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## BLUE-GREEN ALGAL TOXINS: Executive Summary

**ON THE SOURCE OF ALZHEIMER’S, ALS-PDC, AND CANCERS IN OKLAHOMA  
SUBMITTED BY THE NATIONAL CFIDS FOUNDATION WITH  
THE NANCY TAYLOR FOUNDATION FOR CHRONIC DISEASES**

*Prepared by* Dana T. Cesar, Ph.D.



Because of the evolutionary function of blue-green algae (BGA) to biodegrade matter, effluents into Oklahoma’s water system encourage algal blooms that produce toxins that target the nervous system, the brain and the liver. Toxins are lethal or debilitating, even in small amounts. Vulnerable populations, like children, the elderly, or those with lowered immune systems are particularly susceptible to diseases caused by algal toxins.

BMAA (beta-methylamino-alanine) is a particular threat to human health as nearly every genera of blue-green algae produce the neurotoxin which researchers believe is the cause of many common diseases such as Alzheimer's, Parkinson's, and ALS, or Lou Gerhig's Disease, as well as many cancers. Although BGA—also known as cyanobacteria—live in fresh, brackish or salt water as well as in soil, the nearly exclusive finding of BGA in freshwater is especially concerning to drinking, bathing, recreational and irrigational water quality in Oklahoma. Because the bacteria can become airborne or aerosolized through irrigation or recreational activities, it can be inhaled.

Oklahoma's abundant shoreline, coupled with the state's industrial farming of chicken, beef and pork, renders the state particularly vulnerable to algal toxins, as illustrated by the September 2009 federal case, *State of Oklahoma v. Tyson Foods, Inc, et al.* At the judicial level, this case points out the correlation between poultry production and cyanobacterial outbreaks in the Illinois River Watershed and Lake Tenkiller. Further, media report that Lake Thunderbird and Grand Lake have been plagued by algal blooms since the mid-1980s. Lake Elmer near Kingfisher experienced a bloom in summer 2009 that left the lake devoid of wildlife.

Statewide, other blooms persist and go unreported as there are no official mechanisms for predicting, preventing, or reporting blooms. The dramatic effects of blooms—such as fish kills or unpalatable water—are

often superficially investigated. The state has no official state water standards where cyanotoxins are concerned. Water quality authorities lack training and support in testing for and filtering toxins. Consequences to human health are unclear as there is no infrastructure for tracking HAB-related illness.

Because the United States has not yet set guidelines for “any of the cyanotoxins,” state and local authorities are left to determine their own standards, as per the Clean Water Act of 1972. Further, the Safe Drinking Water Act amendment of 1996 requires states to protect “drinking water and its sources: rivers, lakes, reservoirs, springs, and ground water wells.”<sup>1</sup>

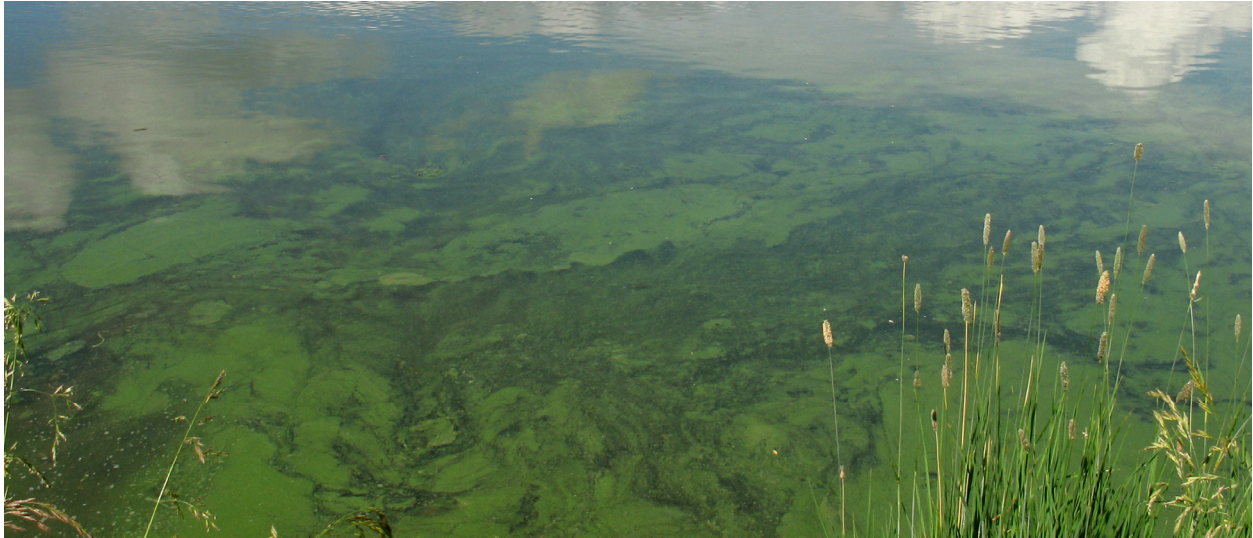
In light of the evolutionary endurance of the bacteria, a current outbreak of harmful algal blooms, and an impending water shortage, this Green Paper calls on authorities to generate focused and coordinated discussion for immediate state action in mobilizing resources at “war-time speed” for the protection of the public against cyanobacterial toxin exposure.

<sup>1</sup> Environmental Protection Agency. Safe Drinking Water Act website. Retrieved 9/14/09 from <http://www.epa.gov/OGWDW/sdwa/basicinformation.html>

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## Introduction

In early September 1985, the *Norman Transcript* released a series of news reports regarding concerns of a University of Oklahoma algologist, Dr. Lois Pfiester, that the foul odors and tastes of drinking and bathing water from Lake Thunderbird may be explained by toxins excreted from a dramatic expansion of algae blooms in the lake. Although city officials assured Norman, Del City and Midwest City residents the water was “100 percent safe,” Pfiester sampled the water and found four types of algae—microcystis, anabeana, aphanizomenon and Lyngbya—with cell counts between 200,000 to 300,000 per millimeter of water. The same news story reported a small cluster of emergency room visits to Norman Regional Hospital for “cases of gastrointestinal disorders, including diarrhea.”<sup>1</sup>

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<sup>1</sup> Linam, Steve Ray. “Lake algae worrisome to officials.” *Norman Transcript*, 9/6/85, p. 1

Pfiester's work with Lake Thunderbird was cited the following day in the *Tulsa World* as it reported "funny" tasting city water that was explained by "a breakdown in equipment at the A.B. Jewel Water Treatment Plant at 21<sup>st</sup> Street and 193<sup>rd</sup> East Avenue" which affected the south, southeast and east sections of Tulsa. However, City-County Health Department official, Tom Drake, attributed the problem to an algal bloom at the "Oologah Reservoir in Rogers and Nowata counties." One Tulsa resident stated that his dog refused the water and the paper reported an increase in the sale of bottled water.<sup>2</sup>

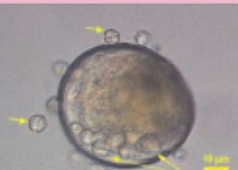
In subsequent news stories that summer, Pfiester was reported to have met with the Norman City Council to inform them of the "correlation between toxin byproducts from blue-green algae and health problems." Children were particularly vulnerable to the health threats caused by the algae.<sup>3</sup> She commented on the death of cattle and fish caused by the toxins and stated, "If it's toxic enough to kill a cow, I think human beings should be concerned."<sup>4</sup>

Water samples were sent to expert Dr. Wayne Carmichael at Wright State University, which came back inconclusive as to toxin content.<sup>5</sup> Regardless, Pfiester later urged officials to restrict nutrient input into the lake, stating that "one observer counted boaters dumping 30 portable toilets into the lake during a one-hour period this spring. Swimmers and leaking septic tanks also add to the problem" as algae feed on the phosphorous and nitrates of such pollutants.

**SPOTLIGHT**

### The Cost of *Pfiesteria*-Related Fish Kills

The economics of HAB outbreaks are extremely hard to quantify accurately. The financial impacts of HABs range from loss of marketable product, costs of maintaining measures necessary to monitor, mitigate, and manage events, and human health impairment, which translates to medical costs and lost wages and earnings (Anderson et al. 2000). During the 1997 outbreak of fish kills in Maryland, which were associated with the dinoflagellate *Pfiesteria piscicida*, it was estimated that the direct cost was at least \$43 million dollars, based solely on the decline in seafood sales (Lipton 1999). When losses to tourism, recreational fisheries, and increased costs of monitoring and analysis are factored in, the economic impacts of this event were staggering. This event further demonstrated that despite the use of educational materials indicating that waters were subsequently safe, consumers were very slow to resume consumption of the impacted product leading to prolonged losses to the seafood community.



*Pfiesteria piscicida* zoospores (~10 µm in diameter) shown swarming around (short arrows), penetrating, and consuming (long arrows) a live eastern oyster larva.

Source: J. Burkholder

Image taken from: Ramsdel, J.S. et al, (eds). *Harmful Algal Research and Response: A National Environmental Science Strategy 2005–2015*, p. 18. Ecological Society of America: Washington, D.C.

Lake Thunderbird . . . a true problem exists, and a lot of people in this community are resistive to facing up to it."<sup>6</sup>

Pfiester, for whom a dinoflagellate was named for her groundbreaking work with algae, was quoted as saying she preferred her roles as "teacher, researcher and mother" over political agitator. However, she wasn't "going to lie about

<sup>2</sup> Pratter, Mark. "Tulsa's Water Tastes Funny, Safe to Drink." *Tulsa World*, 9/7/85, p. 1.

<sup>3</sup> Linam, Steve Ray. "Water tests proposal eyed." *The Norman Transcript*, 9/25/85, p. 2

<sup>4</sup> Wall, Judith. "Botanist links health problems with algae in Norman's water." *OU Update*, section: Profile, not dated, p. 3 & 4

<sup>5</sup> Linam, Steve Ray. "Testing fails to confirm water toxins." *Norman Transcript*, 10/18/85, p. 1

<sup>6</sup> Wall, Judith. "Botanist links health problems with algae in Norman's water." *OU Update*, section: Profile, not dated, p. 3 & 4

Lois Ann Pfiester died in 1992 at the age of 56 with a diagnosis of general autoimmune dysfunction. Her postdoctoral student, Peter Timpano, with whom she closely worked between 1983 and 1986 on an NSF study of algae dinoflagellates, died in 1987 of liver cancer at the age of 38.<sup>7</sup>

It is both appropriate and notable that *Pfiesteria* was specifically mentioned as an immediate threat to human health and the fishing industry in *602 Title VI—Harmful Algal Blooms & Hypoxia Research and Control Act* of 1998, as it is probable that both Pfiester and Timpano died from exposure to the various algae they studied. The “marine algae, dinoflagellates” is said to be “the most important toxin producers” in its devastation of marine waters. However, “in the freshwater environment, toxins have almost exclusively been identified from Cyanobacteria.”<sup>8</sup> The nearly exclusive finding of cyanobacteria in freshwater is highly significant to drinking, bathing, recreational and irrigational water quality in Oklahoma, especially in light of the state’s approximately 11,611 miles of shoreline, “78,578 miles of rivers and streams [and] 1,120 square miles of water area in lakes and ponds.”<sup>9</sup>

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<sup>7</sup> Taylor, Thomas N. (ed). December 1987. *Plant Science Bulletin*. Volume 33 No. 4 Retrieved online at <http://www.botany.org/plantsciencebulletin/psb-1987-33-4.php>

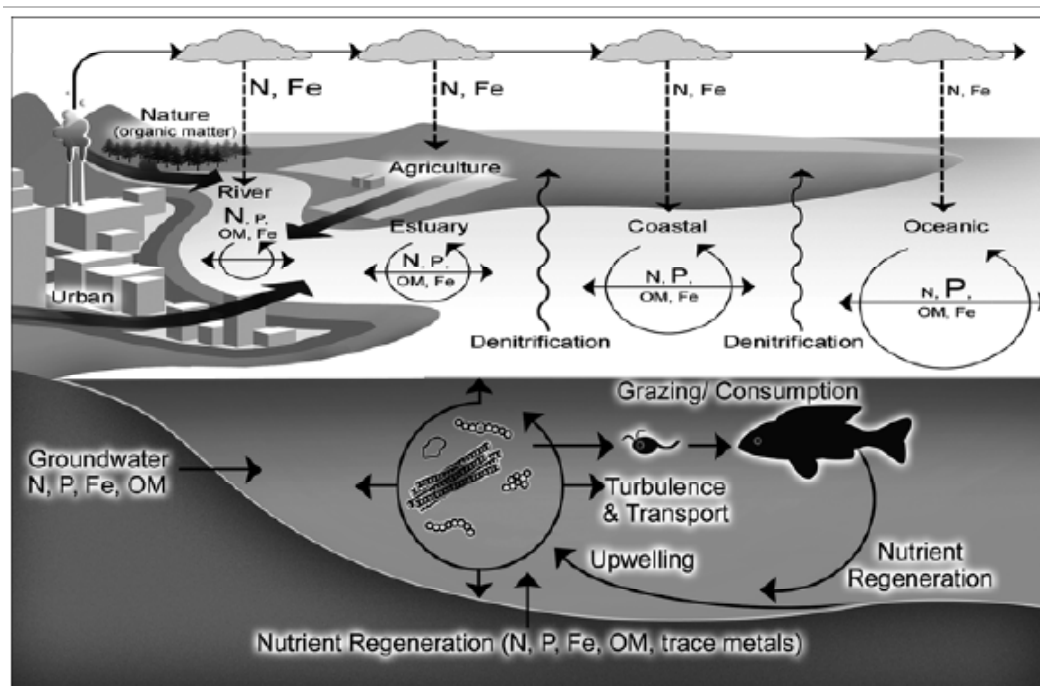
<sup>8</sup> Chorus, I. and Salas, H.J. 1997. “Health Impacts of Freshwater Algae: Draft for Guidelines for Recreational Water and Bathing Beach Quality.” Paper presented at the III Regional AIDIS congress for North America and the Caribbean, San Juan, Puerto Rico, 7 – 12 June. p. 1 – 2.

<sup>9</sup> Oklahoma Water Resources Board. “Oklahoma Water Facts.” Retrieved online at <http://www.owrb.ok.gov/util/waterfact.php>

## What Are Cyanobacteria?

Cyanobacteria, the second oldest life form on earth, have played a pivotal role in the evolutionary history of the planet. The bacteria evolved 2,500 million years ago with the ability to produce oxygen, making life on earth possible.<sup>10</sup> Due to its evolutionary function to degrade dead matter, cyanobacteria are responsible for producing raw crude from prehistoric waste. It has also been found effective at biodegrading industrial agricultural waste, human and animal waste, chemical waste including fertilizers, water run-off from urban centers, and oil spills.<sup>11</sup>

### THE INTERACTIVE PHYSICAL, CHEMICAL AND BIOTIC VARIABLES CONTROLLING CYANOHAB EXPANSION ACROSS THE FRESHWATER-MARINE INTERFACE



Above image taken from: Paerl, H.W. and Fulton III, R.S. 2006. *Ecology of Harmful Algae*. Ecological Studies, Vol. 189, p. 98, Springer-Verlag Bwerlin Heidelberg.

A recent and rapid increase in phosphorous and nitrogen-rich pollutants into the water system are feeding cyanobacteria, enabling them to populate and form mats with the potential to “expand over an area equivalent to a football field within an hour. . . [Australian Environmental Protection

<sup>10</sup> Paerl H.W. & Fulton, R.S. III. 2006. “Ecology of Harmful Cynobacteria” in *Ecological Studies*. Vol 189, chapter 8, p. 95. Graneli Edna & Turner, Jefferson T. (eds).

<sup>11</sup> Raghukumar, C., Vipparty, V., David, J.J., Chandramohan, D. 2001. “Degradation of crude oil by Marine Cyanobacteria.” *Applied Microbiology and Biotechnology*. Vol. 57, pp. 433-436.; Eve Riser-Roberts. 1998. *Remediation of Petroleum Contaminated Soils: Biological, Physical, and Chemical Processes*. Lewis Publishers, Boca Raton, FL.

Agency 2003].”<sup>12</sup> Certain taxa of the bacteria have been “known to cover thousands of square kilometers of the Earth’s oceans” and are “observable by satellite from earth orbit.”<sup>13</sup> Global warming further encourages the duration, scope and density of bacterial “blooms” and the bacteria is spread by boats and ships’ ballast water.

Also known as blue-green algae, cyanobacteria can live in fresh, brackish or salt water as well as in soil. The bacteria can become airborne or aerosolized through irrigation or recreational activities and so can be inhaled. Detailed records of the harmful effects of cyanobacteria date to the 19<sup>th</sup> century, although researchers suggest that even early aboriginal people had knowledge of the poisonous effects of algae to humans, livestock, fish and pets. Paleontologists have reported “cyanotoxin-related mass mortality event[s]” near lakes containing oil shale pits between the Middle Eocene and Pleistocene eras when fossilized remains of cyanobacteria, deer, forest elephant, rhinoceros, ox, lion, horse, turtle, bat and bird skeletons were found to have died in autumn when algal blooms are frequently at their peak.<sup>14</sup>

The commercial value of cyanobacterial compounds has complicated the study of effects on human health and the larger ecosystem. The blue-green pond scum has been used in traditional Chinese medicine for centuries as an anti-fungal and antibacterial and continues to be sold in health food stores as a food supplement.<sup>15</sup> It has recently been genetically modified as a treatment for malaria. Researchers have identified cyanobacteria as an alternative energy source and so are genetically modifying the organism so that more hydrogen compound may be harvested.<sup>16</sup> For example, Dr. Daniel Crunkleton, Director of the Alternative Energy Institute at the University of Tulsa, is working on the development of algae-based transportation fuel with support from San Diego-based Sapphire Energy.<sup>17</sup> Nevertheless, microbiologists, ethnobotanists, organic chemists, veterinarians, neurologists, ecologists, oncologists and others are incrementally closing the gap between anecdotal or strongly correlative links, and empirical evidence of lethal or debilitating effects of the various compounds produced by algae.

<sup>12</sup> Hudnell, K.H., Dortch, Q. and Zenick, H. 2008. “An Overview of the Interagency, International Symposium on Cyanobacterial Harmful Algal Blooms (ISOC-HAB): Advancing the Scientific Understanding of Freshwater Harmful Algal Blooms.” In Hudnell, Kenneth H. (ed.) 2008. *Cyanobacterial Harmful Algal Blooms: State of the Science and Research Needs*. p. 6 New York: Springer.;

<sup>13</sup> Cox et al April 5, 2005 “Diverse taxa of cyanobacteria produce B-N-methylamino-L-alanine, a neurotoxic amino acid.” *Proceedings of the National Academy of Sciences*. Vol. 102, no. 14, pp. 5074–5078.

<sup>14</sup> Stewart, I., Seawright, A.A., Shaw, G.R. 2008. “Cyanobacterial Poisoning in Livestock, Wild Mammals and Birds—An Overview.” Chapter 28, p. 615–618, in Hudnell, Kenneth H., 2008. *Cyanobacterial Harmful Algal Blooms: State of the Science and Research Needs*. Spring: New York.

<sup>15</sup> Patterson, Gregory and Carmeli, Shmuel. 1992. “Biological Effects of Tolitoxin (6-hydroxy-7-)methyl-scytophycin b), a potent bioactive metabolite from cyanobacteria.” *Archives of Microbiology*. Vol. 157, pp. 406 – 410. Springer-Verlag.

<sup>16</sup> Economist. 4/9/2005. “The Scum of the Earth.” Vol. 375, issue 8421, p 68 – 69; See also Amyris Biotechnologies at <http://www.amyrisbiotech.com/>, that used a grant from the Bill & Melinda Gates Foundation in 2004 to develop a cost-effective medicine for malaria using a genetically modified form of cyanobacteria. See the following links for more information about the company. <http://cleantech.com/news/1806/amyris-pulls-in-70m-for-unique-biofuel/>; <http://barrier-busting.com/2008/09/biofuels-balancing-equations/>. See also a talk by John Doerr who briefly refers to “bugs,” which are cyanobacteria. [http://www.ted.com/index.php/talks/john\\_doerr\\_sees\\_salvation\\_and\\_profit\\_in\\_greentech.html](http://www.ted.com/index.php/talks/john_doerr_sees_salvation_and_profit_in_greentech.html); From <http://www.councilforresponsiblegenetics.org/GeneWatch/GeneWatchPage.aspx?pagelid=45>; See also Rosenwald, M.S., “J. Craig Venter’s Next Little Thing,” *Washington Post*, Monday, February 27, 2006; D01.

<sup>17</sup> *Tulsa Engineer: Monthly Bulletin of the Engineer’s Society of Tulsa, Inc.* Biography section, February 23, 2009, Issue # 75.

## Human Health and the International Policy Context

In light of the serious health threats posed by freshwater algae, many in the scientific community have expressed dismay at the lack of fiscal support and an organized, “proactive” governmental response on the part of the United States.<sup>18</sup> However, some researchers maintain that a corner was turned in the policy arena when, in 1998, the EPA included “freshwater cyanobacteria and their toxins on the first Candidate Contaminant List (CCL). . . . for two reasons, namely that (1) they are not necessarily associated with fecal contamination, and therefore will not be adequately controlled by provisions of either the Surface Water Treatment Rule or the Enhanced Surface Water Treatment Rule, and (2) they may not be adequately removed from drinking water by conventional water treatment techniques.”<sup>19</sup> Human health and well being in Oklahoma is further framed within a sluggish international policy context that includes the “first *comprehensive* international conference” on freshwater algae in August 1995 and the development of safe cyanotoxin levels by the World Health Organization (WHO) in 1999.<sup>20</sup>

In light of the evolutionary endurance of the bacteria, a current outbreak of harmful algal blooms, and an impending water shortage,<sup>21</sup> policies must be generated at the state level that are particular to Oklahoma’s waterways and socio-economic contexts. Lester Brown encourages haste in confronting contemporary environmental threats, in that nothing less but mobilizing resources at “war-time speed” can reverse a cycle of environmental decline.<sup>22</sup> Immediately implemented state-level policies are urgently required to proactively protect the state’s water cycle and food web from infection by cyanobacteria.

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Also see <http://www.orgs.utulsa.edu/altenergy/Site/Biofuels.html>. See also Klein, Autumn, Silverman, Phillie, and Wasti, Ali. November 2008. “Solarigene: A Platform for Biofuels and Commodities.” Presentation posted to: [www.hbs.edu/units/tom/conferences/docs/Solarigene.ppt](http://www.hbs.edu/units/tom/conferences/docs/Solarigene.ppt)

<sup>18</sup> As stated by Yoo, et al, “One can only speculate as to why the United States has remained reactive rather than proactive on the issue of cyanobacterial toxins . . . it is unlikely that natural toxins would ever receive the same attention, either by the public or the media, as do man-made contaminants or pollutants. As described in the literature on risk communication there are a number of ‘outrage factors’ that contribute to the public’s perceptions about a particular risk. These include whether the risk of Cyanobacterial toxins are generally less newsworthy than industrial chemicals because they are natural and not as morally relevant or dreaded.” Quoted in Chorus, Ingrid and Sala, Henry J. “Health Impact of Freshwater Algae: Draft for Guidelines for Recreational Water and Bathing Beach Quality.” Paper presented to III Regional AIDIS Congress for North American and the Caribbean, San Juan, Puerto Rick, 7-12 June, 1997.

<sup>19</sup> Hoehn, R.C. and Long, B.W. Not dated. “Toxic cyanobacteria (Blue-green algae): An Emerging Concern.” Paper presentation, not specified. p. 1 & 2,

<sup>20</sup> Chorus, I. and Bartram, J. 1999. *Toxic Cyanobacteria in Water: A guide to their public health consequences, monitoring and management*. St. Edmundsbury Press: London. Retrieved online at [http://www.who.int/water\\_sanitation\\_health/resourcesquality/toxiccyanbact/en/](http://www.who.int/water_sanitation_health/resourcesquality/toxiccyanbact/en/)

<sup>21</sup> Both marine and freshwaters are experiencing an increase in the instances of blooms as per HARNESS. 2005. Harmful Algal Research and Response: A National Environmental Science Strategy 2005 – 2015. Ramsdell, J.S., Anderson, D.M. and Gilbert, P.M. (eds). Ecological Society of America, Washing, DC, 96pp. Retrieved online at <http://www.whoi.edu/redtide/nationalplan/2005nationalplan.html>. See also Lopez, C.B., Jewett, E.B., Dortch, Q., Walton, B.T., Hudnell, H.K. 2008. *Scientific Assessment of Freshwater Harmful Algal Blooms*. pp. 21 & 30. Interagency Working Group on Harmful Algal Blooms, Hypoxia, and Human Health of the Joint Subcommittee on Ocean Science and Technology. Washington, DC. Demand for water in the Oklahoma City metropolitan area is expected to increase 2% each year, according to the City of Oklahoma City 2007 *Consumer Confidence Report*. By 2021, water will be pumped from Sardis Lake and a second pipeline will be installed to transport water from Atoka Lake in order to meet growing demand. See also Smith, Rodney T. June 2009. *Water Strategist: Analysis of Water Marketing, Finance, Legislation and Litigation*. pp. 21 – 22. According to Lester Brown, the nonreplenishable Ogallala water aquifer is dropping 100 meters every year. (See footnote 23 for reference).

<sup>22</sup> “Massive change is inevitable. Will the change come because we move quickly to restructure the economy or because we fail to act and civilization begins to unravel? Saving civilization will take a massive mobilization, and at wartime speed.”

Brown, Lester. December 3, 2008. “Book Bytes: A Wartime Mobilization.” Retrieved online at [http://www.earthpolicy.org/index.php?book\\_bytes/2008/pb3ch13\\_ss1](http://www.earthpolicy.org/index.php?book_bytes/2008/pb3ch13_ss1)

## Infection of the Food Web And Global Epidemiology

In general, cyanobacteria are environmentally adaptive and evolutionarily opportunistic. Because of the algae's unique cellular structure, they are capable of living symbiotically within their host, bioaccumulating within infected species. In this way, the bacteria are capable of co-opting and mutating cell structures and even changing the evolutionary trajectory of host species.<sup>23</sup> Most importantly for human health, cyanotoxins, which are produced within algal cell walls and released after cell death, present grave immunological challenges. So far, scientists have identified a few dozen genera of blue-green algae, all of which produce potent toxins that target the nervous system, the brain and the liver.<sup>24</sup>

Most cyanotoxins cannot be metabolized but are capable of surviving digestion where they are transported or carried unimpaired to block or promote proteins and/or sodium channels important to human health.<sup>25</sup>

International agencies identify two toxins in particular—microcystins and saxitoxins—as necessitating focused attention because their molecular structure and particular composition of amino acids render them easily absorbed into the human system. Because of the “molecular switches” of their toxins, they are considered “the most hazardous algal metabolites.”<sup>26</sup>

<sup>23</sup> Because cyanobacteria possess mitochondria, they are capable of establishing themselves symbiotically within other species. Such symbiosis has allowed cyanobacteria to provide plants with the ability to obtain energy from the sun by giving them chlorophyll, to provide amino acids as “sunscreens” for ultraviolet protection from the sun, and to share with their hosts toxic compounds, like b-methylamino-L-alanine (BMAA), as a defense against predators, as outlined in: Usher, Kayley M., Bergman, Birgitta, Raven, John A. August 2007. *Annual Review of Ecology, Evolution, and Systematics*. 38:255-73.

<sup>24</sup> Lipopolysaccharides, which will not be addressed in this paper, also pose threats to human health. as per Hudnell, Kenneth H., 2008. *Cyanobacterial Harmful Algal Blooms: State of the Science and Research Needs*. See also Chapter 3, Section 1.3 in Chorus, Ingrid and Bartram, Jamie. 1999. *Toxic Cyanobacteria in Water: A Guide to Their Public Health Consequences, Monitoring and Management*. Retrieved online at ([http://www.who.int/water\\_sanitation\\_health/resourcesquality/toxycyanchap1.pdf](http://www.who.int/water_sanitation_health/resourcesquality/toxycyanchap1.pdf)) (see also Chapter 3, Table 3.5 for a list of blue-green algal species.) [http://www.who.int/water\\_sanitation\\_health/resourcesquality/toxycyanchap3.pdf](http://www.who.int/water_sanitation_health/resourcesquality/toxycyanchap3.pdf)

<sup>25</sup> Dietrich, D.R, Fischer, A. Hoeger, S.J. 2008. “Toxin Mixture in Cyanobacterial Blooms—a Critical Comparison of Reality with Current Procedures Employed in Human Health Risk Assessment.” Chapter 39, p. 886 in Hudnell, Kenneth H., 2008. *Cyanobacterial Harmful Algal Blooms: State of the Science and Research Needs*. Springer: New York.; Chorus, Ingrid and Bartram, Jamie. 1999. *Toxic Cyanobacteria in Water: A Guide to Their Public Health Consequences, Monitoring and Management*. Chapter 3, Sections 3.1 & 3.1.1 Retrieved online at [http://www.who.int/water\\_sanitation\\_health/resourcesquality/toxycyanchap3.pdf](http://www.who.int/water_sanitation_health/resourcesquality/toxycyanchap3.pdf)

<sup>26</sup> Chorus, Ingrid and Salas, Henry J. “Health Impacts of Freshwater Algae: Draft for guidelines for Recreational Water and Bathing Beach Quality” paper delivered at the III Regional AIDIS Congress for North America and the Caribbean San Juan, Puerto Rico, 7-12 June 1997; Dietrich, Daniel; Fischer, Michael and Hoeger, S.J. 2008. “Toxin Mixture in Cyanobacterial Blooms.” p. 886, Chapter 39; Falconer, I.R. 2008. “Health Effects Associated with Controlled exposures to Cyanobacterial Toxins.” Chapter 17, p. 607 in Hudnell, Kenneth H. (ed). *Cyanobacterial Harmful Algal Blooms: State of the Science and Research Needs*. Springer: New York.; Chorus, Ingrid and Bartram, Jamie. 1999. *Toxic cyanobacteria in water: A guide to their public health consequences, monitoring and management*. Chapter 3 St. Edmundsbury Press: London. Retrieved online at [http://www.who.int/water\\_sanitation\\_health/resourcesquality/toxycyanchap3.pdf](http://www.who.int/water_sanitation_health/resourcesquality/toxycyanchap3.pdf)

**GENERAL FEATURES OF THE CYANOTOXINS (Note that beta-methylamino-alanine (BMAA) is not included in this table, but can be found in each toxin group.)**

Toxin Group	Primary Target Organ in Mammals	Cyanobacterial Genera <sup>2</sup>
<b>Cyclic Peptides</b>		
Microcystins	Liver	Microcystis, Anabaena, Planktothrix (Oscillatoria), Nostoc, Hapalosiphon, Anabaenopsis
Nodularin	Liver	Nodularia
<b>Alkaloids</b>		
Anatoxin-a	Nerve Synapse	Anabaena, Planktothrix (Oscillatoria), Aphanizomenon
Anatoxin-a(S)	Nerve Synapse	Anabaena
Aplysiatoxins	Skin	Lyngbya, Schizothrix, Planktothrix (Oscillatoria)
Cylindrospermopsins	Liver <sup>3</sup>	Cylindrospermopsis, Aphanizomenon, Umezakia
Lyngbyatoxin-a	Skin, gastro-intestinal tract	Lyngba
Saxitoxins	Nerve axons	Anabaena, Aphanizomenon, Lyngbya, Cylindrospermopsis
Lipopolysaccharides (LPS)	Potential irritant; affects any exposed tissue	All

<sup>1</sup> Many structural variants may be known for each toxin group. <sup>2</sup> Not produced by all species of the particular genus. <sup>3</sup> Whole cells of toxic species elicit widespread tissue damage, including damage to kidney and lymphoid tissue. Table, above, taken from Chorus, Ingrid and Bartram, Jamie. 1999. *Toxic cyanobacteria in water: A Guide to Their Public Health Consequences, Monitoring and Management*. p. 57, Chapter 3, St Edmundsbury Press: London.

**MICROCYSTIN.** Microcystin is a water-soluble cyclic peptide and as such is unable to “penetrate lipid membranes of animal, plant and bacterial cells. Therefore, to elicit their toxic effect, uptake into cells occurs through membrane transporters which otherwise carry essential biochemicals or nutrients.” The metabolic pathway or transporter for microcystin is “the bile acid carrier, which is found in liver cells.” This essentially “restricts the target organ range in mammals largely to the liver.” However, “adverse effects” have also been found in “the small intestine and kidney.” Although the “tumor promoting activity of microcystins is well documented, . . . microcystins alone are not carcinogenic.”<sup>27</sup> Individuals who are genetically predisposed or who have lowered immune defenses,

<sup>27</sup> Chorus, I. and Salas, H.J. 1997. “Health Impacts of Freshwater Algae: Draft for Guidelines for Recreational Water and Bathing Beach Quality.” p. 5, Paper Presented to the III Regional AIDIS congress for North America and the Caribbean, San Juan, Puerto Rico, 7 – 12 June.; Dietrich, D.R, Fischer, A. Hoeger, S.J. 2008. “Toxin Mixture in Cyanobacterial Blooms—a Critical Comparison of Reality with Current Procedures Employed in Human Health Risk Assessment.” Chapter 39, p. 886 in Hudnell, Kenneth H., 2008. *Cyanobacterial Harmful Algal Blooms: State of the Science and Research Needs*. Spring: New York.; Falconer, I.R. 2008. “Health Effects Associated with Controlled Exposure to Cyanobacterial Toxins.” Chapter 27, p. 607, in Hudnell, Kenneth H. (ed.) *Cyanobacterial Harmful Algal Blooms: State of the Science and Research Needs*. Springer: New York.

such as children, the elderly and the sick, are highly susceptible to neurotoxic and hepatotoxic effects of microcystins.

According to Codd, about 65 “structural variations of microcystins are known so far [e.g. microcystin-LR, YR, RR, LA, etc.]”<sup>28</sup>

Because microcystins are “geographically most widely distributed in freshwaters,” and because fresh recreational, drinking water and bathing water comes from surface water sources, countries with infected surface water systems may be important case studies for proactive policy evaluation for Oklahoma. China, for example, is considered a “high-risk area” for hepatocellular carcinoma (liver cancer) and colorectal cancer. It accounts “for some 45% of worldwide mortality” as toxins “are ingested [directly] from drinking surface water.”<sup>29</sup> Clusters of infected populations in China were rendered particularly susceptible to cancers because of exposure to “chronic viral hepatitis” and to a fungal toxin sometimes found in stored grain or commercial peanut butter.<sup>30</sup>

Another case in point is Caruaru, Brazil, in which “117 [dialysis] patients developed cholestatic liver disease and at least 47 deaths were attributed to dialysis with water containing cyanobacterial toxins. Examination of the carbon filter from the dialysis unit demonstrated microcystin-LR, as did the blood and liver tissue of deceased patients.” In the 1970s, Australia reported that 140 children and 10 adults were hospitalized after drinking water from a dam containing the microcystin *Cylindrospermopsis raciborskii*.<sup>31</sup>

Global epidemiological evidence of the lethal or debilitating effects of direct uptake of microcystin by humans and animals are too numerous to mention in this report.

However, it should be noted that in addition to exposure through direct ingestion, microcystins can enter the food chain by infecting plants and animals where toxins are biomagnified and, in turn, consumed by people where they can bioaccumulate. The World Health Organization reports, “The most definitive effect of microcystin” on the food chain occurred when “Atlantic Salmon reared in net pens in coastal waters of British Columbia and Washington State, USA . . . [were infected with Net Pen Liver Disease (NPLD) in which microcystin] produce a progressive degeneration of the liver

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<sup>28</sup> Codd, G.A. 2000. p. 53, “Cyanobacterial Toxins, the Perception of Water Quality, and the Prioritization of Eutrophication Control” *Ecological Engineering* 16:51 – 60.

<sup>29</sup> Hilborn, E.D., Fournie, J.W. 2008. “Human Health Effects Workgroup Report.” Chapter 26, p. 589 in Hudnell, Kenneth H. (ed). *Cyanobacterial Harmful Algal Blooms: State of the Science and Research Needs*. Springer: New York.

<sup>30</sup> Chorus, I. and Bartram, J. 1999. *Toxic Cyanobacteria in Water: A Guide to Their Public Health Consequences, Monitoring and Management*. Chapter 5, Section 5.21 and Box 5.3. Retrieved online at [http://www.who.int/water\\_sanitation\\_health/resourcesquality/toxycyanbact/en/](http://www.who.int/water_sanitation_health/resourcesquality/toxycyanbact/en/)

<sup>31</sup> Chorus, Ingrid and Bartram, Jamie. 1999. *Toxic Cyanobacteria in Water: A Guide to Their Public Health Consequences, Monitoring and Management*. p. 128-131, Chapter 4, section 4.1.1 St. Edmundsbury Press: London. Retrieved online at [http://www.who.int/water\\_sanitation\\_health/resourcesquality/toxycyanchap4.pdf](http://www.who.int/water_sanitation_health/resourcesquality/toxycyanchap4.pdf)

in salmon smolts placed into open-water net pens.” The infection presented multiple opportunities for human exposure to the toxin.<sup>32</sup>

Researchers report that in extreme cases, multiple exposures to biomagnified plants and edible animals “can be interpolated from assumed daily or weekly consumption of specific food sources e.g. fish, crayfish, shellfish, vegetables, salads, etc. for the general populace as well as for populations at high risk, e.g. indigenous tribes predominantly existing on a specific food source,” children, and perhaps athletes, who drink a higher percentage of water relative to their body weight, the elderly, or individuals with compromised immune systems.<sup>33</sup>

**TOXIN MIXTURE IN CYANOBACTERIAL BLOOMS (Calculated possible daily ingestion to avoid acute health problems according to the calculations of Fromme et al. (2000). For details, see Dietrich et al (2005)**

Ingestion Route	MC Concentrations	Infants 5 kg = 12.5 µg	Children 20 kg = 50 µg	Adults 60 kg = 150 µg
<b>Food</b>	100 µg kg	125 g	500g	1,500 g
	10,000 µg kg	1.25 g	5g	15 g
<b>Cyanobacterial bloom in lake/river</b>	100 µg l	125 ml	500 ml	1,500 ml
	1,000 µg l	1.25 ml	5 ml	150 ml
<b>Drinking Water</b>	1.0 µg l	12,500 ml	50,000 ml	150,000 ml
	100 µg l	125 ml	500 ml	1,500 ml
<b>BGAS</b>	1.0 µg g	12.5 g	500 g	150 g
	10 µg g	1.25 g	5 g	15 g

Table, above, taken from Hudnell, Kenneth H. (ed). *Cyanobacterial Harmful Algal Blooms: State of the Science and Research Needs*. Chapter 39 by Dietrich, Fischer and Hoeger. p. 903.

**SAXITOXIN.** Where cyclic peptides (microcystins) block protein-phosphates, toxic alkaloids—like saxitoxins—“modulate” protein-to-protein interactions and block nerve cell sodium channels essential to human health.<sup>34</sup> This makes them powerful nerve toxins that can also cause brain cancer. Like microcystins, saxitoxins can be directly ingested or inhaled through drinking, bathing and recreating in water and can also spill into the food web where they are absorbed and biomagnified, increasing the toxicity of edible plants and animals.<sup>35</sup>

<sup>32</sup> Ibid, p. 48, Chapter 3 Retrieved online at ([http://www.who.int/water\\_sanitation\\_health/resourcesquality/toxycyanchap1.pdf](http://www.who.int/water_sanitation_health/resourcesquality/toxycyanchap1.pdf))  
[http://www.who.int/water\\_sanitation\\_health/resourcesquality/toxycyanchap3.pdf](http://www.who.int/water_sanitation_health/resourcesquality/toxycyanchap3.pdf)

<sup>33</sup> Dietrich, D.R., Fisher, M.A. and Hoeger, S.J. 2005. “Toxin Mixture in Cyanobacterial Blooms—A Critical Comparison of Reality with Current Procedures Employed in Human Health Risk Assessment.” Chapter 39, p. 893 in Hudnell, Kenneth H., 2008. *Cyanobacterial Harmful Algal Blooms: State of the Science and Research Needs*. Spring: New York; See also Ernst, B., Dietz, L., Hoeger, S.J. and Dietrich, D.R. 2005. “Recovery of MC-LR in Fish Liver Tissue.” *Environmental Toxicology* 20: 449-458.

<sup>34</sup> Grindber, R.V. Shuman, C.F., Sorrels, C.M. Wingerd, J. and Gerwick, W.H. 2008. “Neurotoxic Alkaloids from Cyanobacteria.” Chapter 6, p. 163 in Fattorusso, Ernesto and Tagliatalata-Scafati, Orazio . (eds) *Modern Alkaloids: Structure, Isolation, Synthesis and Biology*. Wiley VCH Verlag: Germany.

<sup>35</sup> Chorus, I. and Bartram, J. 1999. *Toxic Cyanobacteria in Water: A Guide to Their Public Health Consequences, Monitoring and Management*. Chapter 3, Section 3.1.2 & 3.1.5 Retrieved online at [http://www.who.int/water\\_sanitation\\_health/resourcesquality/toxycyanchap3.pdf](http://www.who.int/water_sanitation_health/resourcesquality/toxycyanchap3.pdf)

Biomagnified saxitoxins were originally observed when they entered the food chain through shellfish, causing “what has come to be known as paralytic shellfish poisoning (PSPs)”<sup>36</sup> However, most bottom-dwelling animals become infected when toxins, released by decaying algae or the fecal matter of infected fish, slowly sink to the lake floor where shellfish, snails, clams and mussels absorb them.<sup>37</sup> The World Health Organization reports that freshwater saxitoxins bioaccumulated “in an Australian species of freshwater mussel to concentrations exceeding international guidelines . . . during as little as seven days exposure to a cell density of 100,000 cells per ml of a toxigenic strain,” which is a “cell density . . . commonly encountered in natural blooms of this species.”<sup>38</sup>

Although WHO specifically refers to Australian species of mussels, there is no doubt that zebra mussels, on Oklahoma’s list of invasive species, also absorb toxins beyond recommended international guidelines. While zebra mussels are not a common food source in Oklahoma, mussels are consumed by wild duck and other game that are, in turn, consumed by humans. In this way, saxitoxin poses multiple risks for human exposure.

Additionally, bioaccumulation of saxitoxin by zebra mussels not only infect food sources, but may also store toxins, releasing them back into the water, thereby prolonging toxic effects on the water system.<sup>39</sup> Studies done “in the late 90s” on the mussel as an invasive species of the Great Lakes “led to the conclusion that zebra mussel selectively filtrate *Microcystis aeruginosa*, promoting toxic *Microcystis* blooms.”<sup>40</sup> Further, studies show that algae rations affect growth and propagation of zebra mussels more than other variables.<sup>41</sup> Consequently, the symbiotic partnership for mutual growth of mussels and algae creates further potential for ever-increased exposure due to the ability of toxins to bioaccumulate up the food chain. Because even brief human exposure “to toxins may

<sup>36</sup> Ibid. Retrieved online at [http://www.who.int/water\\_sanitation\\_health/resourcesquality/toxycyanchap3.pdf](http://www.who.int/water_sanitation_health/resourcesquality/toxycyanchap3.pdf)

<sup>37</sup> Ibelings, B.W. and Havens, K.E. “Cyanobacterial Toxins: A Qualitative Meta-analysis of Concentrations, dosage and effects inn Freshwater, Estuarine and Marine Biota.” Chapter 31, p. 680 – 686, in Hudnell, Kenneth H., 2008. *Cyanobacterial Harmful Algal Blooms: State of the Science and Research Needs*. Springer: New York.

<sup>38</sup> Chorus, Ingrid and Bartram, Jamie. 1999. *Toxic Cyanobacteria in Water: A Guide to Their Public Health Consequences, Monitoring and Management*. Chapter 3, Section 3.4.5 Retrieved online at [http://www.who.int/water\\_sanitation\\_health/resourcesquality/toxycyanchap3.pdf](http://www.who.int/water_sanitation_health/resourcesquality/toxycyanchap3.pdf)

<sup>39</sup> According to researchers, freshwater mussels “accumulate both microcystins (Prepas *et al.*, 1997) and saxitoxins (Negri and Jones, 1995),” as per Chorus, Ingrid and Bartram, Jamie. 1999. *Toxic Cyanobacteria in Water: A Guide to Their Public Health Consequences, Monitoring and Management*. Chapter 4, Section 4.1.4 Retrieved online at [http://www.who.int/water\\_sanitation\\_health/resourcesquality/toxycyanchap4.pdf](http://www.who.int/water_sanitation_health/resourcesquality/toxycyanchap4.pdf); See also Foster, A., Boxrucker, J, Gilliland, G. and Bentroth, B. July 11, 2008. “Oklahoma Aquatic Nuisance Species Management Plan.” Oklahoma Department of Wildlife Conservation. Retrieved online at <http://www.anstaskforce.gov/State%20Plans/OK/OKLAHOMA%20ANS%20PLAN%20JULY08.pdf>; <sup>39</sup> Dietrich, D.R, Fischer, A. Hoeger, S.J. 2008. “Toxin Mixture in Cyanobacterial Blooms—a Critical Comparison of Reality with Current Procedures Employed in Human Health Risk Assessment.” Chapter 39, p. 886 in Hudnell, Kenneth H., 2008. *Cyanobacterial Harmful Algal Blooms: State of the Science and Research Needs*. Springer: New York

<sup>40</sup> Vanderploeg, H.A., *et al.* 2001. “Zebra Mussel (*Dreissena Polymorpha*) Selective Filtration Promoted Toxic Microcystis Blooms in Saginaw Bay (Lake Huron) and Lake Erie.” *Canadian Journal of Fish Aquatic Science* 58 (6598) 1208 – 21 as quoted in Fristachi, A. and Clair, J.L. Chapter 3, p. 51 in Hudnell, Kenneth H. 2008. *Cyanobacterial Harmful Algal Blooms: State of the Science and Research Needs*. Springer: New York.

<sup>41</sup> Cooper, B., Ziara, S and Schooley, J.K. 2005. “The Effects of Temperature, Origin, and Ration Size on Shell Length Growth of the Zebra Mussel (*Dreissena Polymorpha*). Paper presented at the 94<sup>th</sup> Annual Technical meeting of the Oklahoma Academy of Sciences, November 5. Abstract may be retrieved from [http://digital.library.okstate.edu/oas/oas\\_pdf/v85/p93\\_96.pdf](http://digital.library.okstate.edu/oas/oas_pdf/v85/p93_96.pdf). For more on bioaccumulation, see also section 3.4.5 of Chorus, I and Bartram, J. 1999. *Toxic Cyanobacteria in Water: A Guide to Their Public Helath Consequences, Monitoring and Management*. St. Edmundsbury Press: London.

result in long-term injury, and chronic low-level exposure may cause adverse health effects,” such symbiosis for propagation between blue-green algae and zebra mussels is particularly concerning.<sup>42</sup>

According to the World Health Organization, “Sixteen confirmed saxitoxins from cyanobacterial samples have been reported,” so far.<sup>43</sup>

**BMAA.** Space does not allow for a complete summary of findings regarding the nearly 80 algal species and their respective toxins in relation to the state’s water supply. For the sake of Oklahoma’s smaller water treatment systems, it is necessary to identify and target those most widespread and those most dangerous toxins for prevention and monitoring. New findings indicate that nearly all genera of cyanobacteria produce the neurotoxin beta-methylamino-alanine (BMAA), a non-protein amino acid that research suggests is the cause of many common diseases, including Alzheimer’s, Parkinson’s, and ALS or Lou Gehrig’s Disease.<sup>44</sup>

According to researchers, BMAA “acts as an agonist of animal glutamate receptors . . . and is chemically related to excitant amino acids. Its uptake into [the] brain is most likely mediated by the large neutral amino acid carrier of the blood-brain barrier.”<sup>45</sup>

Although more common, BMAA is also more difficult to detect than both microcystins and saxitoxins. Researchers report that “rapid screens are available for microcystins [and] saxitoxins . . . [However,] there is an urgent need for rapid, simple and inexpensive assays for . . . BMAA. Although methods exist for analysis of BMAA, the fact that a recent study showed 95% of cyanobacteria producing this, some at levels >6000 µg g-1 dry wt, is of concern and rapid screening followed by robust analysis is needed.”<sup>46</sup>

Until more thorough detection methods can be developed, along with an understanding of the precise mechanisms of disease, epidemiological data may best illustrate the bioaccumulative impact of BMAA to human health.

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<sup>42</sup> Chorus, I and Bartram, J. 1999. *Toxic Cyanobacteria in Water: A Guide to Their Public Health Consequences, Monitoring and Management*. Chapter 4. Section 4.1.2 St. Edmundsbury Press: London. Retrieved online at [http://www.who.int/water\\_sanitation\\_health/resourcesquality/toxycyanchap4.pdf](http://www.who.int/water_sanitation_health/resourcesquality/toxycyanchap4.pdf)

<sup>43</sup> Chorus, Ingrid and Bartram, Jamie. 1999. *Toxic cyanobacteria in water: A guide to their public health consequences, monitoring and management*. p. 65, chapter 3, New York: Routledge. Retrieved online at [http://www.who.int/water\\_sanitation\\_health/resourcesquality/toxycyanobacteria.pdf](http://www.who.int/water_sanitation_health/resourcesquality/toxycyanobacteria.pdf)

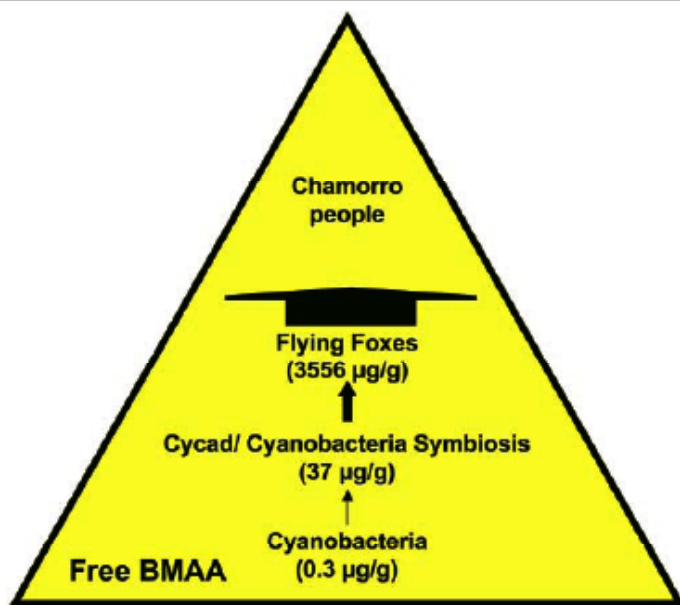
<sup>44</sup> Hudnell, p. 6, p. 68, p. 900 Cyano HABs); Cox, Paul Alan et al. April 5, 2005 Diverse Taxa of Cyanobacteria Produce B-N-methylamino-L-alanine, a Neurotoxic Amino Acid.” *Proceedings of the National academy of Sciences*. vol. 102 no. 14

<sup>45</sup> Dietrich, D.R, Fischer, A. Hoeger, S.J. 2008. “Toxin Mixture in Cyanobacterial Blooms—a Critical Comparison of Reality with Current Procedures Employed in Human Health Risk Assessment.” Chapter 39, p. 893 in Hudnell, Kenneth H., 2008. *Cyanobacterial Harmful Algal Blooms: State of the Science and Research Needs*. Spring: New York.

<sup>46</sup> Lawton, L.A. and Edwards, C. 2008. “Conventional Laboratory Methods for Cyanotoxins.” Chapter 23, p. 524, in Hudnell, Kenneth H., 2008. *Cyanobacterial Harmful Algal Blooms: State of the Science and Research Needs*. Spring: New York.

The presence of algal BMAA in plants was discovered by researchers Vega and Bell in 1967. Since that time, the hypothesis of BMAA as a cause of neurodegenerative disorders grew out of the discovery of high levels of the substance in the brains of the Chamorro people of Guam among whom rates of Alzheimer's and ALS-PDC were, statistically, extraordinarily high. Absent a genetic pattern, researchers were drawn to investigate the diet patterns of the Chamorro. BMAA was not only found in water sources, but also in certain plants, seeds and meat consumed as part of the cultural diet of the Chamorro. Further, the meat source—flying fox—fed almost exclusively on the same infected seeds, resulting in increased bioaccumulation of BMAA in fox as a food source.

#### BIOMAGNIFICATION OF CYANOBACTERIAL BMAA IN GUAM



The widths of the arrows are proportional to the concentration of the free BMAA delivered to the next higher trophic level. Above image taken from Cox, Paul Alan, et al. "Biomagnification of cyanobacterial neurotoxins and neurodegenerative disease among the Chamorro people of Guam." *Proceedings of the National Academy of Sciences*. November 2003 vol. 100 no. 23

and metabolism. Within brain tissues, the endogenous neurotoxic reservoir can slowly release free BMAA, thereby causing incipient and recurrent neurological damage over years or even decades, which may explain the observed long latency period for neurological disease onset among the Chamorro.<sup>48</sup>

Researchers propose that chronic, low dose exposure to the amino acid from water and seeds, as well as higher doses through flying fox meat, caused bioaccumulations and slow release of toxins in the tribe, resulting in gradual neurodegeneration over a period of years.<sup>47</sup> To the point, bound forms of BMAA were found in concentrations "10- to 240-fold." Murch explains that such bound forms of BMAA within the human system may act as "an endogenous neurotoxic reservoir, accumulating and being transported between trophic levels and subsequently being released during digestion

<sup>47</sup> Cox, et al. November 2003. "Biomagnification of Cyanobacterial Neurotoxins and Neurodegenerative Disease Among the Chamorro People of Guam." *Proceedings of the National Academy of Sciences*. vol. 100 no. 23 p. 13380 – 13383; Hudnell, K.H., Dortch, Q. and Zenick, H. 2008. "An Overview of the Interagency, International Symposium on Cyanobacterial Harmful Algal Blooms (ISOC-HAB): Advancing the Scientific Understanding of Freshwater Harmful Algal Blooms." In Hudnell, Kenneth H. (ed.) 2008. *Cyanobacterial Harmful Algal Blooms: State of the Science and Research Needs*. p. 6 New York: Springer.

<sup>48</sup> Susan J. Murch, et al. August 17, 2004 "A Mechanism for Slow Release of Biomagnified Cyanobacterial Neurotoxins and Neurodegenerative Disease in Guam." *Proceedings of the National Academy of Sciences*. vol. 101 no. 33 pp 12228–12231.; See also Grindber, R.V. Shuman, C.F., Sorrels, C.M.

In a separate case, Cox, et al, also found BMAA in the brain tissue of nine Canadians who died of complications from Alzheimer's. The researchers are subsequently calling for further statistically valid studies on the role of BMAA in Alzheimer's and ALS-PDC.<sup>49</sup>

Despite budget cuts to private and governmental agencies, as well as to Tier I research institutions, WHO is additionally calling for more "long-term exposure studies (of at least one year or longer) . . . to assess the chronic toxicity" and uptake routes of cyanobacterial agents. "Further systematic studies are also required into the suggested tumor-promoting effects of some cyanotoxins, particularly in the dose range of potential oral uptake with drinking or bathing water."<sup>50</sup>

### Water Guidelines for the Prevention of Disease

In a 2001 study by Carmichael, 677 water samples volunteered by water utilities throughout the U.S. and Canada returned 80% positive for microcystin. Although only 4.3% of the samples exceeded WHO guidelines, the study highlighted the increasing commonality of toxins in a diversity of water supplies.<sup>51</sup> Due to the global spread of fresh and marine water cyanobacteria, nations are increasingly setting guidelines for harmful algal blooms.

Some argue that guidelines should be set relative to weight and general health of individuals.<sup>52</sup> For example, Chorus and Salas (1997) indicate, "With scum enriching cyanobacterial density by a factor of 1000, . . . **200 ml could kill a child of 20 kg body weight.**"<sup>53</sup> Further, safety levels and TDI's (Tolerable Daily Intakes) cannot account for the bioaccumulative and slow release properties of the toxin.

Nevertheless, risk/benefit analysis (both "qualitative and quantitative") must result in guidelines that can be implemented by local authorities. Among the developed nations, Germany and New Zealand have a microcystin guideline of 1.0 µg/L. "Canada's total microcystin guideline is 1.5 µg/L, while Australia's is 1.3 µg/L in terms of M-LR toxicity equivalents. . . . Australia has suggested 3 µg/L as

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Wingerd, J. and Gerwick, W.H. 2008. "Neurotoxic Alkaloids from Cyanobacteria." Chapter 6, p. 148 in Fattorusso, Ernesto and Tagliatalata-Scafati, Orazio . (eds) *Modern Alkaloids: Structure, Isolation, Synthesis and Biology*. Wiley VCH Verlag: Germany.

<sup>49</sup> Cox, P.A. et al. 2005. "Diverse Taxa of Cyanobacteria Produce B-N-methylamino-L-alanine, a Neurotoxic Amino Acid." *Proceedings of the National Academy of Sciences*. Vol. 102. No. 14, pp 5074 – 5078.

<sup>50</sup> Chorus, I. and Bartram, J. 1999. *Toxic Cyanobacteria in Water: A guide to their public health consequences, monitoring and management*. St. Edmundsbury Press: London. Also retrieved online at [http://www.who.int/water\\_sanitation\\_health/resourcesquality/toxiccyanact/en/](http://www.who.int/water_sanitation_health/resourcesquality/toxiccyanact/en/)

<sup>51</sup> Carmichael, W.W., 2001. *Assessment of Blue-Green Algal Toxins in Raw and Finished Drinking Water*. AWWA Research Foundation, Denver, CO. 179 pages.

<sup>52</sup> Dietrich, D.R, Fischer, A. Hoeger, S.J. 2008. "Toxin Mixture in Cyanobacterial Blooms—a Critical Comparison of Reality with Current Procedures Employed in Human Health Risk Assessment." Chapter 39, p. 891 in Hudnell, Kenneth H., 2008. *Cyanobacterial Harmful Algal Blooms: State of the Science and Research Needs*. Spring: New York

<sup>53</sup> Chorus, I. and Salas, H.J. 1997. "Health Impacts of Freshwater Algae: Draft for Guidelines for Recreational Water and Bathing beach Quality." p. 9 Paper presented to the III Regional AIDIS congress for North America and the Caribbean, San Juan, Puerto Rico, 7 – 12 June.

potential guidelines for both anatoxin-a and the saxitoxins and is considering a cylindrospermopsin guideline in the range of 1-15 µg/L.” The table below provides guidance for water quality authorities where analysis of microcystins are concerned.

**ANALYTICAL ACHIEVABILITY FOR CYANOBACTERIAL TOXINS FOR WHICH  
GUIDELINE VALUES HAVE BEEN ESTABLISHED**

	PPA	ELISA	GC/MS	HPLC/UVPAD	LC/MS
Microcystin-LR	+	+	+	++	++

World Health Organization. 2004. *Guidelines for Drinking-Water Quality: Recommendations. Third Edition, Vol 1. p. 164. Geneva: Marketing and Dissemination, World Health Organization. Retrieved from [http://www.who.int/water\\_sanitation\\_health/dwq/GDW8rev1and2.pdf](http://www.who.int/water_sanitation_health/dwq/GDW8rev1and2.pdf)*

Because the United States has not yet set guidelines for “any of the cyanotoxins,” state and local authorities are left to determine their own standards, as per the Clean Water Act of 1972.<sup>54</sup> By way of example, Vermont established guidelines after media reports of the death of two dogs from microcystin in Lake Champlain. A “tiered system was established for surveillance of cyanobacteria” in the lake with beaches remaining open if “the concentration of microcystin-LR is 6 µg/L or less,” a level based on “recreational childhood swimming.” In Oregon, beaches are to be closed when “toxigenic cell densities exceeded 20,000 cells/mL, corresponding to an Alert Level II according the World Health Organization recommendations.”<sup>55</sup>

Oklahoma requires a minimum of 20 samples to be taken from lakes that are measured for chlorophyll-a (by calculating “TSI = 9.81 x Ln9chlorophyll-a + 30.6.”) and “other routine water quality constituents,” which are not specified. The state has further determined that “A wadable stream shall be deemed threatened by nutrients if the arithmetic mean of benthic chlorophyll-a data exceeds 100 mg per square meter under seasonal base flow conditions, or if two or more benthic chlorophyll-a measurements exceed 200 mg per square meter under seasonal base flow conditions. A non-wadable stream shall be deemed threatened by nutrients if planktonic chlorophyll-a values in the water column indicate it has a Trophic State Index of 62 or greater.”<sup>56</sup>

Further, in March of 2002 the Oklahoma EPA established a phosphorous criterion of 0.037 mg/L that was approved by the U.S. EPA Region VI in May of 2004. However, this level appears to apply as a standard or baseline only to Oklahoma’s six Scenic Rivers (as per the 1970 Scenic Rivers Act, 82

<sup>54</sup> Hoehn, R.C. and Long, B.W. Not dated. “Toxic cyanobacteria (Blue-green algae): An Emerging Concern.” p. 10, Paper presentation, not specified. Retrieved from Ed Taylor archives.

<sup>55</sup> Stone, D. and Bress, W. 2007. “Addressing Public Health Risks for Cyanobacteria in Recreational Freshwaters: The Oregon and Vermont Framework.” *Integrated Environmental Assessment and Management*. Vol. 3, no. 1, pp. 137 – 143.

<sup>56</sup> Unofficial TITLE 785. OKLAHOMA WATER RESOURCES BOARD CHAPTER OKLAHOMA'S WATER QUALITY STANDARDS Chapter 45. BENEFICIAL USES AND CRITERIA TO PROTECT USES p. 13. Retrieved online at [http://www.owrb.ok.gov/util/rules/pdf\\_rul/Chap45.pdf](http://www.owrb.ok.gov/util/rules/pdf_rul/Chap45.pdf); Chapter 46, IMPLEMENTATION OF OKLAHOMA'S WATER QUALITY STANDARDS. p. 26 retrieved online at [http://www.owrb.ok.gov/util/rules/pdf\\_rul/2008\\_adopied/Chap46\\_2008.pdf](http://www.owrb.ok.gov/util/rules/pdf_rul/2008_adopied/Chap46_2008.pdf)

O.S. 145101471), and to Grand Lake (as per Senate Bill 408). Some water sources, such as public and private drinking water sources, the Tenkiller Ferry Reservoir, Wister Lake and “lakes designated as SWS in Appendix A of the Oklahoma Water Quality Standards,” have a criterion of 10 µg/L for chlorophyll-a.<sup>57</sup>

Local water quality authorities and small municipalities should be queried as to the extent to which these guidelines can be implemented to their specific contexts. Part of Oklahoma’s approach to protecting human health should include more stringent, updated and standardized criterion for water quality and support in the way of training and testing materials. Water quality authorities for all 86 systems should be enabled by the state to test for BMAA in both raw and treated drinking water sources.

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<sup>57</sup> Office of the Oklahoma Secretary of the Environment. 2008. “Coordinated Watershed Restoration and Protection Strategy for Oklahoma’s Impaired Scenic Rivers: Update per Senate Bill 972.” P 2 – 3, & 19. Retrieved online at: [http://www.environment.ok.gov/documents/Final%20SB972report\\_2008update.pdf](http://www.environment.ok.gov/documents/Final%20SB972report_2008update.pdf)

## Oklahoma's Approach to Protecting Human Health

The Safe Drinking Water Act amendment of 1996 requires states to protect “drinking water and its sources: rivers, lakes, reservoirs, springs, and ground water wells.”<sup>58</sup> Oklahoma has taken certain proactive steps to recognize, investigate and minimize a growing HAB problem.

For example, the Oklahoma Water Resources Board (OWRB) has developed a number of assessments, as follows:

- **Water Plan Research Studies (Water Resources Research Institute).** Designed to determine transport of phosphorous into streams and measure phosphorous levels and costs to drinking water treatment. Ongoing findings should be made readily available to the public.
- **Regional Environmental Assessment Program (REMAP).** Initiated by the EPA, this program randomly selected between 2005 and 2007 sites for sampling algae and bacteria, among other things. The results for Region VI, as well as highlighted findings specific to Oklahoma, should be posted to the OWRB website.
- **Volunteer Monitoring HAB Assessment.** This program is designed for the “long-term monitoring of harmful algal blooms (HAB). These procedures will include the initial use of Enzyme-Linked ImmunoSorbent Assay (ELISA) kits by Oklahoma Water Watch (OWW) volunteers to determine if and in what concentration cyanobacteria are present in the water being monitored.” Using data collected by volunteers, the OWRB will issue a report of the Beneficial Use Monitoring Program (BUMP) in December of 2009, reporting on algae levels in several Oklahoma lakes.<sup>59</sup>
- **Nutrient Limited Watershed Impairment Study.** “The OWRB is currently developing a process to determine whether lakes with high algae growth are impaired due to excessive nutrients. Project results may be used to set water quality standards for nutrient impairment.”
- **Illinois River Probabilistic Sampling Project.** “The OWRB is conducting a probabilistic sampling study in the Illinois River sub-basin. From 2007-2009, randomly chosen sites will be sampled to characterize the biological, chemical, and physical properties of the water. Specifically, OWRB staff will collect fish, benthic macroinvertebrates, habitat, algae, bacteria, and nutrient samples during a series of

<sup>58</sup> Environmental Protection Agency. Safe Drinking Water Act website. Retrieved 9/14/09 from <http://www.epa.gov/OGWDW/sdwa/basicinformation.html>

<sup>59</sup> Oklahoma Water Resource Board. “Water Log” 2008. Vol. 13, no. 2. Retrieved online at [http://www.owrb.ok.gov/news/news2/pdf\\_news2/WaterLog/2008v13-2.pdf](http://www.owrb.ok.gov/news/news2/pdf_news2/WaterLog/2008v13-2.pdf)

site visits. Data will be used to determine the biotic integrity of the watershed prior to full implementation of the OWRB's scenic river total phosphorus criterion."<sup>60</sup>

In addition to ongoing assessments by the OWRB, the Oklahoma Conservation Commission (OCC) sponsors a Rotating Basin Monitoring Program to measure phosphorous, nitrates and macroinvertebrates—including zebra mussels—in Flint Creek, Barren Fork Creek, the Illinois River, and Peach Creek.<sup>61</sup> Additional assessments for microcystins, saxitoxins or BMAA bound in zebra mussels would further the scope of this OCC project in protecting human health. In late summer of 2009, the Department of Environmental Quality appointed a blue-green algae coordinator to encourage communication among agencies and with the public. Such infrastructure is in the early phases of determination.

Specific monitoring for cyanotoxins in drinking and recreational water appear limited to those programs in partnership with the U.S. Army Corps of Engineers. (e.g., see reports of the Tulsa District Water Quality Monitoring Program which report levels of a variety “cyanophyta”). Tony Clyde, from the USACE, and Dr. Robert Lynch, from the University of Oklahoma Health Science Center, have presented findings of cyanotoxins in some Oklahoma reservoirs. Ten samples were taken from Lake Marion between May 25, 2005 and September 28, 2005 that revealed concentrations up to 16.4 µg/L. Similarly, Fort Gibson contained up to 4.4 µg/L. Microcystin was found at two sites in Lake Tenkiller on July 13, 2005 with levels ranging from .35 µg/L to 3.3 µg/L.<sup>62</sup>

The Office of the Secretary of the Environment, the U.S. Army Corps of Engineers, the Oklahoma Water Resources Board, the Department of Environmental Quality and other lead agencies generally keep classified or unpublished many findings on blue-green algae concentrations in Oklahoma's drinking, bathing, recreational and irrigational water sources. This makes protecting the public interest particularly difficult for local water authorities or small municipalities. Smaller systems oftentimes fall through the cracks, as a recent HAB event at Lake Elmer, near Kingfisher, tragically illustrates.

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<sup>60</sup> Oklahoma Water Resources Board. “2008 Projects and Programs Update.” Available from <http://www.owrb.ok.gov/about/management/OWRBprograms.php>

<sup>61</sup> Office of the Oklahoma Secretary of the Environment. 2008. “Coordinated Watershed Restoration and Protection Strategy for Oklahoma's Impaired Scenic Rivers: Update per Senate Bill 972.” p. 2 & 19 - 20 Retrieved online at: [http://www.environment.ok.gov/documents/Final%20SB972report\\_2008update.pdf](http://www.environment.ok.gov/documents/Final%20SB972report_2008update.pdf)

<sup>62</sup> Lynch, Robert and Clyde, Tony. April 2009. “A Survey of Cyanobacterial Toxins in Oklahoma Reservoirs.” Paper presented to The 18<sup>th</sup> Annual Oklahoma clean Lakes and Watersheds Association Conference on April 1 – 3, 2009. Quarts Mt. Resort, Arts and Conference Center.

## Oklahoma State and National Epidemiology

On August 23, 2009 *The Oklahoman's* outdoor editor, Ed Godfrey, reported in a few sentences a “major” fish kill at Lake Elmer.<sup>63</sup> However, several weeks prior to such limited public dissemination of the event, a few online discussion groups alerted sportsmen to the problem. In a post to [www.fishingworks.com](http://www.fishingworks.com), “Karen” wrote August 9, 2009 “What is going on at Lake Elmer? We were there today and the entire shoreline is covered with dead fish. I was checking here to see if there were any notices about it.” Five days later, “Jesse” posted to the same group, “Me and my grand parents went there today and the whole place was covered with dead fish, big and small. There's not a single one alive and we don't know why.”<sup>64</sup> “Shortbus” posted to [www.huntandfishfinders.com](http://www.huntandfishfinders.com) on August 12, stating “I received a phone call from my brother on Monday telling me he was at Lake Elmer in Kingfisher and that there was a massive fish kill. He said the lake had turned over and that it looked like ALL of the fish in the lake had died--every single one of them. I could tell by his voice that he was absolutely SICK at the sight of all those dead fish -- Huge flathead catfish, 10 pound bass, carp and shad everywhere.”<sup>65</sup>

John Stahl, northwest regional fisheries supervisor for the Oklahoma Department of Wildlife Conservation, recalled during a phone interview October 2, 2009<sup>66</sup> that the lake was once “a jewel among fisheries.” He was present that Saturday morning in early August to witness the fish, just as they were in “the act of dying.” He equated watching the death in the lake with that of experiencing the loss of a child.

A longtime veteran of water conservation, Stahl indicated that *initial* deterioration of the lake began in 1986 when a flood caused an unusual flow into the lake of nutrient-rich sediment from fertilized farmlands across the street. “Phytoplankton” subsequently persisted until approximately 2005/2006 when the lake experienced a bloom-induced fish kill. Although this kill was not nearly as significant, comparatively, it alarmed officials who were able to circulate the water early enough and sufficiently enough to prevent further blooms, and so limited algal growth and toxin production that also stimulates oxygen depletion.

Likewise, Stahl attributed the summer 2009 fish kill at Lake Elmer to a bloom that endured between mid-July to early August when it covered the lake in a “brilliant emerald green.” Three inches of rain on the watershed then “caused the lake to flip,” Stahl explained, which stimulated a “crash” of the bloom. The crash, or death of algal cells, would have inevitably caused toxin release into the water.

<sup>63</sup> Godfrey, Ed. August 23, 2009. “Outdoors Notebook: Lake Elmer Suffers Major Fish Kill.” *The Daily Oklahoman*. Retrieved online at: <http://newsok.com/article/3394727>

<sup>64</sup> Lake Elmer Fishing in Kingfisher County, Oklahoma. Select “Comments.” Retrieved online at <http://www.fishingworks.com/lakes/oklahoma/kingfisher/dover/lake-elmer/>

<sup>65</sup> [huntandfishfinders.com](http://www.huntandfishfinders.com): The ultimate Online Hunting and Fishing Resource.: See “Massive Fish Kill at Lake Elmer in Kingfisher Oklahoma. Retrieved online at: <http://www.huntandfishfinders.com/forum/general-fishing-discussion/6540-massive-fish-kill-lake-elmer-kingfisher-oklahoma.html>

<sup>66</sup> Phone interview initiated by Dana T. Cesar

Lacking support in the way of EPA water safety guidelines and funding for training and continual monitors of microcystins, saxitoxins and BMAA, the water was merely tested for oxygen depletion. It appears that officials, armed with such findings, are often lead to dismiss the potential for further explanation for fish kills, or else they determine toxicity as “natural” and thus unavoidable. Due to a broad lack of awareness, investigations of fish kills in the state are frequently both under-analyzed and inconclusive as to any potential threat to human health. Officials charged with the task of protecting Oklahoma’s fisheries and recreational water, and those responsible for the quality of drinking water, appear uncoordinated in reporting HAB events and testing protocols, despite the fact that consumers may be exposed similarly.<sup>67</sup> Consequences to human health are unclear as there is no infrastructure for tracking HAB-related illness.

Devastated Kingfisher residents held a Town Hall meeting for which state Senator Mike Johnson was present. A meeting was held for the Kingfisher Lion’s Club, as well. The town’s people have determined that the lake should be dragged to remove nutrient sediment. The cottonwood trees on the south and west sides of the lake should be removed to allow more wind flow that would discourage blooms, but would leave fertilizer runoff undeterred.<sup>68</sup> Other structural improvements are planned to make the lake more accessible and inviting, once again.

Unfortunately, Kingfisher is not unique in its experience of lethal blooms. On May 26, 2009 the *Daily Oklahoman* reported as “natural” a fish kill at Crystal Beach Lake in Woodward.<sup>69</sup> In the April 1, 2008 Altus Municipal Trust Authority Meeting Minutes, a report was given on a fish kill that was attributed to Golden Algae. Twelve tons of fish were removed by “workers from every department within public works as well as other volunteers.” There was no mention of further tests for microcystins, saxitoxins or BMAA for which international safety guidelines have been suggested.<sup>70</sup> An August 4, 2007 story reported that “Officials are trying to determine what killed about 190,000 fish that were found dead Wednesday in Grand Lake.”<sup>71</sup> Fish kills were also reported in Lake

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<sup>67</sup>Rottmann, R.W., Francis-Floyd, R., Reed, P.A. and Durborow, R. February 1992. “Submitting a Sample for Fish Kill Investigation.” Southern Regional Aquaculture Center. Publication No. 472. The following entities should be expected to test for cyanotoxins in fish kills. Certain samples may be submitting to: Southeast District Fish Disease, Diagnostic Labs, Oklahoma Cooperative Extension Service, P.O. box 1378, Ada OK 74820 (405) 332-4100 or Fish Culture Project, Langston University, P.O. box 730, Langston OK, 73050, (405) 466-3836. Retrieved online at [http://www.fws.gov/southwest/es/Oklahoma/Documents/Contaminants/PAS%20S\\_Canadian%20River%20Final.pdf](http://www.fws.gov/southwest/es/Oklahoma/Documents/Contaminants/PAS%20S_Canadian%20River%20Final.pdf)

<sup>68</sup> Bunds, terraces and three-shrub-grass buffers are important to controlling runoff that causes nutrient load, which, in turn feeds BGA and HABs. See McIntyre, B., Herren, H.R., Wakhungu, J., Watson, R.T. 2009. *International Assessment of Agricultural Knowledge, Science and Technology for Development: Global Report*. pp. 40 & 180 IAASTD: Washington, DC.

<sup>69</sup> *The Daily Oklahoman*. May 26, 2009. State Briefs: “Fish Kill Said to be Natural.” Retrieved online at <http://newsok.com/state-briefs-fish-kill-said-to-be-natural/article/3372620#ixzz0SgwbR17K>

<sup>70</sup> Altus Municipal Trust Authority Meeting Minutes. April 1, 2008 RECEIVE BRIEFING ON STATUS OF CITY RESERVOIR REGARDING GOLDEN ALGAE AND ASSOCIATED FISH KILL. Retrieved online at [http://www.cityofaltus.org/images/uploads/dept\\_0105/FY2008/ta040108.pdf](http://www.cityofaltus.org/images/uploads/dept_0105/FY2008/ta040108.pdf)

<sup>71</sup> Godfrey, Ed. August 3, 2007. “Thousands of Dead Fish Found.” *Daily Oklahoman*. Retrieved online at <http://newsok.com/article/3097720>; See also Morgan, Rhett. August 4, 2007. “Massive Fish Kill at Grand Lake is Being Investigated.” Retrieved online at [http://www.tulsaworld.com/news/article.aspx?articleID=070804\\_1\\_A15\\_spnc51268](http://www.tulsaworld.com/news/article.aspx?articleID=070804_1_A15_spnc51268); See also p. 5 & A3 of the “Comprehensive Study of the Grand Lake Watershed,” called for by Senate Bill 408 and published December of 2005, “. . . the State first documented serious water quality threats in the 1980s when algae blooms and other signs of nutrient enrichment began to surface in certain areas of the lake. While water quality in Grand Lake was historically excellent, evidence of accelerated eutrophication (driven in this case by excessive phosphorus) has become an alarming trend that shows no signs of improvement with each passing year of water quality data collection. Today, Grand Lake and many of its tributaries are listed as impaired

Lawtonka in 2003, during which “several-hundred channel catfish and several-thousand drum died in episodes over 2 months during the peak public use period,” and in 2004 during which “20,000 drum and crappie died sporadically near the dam.”<sup>72</sup> In general, algal blooms in the state appear to be *far* under-reported in comparison with the dramatic consequences of blooms after toxin release, such as foul-tasting drinking water or fish kills in state waterways.

Notably, a recent epidemiological case in New England goes much further in illustrating the dramatic consequences of blooms to human health. This more recent case, reported in June 2009 by the *Valley News*, involves communities surrounding the Mascoma Lake and River in New Hampshire and Vermont in which nine people living near the lake have “been diagnosed with ALS since 1990 . . . all but one between 2000 and 2006.” The local paper reported that researchers were additionally investigating suspected clusters in Burlington and Plainfield Vermont, Sommersworth, New Hampshire, and Middleboro, Massachusetts. A Dartmouth-Hitchcock Medical Center (DHMC) neurologist who has been mapping cases of the disease in New Hampshire, Vermont and Maine, indicated rates of ALS around Mascoma Lake are “25 times greater [in prevalence] than national norms.”<sup>73</sup>

The local newspaper informed the public,

**ALS is a degenerative neurological disease with no known cause or cure. Although microcystin can affect the nervous system, the researchers are interested in a different neurotoxin produced by cyanobacteria, called Bmaa (B-Methylamino-L-alanine). A study published in the journal Neurology in 2002 found a potential link between Bmaa and a cluster of ALS in Guam. ‘We have not found the actual toxin in the bodies of water,’ said Elijah Stommel, a neurologist leading the study at DHMC.<sup>74</sup>**

In light of the slow release properties of BMAA, New Hampshire’s ALS victims may have been exposed to toxins many years prior to the presentation of their symptoms. Further, medical researchers studying the New Hampshire cluster related, “incidence of ALS cases can double for people living near waterways with cyanobacteria blooms.”

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on the 303(d) lists of Oklahoma, Missouri, and Kansas for such causes as nutrients, sedimentation, low dissolved oxygen, lead, zinc, and cadmium.” Although the report stipulated that risk to exposure was “low,” it cited “Algae of the genus *Aphanocapsa* presented the greatest risk for cyanotoxins. *Aphanocapsa* is known to produce microcystin, a hepatotoxin. An unusual note was that the Haptophyte, *Chysochromulina parva*, was noted in the February and May 2005 samples. This mixotrophic algae is most recognizable as the successor following a *Prymnesium parvum*, AKA ‘Golden Algae’, bloom.” Report retrieved online at: <http://www.environment.ok.gov/documents/bills/GrandLakeSB408FinalReport.pdf>

<sup>72</sup> Cofer, L., Bodine, K., Perry, J. December 31, 2008. “Lake Lawtonka 5-Year Fisheries Management Plan.” Southwest Region Oklahoma Department of Wildlife Conservation. Retrieved online at <http://129.15.97.19/odwc/Lawtonka%20Lake%20Management%20Plan%202008.pdf>

<sup>73</sup> Gregg, John P. and Woodrow Cox, John June 9, 2009.. “Disease Cluster Found at Lake: Researchers Seek Link Between Mascoma ALS, Algae.” *Valley News*. Retrieved online at <http://www.vnews.com/als/20090609-als.html>; Downs, Martin F. June 17, 2009 “Warning Posted at Mascoma: Cyanobacteria Bloom Identified.” *Valley News*. Retrieved online at <http://www.vnews.com/als/20090617a-als.html>; See also <http://des.nh.gov/organization/divisions/water/wmb/beaches/index.htm>; and <http://www.owrb.ok.gov/about/management/OWRBprograms.php#hab> for information on volunteer programs.

<sup>74</sup> Downs, Martin. June 17, 2009. “Warnings Posted at Mascoma: Cyanobacteria Identified.” *Valley News*. Retrieved online at <http://www.vnews.com/als/20090617a-als.html>

Water treatment officials in one local community were quoted as saying that “cyanobacteria toxins are not among the harmful contaminants for which the Environmental Protection Agency requires drinking water to be tested.”<sup>75</sup>

It’s true that while the EPA did include freshwater cyanobacteria and their toxins on the first Candidate Contaminant List (CCL) in 1998, the national agency has not required states to monitor for cyanotoxins. However, in August 2009, the national State EPA Nutrient Innovations Task Group finally released an *Urgent Call to Action* for the immediate reversal of phosphorus and nitrogen pollution that feeds algal blooms. Oklahoma water quality veterans, Derek Smithee and Jon Craig, served and/or deliberated on the national Task Group that released the call. In addition to the impacts to human health and quality of life in Oklahoma not fully covered in the report, the task force proposed “the long-term cost savings in drinking water treatment for 86 systems would range between \$106 million and \$615 million if [nitrogen and phosphorous] regulations were implemented (Moershel and Derischweiler 2009, personal communication).”<sup>76</sup>

The beneficial relationship between algae and zebra mussels in their mutual propagation highlights the vital importance of the proactive civil service of Smithee and Craig on the EPA Nutrient Innovations Task Group. Ironically, our modes of food production, often out of sympathy with the larger ecosystem, are a primary contributor to conditions that infect the water cycle and food web with cyanotoxins.<sup>77</sup>

Oklahoma’s abundant shoreline, coupled with the state’s industrial farming of chicken, beef and pork, renders the state particularly vulnerable to algal toxins, as illustrated by the September 2009 federal case, *State of Oklahoma v. Tyson Foods, Inc, et al.*<sup>78</sup> At the judicial level, this case points out the relationship between poultry production since the 1940’s to “over 150 million in 2002,” and the correlation with cyanobacterial outbreaks in the Illinois River Watershed and Lake Tenkiller.<sup>79</sup> This history-making case against major producers of poultry waste is the state’s most definitive step toward protecting the public health of Oklahoman’s. The outcome of the case will set national precedent for state responses to nutrient pollution by transnational or multinational corporations that have encouraged deadly blooms in the nation’s waterways.

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<sup>75</sup> Downs, Martin. June 11, 2009. “Scientists Allay Fears of ALS: Lebanon Confident Its Water is Safe.” *Valley News*. Retrieved online at <http://www.vnews.com/als/20090611-als.html>

<sup>76</sup> Environmental Protection Agency. State-EPA Nutrient Innovations Task Group. *An Urgent Call to Action: Report of the State-EPA Nutrient Innovations Task Group*. Retrieved online at <http://www.epa.gov/waterscience/criteria/nutrient/nitreport.pdf>

<sup>77</sup> McIntyre, B., Herren, H.R., Wakhungu, J., Watson, R.T. 2009. *International Assessment of Agricultural Knowledge, Science and Technology for Development: Global Report*. p. 8 – 9, 24, 40 – 41 & 44 IAAKSTD: Washington, DC.

<sup>78</sup> United States District Court for the Northern District of Oklahoma. Case No. 05-CV-329-GKF-SAJ. Filed in USDC ND/OK on December 7, 2007.

<sup>79</sup> Teaf, Christopher M., et al. October 2008. “Toxicological Considerations, Microbial Issues and Health Risks Related to Field Application of Poultry Waste.” Presented at the University of Massachusetts Amherst Annual International Conference on Soils, Sediments, Water and Energy. Center for Biomedical & Toxicological Research, Florida State University

## Conclusion

In April of 2008 the UN released a preliminary report of the International Assessment of Agricultural Knowledge, Science and Technology for Development (IAAKSTD) that represented the findings of over five years of collaboration among more than 400 scientists from over 100 countries, and that was peer reviewed by individuals, governments and private and non-governmental organizations. The IAAKSTD has determined that

**A key objective of agricultural policies since the 1950s, both in industrialized and in developing countries, has been to increase crop [and breeding] production. In its production focus, these policies have often failed to recognize the links between agricultural production and the ecosystems in which it is embedded. By maximizing provisioning services, crop production has often affected the functioning of the supporting ecosystem services.<sup>80</sup>**

Impacts to the ecosystem are likely to intensify. In analyzing the effects of effluents of agricultural waste on the environment, projections indicate that “the global river N flux to coastal marine systems may increase by 10-20% in the next 30 years.” Rates will be much higher for developing countries.<sup>81</sup>

Livestock currently accounts for “a third of the loads of nitrogen and phosphorus into freshwater resources.”<sup>82</sup> Additionally, Lester Brown reports that topsoil is eroding from “U.S. cropland at 3.1 billion tons per year.”<sup>83</sup> The IAAKSTD estimates the global cost of soil erosion at more than US\$400 billion per year. These costs include

**. . . the cost to farmers as well as indirect damage to waterways, infrastructure, and health. . . Agricultural runoff pollutes ground and surface waters with large amounts of nitrogen and phosphorus from fertilizers, pesticides and agricultural waste. Agriculture is the main cause of pollution in US rivers and contributes to 70% of all water quality problems identified in rivers and streams.<sup>84</sup>**

Because the sole function and evolutionary mandate for cyanobacteria is to degrade matter, or clean up the mess, excess nutrients in freshwater systems naturally leads to expanding algal blooms, oxygen depletion, and “changes in resident organisms”<sup>85</sup> Once blooms are established, there is little to be

<sup>80</sup> McIntyre, B., Herren, H.R., Wakhungu, J., Watson, R.T. 2009. *International Assessment of Agricultural Knowledge, Science and Technology for Development: Global Report*. p. 41 IAAKSTD: Washington, DC.

<sup>81</sup> Ibid, p. 274.

<sup>82</sup> Ibid, p. 520.

<sup>83</sup> Brown, Lester. 2003. “Plan B: Rescuing a Planet Under Stress and a Civilization in Trouble.” Retrieved online from [http://www.earth-policy.org/index.php?/books/pb/pbch3\\_ss2](http://www.earth-policy.org/index.php?/books/pb/pbch3_ss2)

<sup>84</sup> McIntyre, B., Herren, H.R., Wakhungu, J., Watson, R.T., 2009 *International Assessment of Agriculture Knowledge, Science and Technology for Development: Global Report*. P. 518. IAAKSTD: Washington, DC.

<sup>85</sup> Ibid, p. 274

done aside from alerting citizens who may have few alternatives for access to water. Toxins, once released, may persist for months. Those with compromised immune systems are at greater risk for neurological disorders and cancers caused by algal toxins.

The precise etiology or metabolic pathways between agricultural waste, blue-green algae and such neurological diseases as Alzheimer's, Parkinson's, ALS or Lou Gehrig's Disease, as well as certain cancers, is still being determined. Nevertheless, pending empirical evidence and extensive epidemiological evidence warrants focused, coordinated discussion for immediate state action in mobilizing resources at "war-time speed" for the protection of the public against cyanobacterial toxin exposure.

Without immediate state action, the important, life-giving work began by Dr. Lois Pfiester in the mid-1980s will continue to languish, to the detriment of Oklahoma's people. To date, the water quality of Lake Thunderbird remains under-analyzed and inconclusive. The lake has been "listed on the 2002 303(d) list as impaired due to low dissolved oxygen and turbidity, both as the cause unknown." By 2006, "86% of the main body site samples were eutrophic . . . while six of forty-two (14%) samples exceeded the COMCD [Oklahoma Master Conservancy District] goal (20 µg/L)." The lake has not met chlorophyll-a standards and is currently "in queue by the Oklahoma Department of Environmental Quality for a TMDL[Total Maximum Daily Load]."<sup>86</sup> In the meantime, the *Norman Transcript* reported on August 15, 2009, "Although it's not yet time for Lake Thunderbird to 'turn over,' the warm, sunny days are causing the lake to turn green from a proliferation of alga, affecting the water's taste and odor, officials said. . . ."<sup>87</sup>

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<sup>86</sup> Oklahoma Water Resources Board. September 2007. "Lake Thunderbird Water Quality 2006 for the Central Oklahoma Master conservancy District. Final Draft Report" pp. 25, 28 & 32. Retrieved online at [http://www.owrb.ok.gov/studies/reports/reports\\_pdf/ThunderbirdWaterQualityReport2006.pdf](http://www.owrb.ok.gov/studies/reports/reports_pdf/ThunderbirdWaterQualityReport2006.pdf)

<sup>87</sup> Blakey, Tom. August 15, 2009. "Lake Turns Green: Taste, Odor Issues Being Addressed." *The Norman Transcript*. Retrieved online at [http://www.normantranscript.com/archivesearch/local\\_story\\_227011617](http://www.normantranscript.com/archivesearch/local_story_227011617)