State of Utah
GARY R. HERBERT
Governor

SPENCER J. COX
Lieutenant Governor

Department of
Environmental Quality

Amanda Smith
Executive Director

DIVISION OF WATER QUALITY
Walter L. Baker, P.E.
Director

M E M O R A N D U M

TO: Erica Gaddis

FROM: Chris Bittner

DATE: October 13, 2014

SUBJECT: Cyanobacteria and dog fatality in Utah Lake

Executive Summary

On October 6, 2014, a dog owner reported that his dog had died over the weekend approximately three hours after drinking water that contained a lot of "algae" from the Lindon Marina at Utah Lake. That same day, division scientists traveled to the lake to collect samples of plankton and water for analysis. The dog's body was sent the following day, October 7, 2014, to Utah State University for a necropsy (autopsy). The necropsy report dated October 10, 2014, indicates that acute cardiovascular collapse consistent with exposure to a neurotoxin was the cause of death. Tissue samples are currently being analyzed for cyanotoxins, and the results may affect the conclusions presented in this memorandum.

Three genera of cyanobacteria known to produce cyanotoxins were identified in the plankton samples taken from the lake, with two cyanotoxins detected in the water samples. At this point, the dog's death appears to be linked to drinking the water and ingesting cyanobacteria from Utah Lake. This conclusion is based on the following findings:

1) The rapid death after drinking the water/cyanobacteria and the cause of death are consistent with neurotoxin exposure
2) Cyanobacterium known to be capable of producing neurotoxins were present in the lake water
3) Testing confirmed the presence of cyanotoxins, but not neurotoxins, in the water.

The two cyanotoxins detected in the samples can be lethal, but are not as rapidly as observed for the dog. The neurotoxin anatoxin-a, which is known to be produced by one of the identified cyanobacteria, is hypothesized to be the immediate cause of the dog's death. The apparent absence of neurotoxins in the water sample cannot be explained other than to note the relatively short half-life (24 hours) of anatoxin-a in the environment and the potential presence of anatoxin-a analogs that were not analyzed for because standard methods are unavailable. The anatoxin-a detection

limit is likely adequate for detecting lethal concentrations.

Introduction

The purpose of this memorandum is to evaluate the currently available data regarding the dog fatality at Utah Lake on October 6, 2014. The following day, the dog's owner reported the incident to Carl Adams at the Utah Division of Water Quality (DWQ). The owner reported that the dog died approximately three hours after playing in and drinking the water at the Lindon Marina. The owner reported that the water contained a lot of "algae," and he tried to keep the dog out of the water.

In addition to notifying the Utah County Health Department, DWQ collected and analyzed samples of the water for the presence of cyanobacteria and cyanotoxins. The dog's body was sent to the Veterinary Diagnostic Laboratory at Utah State University for a necropsy (autopsy). The conclusions and recommendations in this memorandum are based on the information available at the time of its preparation. The conclusions and recommendations may change based on new data, in particular the pending results of the toxin analyses for tissue samples.

Investigation Results and Discussion

1. The cyanobacteria identified in the water samples (*Microcystis*, *Aphanizomenon*, and *Dolichospermum)* are primarily known for producing liver toxicants. *Aphanizomenon* is also known to produce the potent neurotoxin anatoxin-a.

2. The two cyanotoxins detected in the water samples, microcystin and cylindrospermopsin, are liver toxins (hepatotoxins). These were measured in the samples using enzyme-linked immunosorbent assay (ELISA) methods. Microcystin causes gastrointestinal disturbances, including vomiting. These toxins may also cause lung edema if aerosols are inhaled or aspirated.

The concentrations (11 micrograms per liter (µg/l) that were detected in the water samples are considered moderate when compared to the available public health advisory levels. For human health, the health advisory concentrations for water recreation recommended by the World Health Organization (WHO) and several states is10 µg/l. The 10 µg/l is based on exposure assumptions for humans recreating in the water where water is not intentionally ingested which are not representative of a dog intentionally drinking the water, but should be protective of a one-time exposure in dogs even allowing for a higher toxicological sensitivity in dogs to cyanotoxins.

It should be noted that the neurotoxins anatoxin and saxitoxin were not detected in the water samples, which were tested using ELISA methods. The anatoxin-a analysis requires high-performance liquid chromatography (HPLC) methods because no ELISA method is available to measure for these neurotoxins. In addition, standard analytical techniques are unavailable for anatoxin-a analogs (for instance, anatoxin-a(s), homoanatoxin) that have, or are expected to have, the same mode of toxic action as anatoxin-a. No saxitoxin-producing cyanobacteria were observed at the lake, so detection of saxitoxin is not expected.

Anatoxin readily degrades under environmental conditions with an environmental half-life of 24 hours, whereas the microcystins are much more persistent. The half-life of anatoxin-a decreases

with increasing pH. Anatoxin-a was originally known as Very Fast Death Factor (VFDF) because it caused rapid death in animals exposed to the toxin.

Very limited data is available for determining health advisory concentrations for anatoxin. The Washington State Department of Health recommends a provisional value for human health of 1 µg/l that is anticipated to be protective of acute and chronic exposures. Lethal concentrations of anatoxin-a in water are predicted to be much higher than the 1 µg/l laboratory detection limit, supporting the conclusion that this method is sufficiently sensitive to detect lethal concentrations. This conclusion is based on a comparison to other potent neurotoxins such as botulinum toxin and the chemical warfare agent VX.

3. The dog's stomach had some dog food, but no plankton was observed in the stomach or intestines. The dog's owner reported that the dog vomited "algae", so the absence of plankton in the gut is not a reliable indicator that there was no exposure to cyanobacteria.

The dog necropsy concluded acute cardiovascular collapse as the cause of death. The animal's death was likely due to exposure to a toxin due to the absence of disease or structural abnormalities. No gross abnormalities of the liver were observed. Obvious liver damage is a characteristic of microcystin or cylindrospermopsin, but these affects would be expected to take more than three hours to manifest. Necropsies on waterfowl that died and were collected by the Division of Wildlife Resources (DWR) on October 8, 2014, also did not demonstrate visible liver damage.

Cardiovascular collapse can cause the observed congestion in the liver and spleen and pulmonary edema (fluid buildup) in the lungs of the animal, but exposure to hepatotoxins can also cause similar symptoms in the liver. Lung edema is also a characteristic of neurotoxins such as homoanatoxin that affect smooth muscles, but could also be a direct, as opposed to a systemic, effect from exposure to cylindrospermopsin or microcystin aerosols or aspiration when vomiting. The hepatotoxins detected in the water samples clearly can cause death, but death would occur days or weeks after exposure, making these toxins the unlikely cause of the dog's rapid death.

The rapid death due to cardiovascular collapse strongly supports a neurotoxin as the immediate cause of death. *Aphanizomenon* is known to produce anatoxin-a, which is potent and fast-acting but has a relatively short half-life. This conclusion will be further supported if anatoxin-a is detected in any of the tissue samples. However, anatoxin-a is rapidly degraded or metabolized in mammals, and the test results showing that anatoxin-a was not detected in the Utah Lake samples is not conclusive of its absence. The apparent absence of detectable of a neurotoxin in the water samples cannot be explained based on the currently available data.

Recommendations for future investigations include collecting environmental samples as soon as possible and analyzing the gut contents of affected animals for cyanotoxins.

References

R. Araoz, H.O. Nghiem, R. Rippka, N. Palibroda, N. Tandeau de Marsac, and M. Herdman. J. 2005. Neurotoxins in axenic oscillatorian cyanobacteria: coexistence of anatoxin-a and homoanatoxin-a determined by ligand-binding assay and GC/MS. Microbiology. 151(Pt 4):1263-1273

Patockaa and L. Stredab. Brief Review of Natural Nonprotein neurotoxins. 2002. Applied Science and Analysis Newsletter, ed. Col. R. Price

B. Puschner,1 B. Hoff, E.R. Tor, 2008. Diagnosis of anatoxin-a poisoning in dogs from North America. J Vet Diagn Invest 20:89–92

J. A. Sanchez, P. Otero, A. Alfonso, V. Ramos, V. Vasconcelos, R. Araoz, J. Molgo, M.R. Vieytes, and L.M. Botana. 2014. Detection of Anatoxin-a and Three Analogs in Anabaena spp. Cultures: New Fluorescence Polarization Assay and Toxin Profile by LC-MS/MS. Toxins 6:402-415

T.A. Sarma. 2013. Handbook of Cyanobacteria. CRC Press

United States Environmental Protection Agency. 2006. Toxicological Reviews of Cyanobacterial Toxins: Anatoxin-A. External Review Draft. November

United States Environmental Protection Agency. 2006. Toxicological Reviews of Cyanobacterial Toxins: Microcystins LR, RR, YR and LA. External Review Draft. November

United States Environmental Protection Agency. 2006. Toxicological Reviews of Cyanobacterial Toxins: Cylindrospermopsin. External Review Draft. November

Washington State Department of Health, 2008. Final Report Washington State Recreational Guidance for Microcystins (Provisional) and Anatoxin-a (Interim/Provisional). July

World Health Organization (WHO), 1999. Toxic Cyanobacteria in Water: A guide to their public health consequences, monitoring and management. edited by Ingrid Chorus and Jamie Bartram