 (Toxic Golden Algae in Texas)

[67] App. 66 (TPW: Golden Algae in Texas)

[68] App. 67 (AZ HAB Meeting: Responding to Harmful Algal Blooms)

[69] App. 67 (AZ HAB Meeting: Responding to Harmful Algal Blooms)

[70] App. 68 (Texas Parks and Recreation: Images of Golden Algae Fish Kills)

[71] App. 69 (Mitrovic: Bioaccumulation and harmful effects of microcystin-LR in the aquatic plants *Lemna minor* and *Wolffia arrhiza* and the filamentous alga *Chladophora fracta*); App. 70 (Freitas de Magalhaes: Microcystin contamination in fish from the Jacarepagua Lagoon (Rio de Janeiro, Brazil): ecological implications and human health risk); App. 71 (Yin: Microcystin-RR Uptake and Its Effects on the Growth of Submerged Macrophyte *Vallisneria natans* (lour.) Hara)

[72] App. 69 (Mitrovic: Bioaccumulation and harmful effects of microcystin-LR in the aquatic plants *Lemna minor* and *Wolffia arrhiza* and the filamentous alga *Chladophora fracta*); App. 71 (Yin: Microcystin-RR Uptake and Its Effects on the Growth of Submerged Macrophyte *Vallisneria natans* (lour.) Hara)

[73] App. 72 (Williams: Investigation of a novel epiphytic cyanobacterium associated with reservoirs affected by avian vacular myelinopathy)

[74] App. 73 (Fahnenstiel: Phytoplankton Productivity in Saginaw Bay, Lake Huron: Effects of Zebra Mussell (*Dreissena polymorpha*) Colonization)

[75] App. 74 (Lake Erie Center: Water Quality Monitoring)

[76] App. 74 (Lake Erie Center: Water Quality Monitoring)

[77] App. 74 (Lake Erie Center: Water Quality Monitoring)

[78] App. 74 (Lake Erie Center: Water Quality Monitoring)

[79] App. 75 (NOAH: Zebra Mussels Changing Great Lakes Ecosystem)

[80] App. 74 (Lake Erie Center: Water Quality Monitoring)

[81] App. 36 (Lehman: Distribution and Toxicity of a New Colonial Microcystis Aeruginosa Bloom in the San Francisco Bay Estuary)

[82] App. 37 (USEPA: Federal, Tribal and State Authorities Advise Caution on Dangerous Klamath River Algae); App. 38 (Hill: Dog Deaths in Humboldt and Mendocino County Water Bodies Possibly Related to Cyanobacterial Toxicity PowerPoint)
[83] App. 84 (Martin County Health Department (FL): St. Lucie River Blue Green Algae Warning); App. 85 (Martin County Health Department (FL): Warning Sign)
[84] App. 39 (THE NEWS-REVIEW (OR): High algae levels detected in Lemolo Lake); App. 40 (Oregon DHS Powerpoints: Overview of Oregon Cyanobacteria Experience and Cyanobacteria & Public Health in Oregon); App. 41 (MAIL TRIBUNE (OR): Officials warn of toxic algae)
[85] App. 42 (PENINSULA DAILY NEWS (WA): Poisonous algae closes state park); App. 43 (Washington State Parks: Anderson Lake Park Closure)
[86] App. 44 (NOAA: The Zebra Mussel Connection: Nuisance Algal Blooms)
[87] App. 45 (UNION LEADER (NH): Toxic bacteria found in ponds); App. 45 (UNION LEADER (NH): Rains blamed for high water pollution)
[88] App. 47 (MUSKEGON CHRONICLE (MI): Toxic blooms likely a growing problem)
[89] App. 48: (NE DEQ: Nebraska Cyanobacteria Experience); App. 49 (University of Nebraska: Potential for Algae Blooms Unchanged from Last Year); App. 19 (USEPA: Hudnell Cyanobacteria PowerPoint)
[90] App. 50 (UTAH AG NEWS: Blue-Green Algae Bloom Blamed for Cattle Deaths: Uintah County Health Advisory Issued)
[91] App. 51 (SUNY: New York's Toxic Algae Blooms)
[92] App. 52 (Minnesota Environmental Partnership: 3 Minnesota lakes have toxic algae)
[93] App. 53 (WI DNR: Public health advisory issued for Tainter Lake and areas downstream: Lake users can avoid risks from blue-green algae blooms)
[94] App. 54 (Indiana University: Distribution and Abundance of *Cylindrospermopsis raciborskii* in Indiana Lakes and Reservoirs)
[95] App. 55 (KS DHE: KDHE Issues Public Health Advisory for Parts of Eightmile Creek: Advisory also applies to livestock and pets)
[96] App. 56 (USGS: Cyanobacterial (Blue-Green Algal) Blooms: Tastes, Odors, and Toxins)
[97] App. 126 (USEPA: Klamath River System Struggles with Toxic Algae, Water Diversions); App. 127 (Kann: Toxic Cyanobacterial Blooms in the Klamath River System)
[98] App. 37 (USEPA: Federal, Tribal and State Authorities Advise Caution on Dangerous Klamath River Algae)
[99] App. 37 (USEPA: Federal, Tribal and State Authorities Advise Caution on Dangerous Klamath River Algae)
[100] App. 37 (USEPA: Federal, Tribal and State Authorities Advise Caution on Dangerous Klamath River Algae)
[101] App. 19 (USEPA: Hudnell Cyanobacteria PowerPoint)