

Management of Cyanobacteria (Blue-green Algae) and Other Harmful Algal Blooms in Ponds: Protecting Your Livestock, Companion Animals, and Fish

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Many news stories in recent years have made national headlines due to cyanobacteria, more commonly referred to as blue-green algae. In 2019, three dogs died after swimming in a pond in Wilmington, North Carolina, while another died after swimming in Lake Allatoona, Georgia (Fig. 1). In 2022, several dogs died after swimming in Jordan Lake, North Carolina, while in Texas, several dogs died after swimming in Barton Creek or Lady Bird Lake near Austin. That same year, more dog deaths were suspected to be linked to cyanobacteria after swimming in water where blooms were occurring near Eureka, California.

In 2024, multiple dogs died after playing in the Columbia River, Oregon, with at least one case being confirmed as cyanotoxin-induced. A dog died in August 2024 near St. Helens, Oregon, after ingesting water containing cyanobacteria. Many more cases of mysterious companion animal deaths every year are likely cyanotoxin-induced, but the symptoms may be missed, as they may be confused with

similar symptoms caused by different conditions. Cyanotoxins are difficult to test for in tissues. There are very few laboratories in the U.S., even at major university veterinary hospitals, that can test for cyanotoxins. Often, veterinarians may not know the symptoms and signs, or understand that cyanotoxin poisoning could be the culprit, so many times, cyanotoxin deaths go unreported.



Figure 1. It is important to immediately remove access for all animals to water bodies suspected of having a cyanobacteria bloom. Do not allow livestock or companion animals to swim in or drink from water that you suspect may have an active cyanobacteria bloom.

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Unfortunately, this is just the tip of the iceberg, as each year, the Texas A&M Aquatic Diagnostics Laboratory receives dozens of cases of livestock deaths caused by cyanobacteria, while the Texas A&M Veterinary Medical Diagnostic Laboratory and the Texas A&M AgriLife Extension veterinarians receive numerous more cases of livestock deaths. Statewide, dozens if not hundreds of fish kills are caused by cyanobacteria or other harmful algae species. Most are attributed to low dissolved oxygen, as many of the symptoms are the same, or fish kills are simply classified as unknown causes because the landowners do not know the signs or symptoms of a harmful algal bloom or cyanotoxin-induced fish kills (Fig. 2).



Figure 2. Cyanotoxin-induced fish kills are common occurrences in Texas farm ponds during hot summer months, but they can occur at any time of the year.

Description

What are cyanobacteria?

Cyanobacteria, also known as blue-green algae, are among the oldest life forms on Earth. They first appeared around 2.7 to 3.5 billion years ago, although some studies place them as old as 4 billion years. Most cyanobacteria are very small, less than 1 μm up to 100 μm , but most fall within 2–25 μm (0.002–0.025 millimeters). However, cyanobacteria can be unicellular (smallest), colonial (larger; Fig. 3), and filamentous (largest), so they can appear to be a much larger organism than the individuals that form these colonial units. Cyanobacteria were the first organisms to perform oxygenic photosynthesis, using water as an electron donor and producing oxygen. Their photosynthetic system includes photosystem I and photosystem II, resembling those found in modern plants, suggesting that chloroplasts evolved from an endosymbiotic cyanobacterium. Cyanobacteria were pivotal in the Great Oxygenation Event about 2.4 billion years ago. Through oxygenic photosynthesis, they released oxygen as a byproduct, drastically changing Earth's atmosphere and enabling the evolution of aerobic organisms to support life on Earth.

Cyanobacteria have competitive advantages over the beneficial phytoplankton—specifically green algae species—that pond owners want to support. Green algae form the basis of a pond's food chain through primary production and sugar formation from



Figure 3. Some species of cyanobacteria are colonial and will start to form clumps within the water body. Many others are filamentous and may be mixed in among mats of harmless green algae. The majority of issues in Texas are caused predominantly by species comprised of single planktonic cells within the water column or at the surface.

photosynthesis, but they also are responsible for producing up to 80 percent of the available oxygen in a pond. Unlike green algae, when nitrogen becomes limited and growth becomes reduced, many, but not all, species of cyanobacteria can fix atmospheric nitrogen through specialized cells called heterocysts. This means that when nitrogen becomes limiting in a pond, the phytoplankton community can quickly shift over from being green algae-dominated to cyanobacteria-dominated. In addition, many species of cyanobacteria can produce toxins. These toxins are primarily used to limit competition for limited available nutrients by other phytoplankton species or to suppress the predation of cyanobacteria when grazing predation from zooplankton is increased.

Ecology and Distribution

Where are cyanobacteria found?

This is a common question, and often, pond owners and livestock producers have the false belief that these organisms are not in *their* pond because it looks clean or clear. However, cyanobacteria are found almost anywhere. They are what is called ubiquitous in the environment, meaning they can be found in most places. A pond owner can send a surface water sample to an algae identification laboratory from almost anywhere in the world, and the laboratory personnel will most likely find at least one species of cyanobacteria in the sample, if not multiple species. Cyanobacteria inhabit a wide range of ecosystems, from freshwater and marine environments to extreme habitats like deserts, hot springs, polar ice, and rock surfaces. They are aquatic and planktonic in lakes, rivers, and oceans, benthic or epiphytic, forming biofilms or mats on surfaces, or they can be symbiotic with fungi (lichens), protozoa, sponges, plants (e.g., *Azolla* and *Hydrilla*), and corals.

Cyanobacteria have been found in a wide range of extreme environments. They are known to thrive in deserts, Antarctica, and almost any damp soil. They have been observed in the following locations.

- **Deep underground:** Cyanobacteria have been discovered thriving nearly 2,000 feet below the Iberian Pyrite Belt in southwestern Spain. These microbes were found within fractures in the rock, utilizing hydrogen gas for energy (Bartels, 2018).

- **International Space Station:** Cyanobacteria have also been studied in the International Space Station, demonstrating their ability to survive in space (Gaskill, 2022).
- **Antarctica and deserts:** Cyanobacteria are well-known for their ability to survive in extreme conditions, including the harsh environments of Antarctica and deserts (Zych & Wing, 2017; Bowange, 2019).
- **Thermal springs:** Cyanobacteria are also found in thermal springs, including those in Hot Springs National Park, where they can tolerate high temperatures (National Park Service, 2021).
- **Various water bodies:** Cyanobacteria can be found in a wide variety of water bodies, including rivers, lakes, and even seemingly pristine subalpine watersheds.
- **Other environments:** They have also been found in saline environments, frozen systems including Siberian lakes under 2,000 feet of glacier, and in geothermal habitats.

This means they are most likely in any pond or local lake, river, or reservoir, as well.

What ecological role do they play?

Cyanobacteria are primary producers in aquatic food webs, meaning they undergo photosynthesis to produce sugars, creating new energy resources for the food chain. However, they are less desirable in this respect than green algae due to the fact that cyanobacteria can produce toxins. Cyanobacteria can fix atmospheric nitrogen using heterocysts, so they can introduce new nitrogen into nitrogen-limited systems. However, most of the nitrogen is first utilized by the cyanobacteria until death, at which time nitrogen may be released back to the aquatic environment. Cyanobacteria can create microbial mats and contribute to sediment stabilization. Although rooted, vascular aquatic vegetation is much better at sediment stabilization. Cyanobacteria can participate in biogeochemical cycles of carbon, nitrogen, and phosphorus. And finally, perhaps their most important function and the reason for the current habitable atmosphere, they produce copious amounts of oxygen through photosynthesis. They produce oxygen far in excess of the amount required by the cyanobacterial cell itself to survive, so they produce a surplus of oxygen.

Cyanobacteria Blooms

Under favorable conditions (e.g., nutrient enrichment, warm temperatures, and stable water columns with little mixing/stagnate water), cyanobacteria can form harmful algal blooms (HABs). Harmful algal blooms occur when colonies of algae—in this case, cyanobacteria or blue-green algae—grow out of control and produce toxic or harmful effects to fish, shellfish, wildlife, livestock, companion animals, or people. Human illnesses caused by HABs, such as paralytic, diarrhetic, neurotoxic, and amnesic shellfish poisoning, ciguatera fish poisoning, and red tide, though rare, can be debilitating or even fatal. Cyanobacteria blooms shade aquatic plants, reduce beneficial phytoplankton such as green algae, alter pH and oxygen levels, and produce toxins that harm animals or humans. They produce powerful neurotoxins and hepatotoxins that can kill fish within hours and livestock, wildlife, and companion animals within hours to weeks, depending on the toxin type and severity of exposure.

There are no set guidelines on what constitutes a cyanobacteria bloom, as cell count thresholds will vary by cyanobacteria species and type of toxin(s) produced, if any. Some sources state that any cyanobacteria species with cell counts of greater than 100,000 cells/mL of water constitutes a HAB, while the World Health Organization states that cell counts below 20,000 cells/mL pose a low risk of adverse health effects (Environmental Protection Agency, 2024). However, it is known that toxic levels of cyanotoxins, especially powerful neurotoxins such as saxitoxins and anatoxins, can be obtained from cell counts below 20,000 cells/mL. In general, any cyanobacteria cell count above 40,000 cells/mL should be cause for concern and treated as a HAB, or if the cell count of any cyanobacteria species is greater than 50 percent of the entire phytoplankton cell count, it should also be treated as a HAB.

The cell counts presented are a general guideline. Cell counts and the level of danger posed are highly variable, dependent upon cyanobacteria species and type of toxin produced. Some species of cyanobacteria that produce neurotoxins may be dangerous at cell counts as low as 20,000 per mL.

Cyanobacteria Species

The issue with using cell counts to determine HAB status is that cell counts are, at best, indicators of whether an issue *may* exist, but they do not complete the entire picture. Cell counts are best used to indicate the presence and density of a species, not the toxicity of the organism or the water. It is important to understand that there are over 2,700 scientifically described species of cyanobacteria, but it is estimated that there are between 6,000 and 8,000 species of cyanobacteria worldwide. At least 600 of these species are known in Texas. Not all cyanobacteria are created equally. Not all produce toxins. It is estimated that only about 50 to 55 percent of the known species in Texas produce toxins. Of the species that produce toxins, they do not produce toxins all the time. (There is debate among algal ecologists that they may always be producing and, possibly, releasing toxins at low levels, but these levels are too low for current methods to detect.) If they have the ability to produce toxins, that does not mean they always release toxins.

Key genera of concern include *Microcystis*, *Anabaena* (now *Dolichospermum*), *Nodularia*, *Planktothrix*, *Cylindrospermopsis*, and *Lyngbya*. These genera are frequently involved in blooms and cyanotoxin production and, therefore, should always be of concern when positively identified in samples and cell counts are greater than 20,000 cells/mL.

For example, if a cyanobacteria species is capable of producing a toxin, it does not necessarily need to produce and release a toxin (at least in large quantities) if the plankton population is low, there is negligible or limited competition from other plankton for nutrients, nutrient loads in the water are sufficient and nothing is limiting, and there is limited grazing predation from zooplankton or other organisms. If competition for nutrients or sunlight increases, a nutrient in the water becomes limiting, or grazing predation by zooplankton intensifies, then the organism is highly likely to start producing and releasing elevated levels of toxins to reduce competition or predation. Referring back to cell counts, there may be a population of cyanobacteria, say *Microcystis aeruginosa*, which produces microcystin, with cell counts of 60,000 cells/mL, but nutrients and sunlight are not limited, there is not a great deal of competition from other phytoplankton species, and there is limited grazing predation by

zooplankton. In that instance, the cyanobacteria would be perfectly happy and may never release any toxins at a significant level that could harm fish, wildlife, companion animals, livestock, or humans.

Now examine a population of *M. aeruginosa* with cell counts of only 20,000 cells/mL, throw in competition from copious other plankton species for light and nutrients, allow a nutrient such as nitrogen or phosphorus to become limiting, or increase grazing predation from zooplankton, and suddenly this low cell count is producing enough microcystin to cause a fish kill or poison livestock. Knowing the species and cell count is not sufficient to determine danger. You must determine the presence of the toxins to determine the safety of the water. And because cyanobacteria can switch toxin production on and off rapidly, if the water is safe today, it does not mean it will be safe tomorrow or next week.

An example of needing to know both species and cell count, along with toxin release, is presented in the next case, involving the ingestion of live cyanobacteria cells. Cyanobacteria in a pond may be present in large numbers, such as 120,000 cells/mL, and they might be producing toxins but not releasing them because they are not currently under threat of competition for nutrients or light, no nutrients are limiting, and grazing pressure is light. This is a case in which there are often dead livestock or companion animals, but there has been a complete lack of any fish kill. The cyanobacteria were not releasing any toxins to the water, so fish were not absorbing any toxins at the gill or through the skin and membrane layers. However, a cow that came along to drink from the pond not only took in 2.5 gallons of water (9.46 L), but it also ingested 1,135,200 toxin-containing algal cells. These cells rupture in the stomach acid of the cow, releasing the toxin. The cow has just become poisoned, possibly fatally, yet no fish kill has occurred.

Cyanotoxin Testing

An important aspect of any fishery or livestock operation is as soon as cyanobacteria are suspected in a water source, the water should be tested for cyanotoxins and possibly have cell counts and species identification performed. Most importantly, corrective action should begin immediately, whether it be through corrective management actions or

preventative actions, before the bloom gets to toxic levels. There are numerous private industry and university laboratories, and even many larger pond and lake management companies, that can perform algal (including cyanobacteria) counts and species identification. The [Texas Commission on Environmental Quality website](#) has a list of labs accredited by the National Environmental Laboratory Accreditation Program (NELAP), and many of them can perform algal counts and species identification.

Cyanotoxin testing is a bit more difficult. As stated previously, there are very few laboratories in the U.S., even at major university veterinary hospitals, that can test for cyanotoxins. The [Texas A&M Aquatic Diagnostics Laboratory](#) (ADL) can perform base detection of cyanobacteria-produced hepatotoxins in water samples. This qualitative test determines if one or more of 11 common hepatotoxins, produced by cyanobacteria, are present in the sample. Reports will indicate the presence or absence of toxins. These hepatotoxins are primarily microcystins (Genus *Microcystis*, one of the largest and most common genera of cyanobacteria) and nodularins (*Nodularia spumigena*, some of the most common late-summer cyanobacteria blooms), so it covers toxins produced by some of the largest groups of cyanobacteria. The test can detect cyanotoxin levels below 0.1 µg/L and indicate if there is an immediate danger to fish, livestock, or companion animals due to the presence of these hepatotoxins. The ADL does not offer quantitative testing to determine the total quantity of toxins. It only detects whether they are present.

It is important to note that hepatotoxins are not the only toxins that may be produced by cyanobacteria species. Hepatotoxins are highly toxic, resulting in severe liver damage and potential failure. They typically require repeated ingestion of contaminated water as the toxins build up in the liver over time, resulting in death in days to weeks. Hepatotoxins are the most common toxins affecting livestock production and the leading cause of cyanotoxin-induced death in livestock, but neurotoxins can kill within hours to days and cannot be detected by this method. It is important to note that if a test is negative for hepatotoxins, it does not guarantee that the water is free of toxins after the sample was collected, as there may be neurotoxin present, or the cyanobacteria present in the pond can later begin to release toxins. However, this assay method

uses a digestion phase, so if cells are present and producing toxin but not releasing it and the water is not yet toxic, the toxin will still be detected as the cells are digested, and the toxins are released during the assay. Other cyanotoxins that this test does not address include endotoxins (least toxic), cytotoxins (low to moderate toxicity), and neurotoxins (extremely toxic).

Cyanobacteria Toxins

The toxins produced by cyanobacteria are collectively known as cyanotoxins, but there are several subgroups within this category, such as cytotoxins, endotoxins, neurotoxins, hepatotoxins, and ichthyotoxins. For example, ichthyotoxins are classified as such because they specifically affect fish and can induce fish kills, whereas neurotoxins affect the central nervous system of any animal that ingests them. The dangers and impacts of cyanobacteria include toxin production that affects the liver, nervous system, and/or skin, water quality degradation in drinking sources and reduced livestock production, economic losses in fisheries, aquaculture, and tourism, and ecological disruptions, including mass mortality of aquatic fauna.

Human and animal health effects include acute poisoning from ingestion, inhalation of water or windblown aerosolized droplets containing cyanobacteria or cyanotoxins, or skin contact. Chronic exposure is linked to liver cancer, and neurodegenerative diseases, such as Alzheimer's and pet and livestock poisoning and deaths, occur from drinking contaminated water.

Endotoxins

Endotoxins are typically the least toxic and only become problematic after ingesting large quantities of the live cyanobacteria. They typically result in intestinal discomfort, vomiting, and diarrhea.

Cytotoxins

Cytotoxins are typically low to moderate in toxicity, causing minor to moderate irritation of the skin and mucous membranes. Throat, stomach, and intestinal irritation are common, and cytotoxins can lead to scarring over long periods, as well as bloody stool and diarrhea. Types of cytotoxins include:

- ▶ **Cylindrospermopsin:** Affects multiple organs; inhibits protein synthesis
- ▶ **Lyngbyatoxins:** Skin irritants with tumor-promoting activity

Hepatotoxins

Hepatotoxins are highly toxic and include substances that are harmful to the liver, causing damage or dysfunction. Exposure to hepatotoxins can lead to a variety of liver problems, including toxic hepatitis and liver failure. Hepatotoxins are the most common toxins affecting livestock production and the leading cause of cyanotoxin-induced death in livestock. Because of the slower nature of the toxin and the typical need to build up in the liver, dead livestock may be found spread out at various locations around the ranch and may be nowhere near water. Abdominal pain, fatigue, nausea, vomiting, loss of appetite, dark urine, and pale stools are other symptoms of hepatotoxins. Notice these symptoms overlap with several ailments or conditions in livestock and can easily be misinterpreted as caused by an issue other than cyanotoxins. Types of hepatotoxins include:

- ▶ **Microcystins (e.g., MC-LR):** Most common; affect liver cells by inhibiting protein phosphatases
- ▶ **Nodularins:** Similar action, mainly from *Nodularia spumigena*

Neurotoxins

Neurotoxins are highly toxic and can cause failure of the neurological system, resulting in heart cessation and breathing failure in minutes to hours. Neurotoxins kill quickly, sometimes in minutes to hours. It has been conjectured that they induce thirst in the affected organism, as they may continue to drink copious amounts of water and may ingest even more toxins at the same time. The rapid onset of neurological failure coupled with induced thirst is why livestock that have ingested neurotoxins from cyanobacteria are often found dead within, along the bank, or within a few feet of the pond or water source. Numbness, weakness, loss of coordination, paralysis, involuntary muscle movements, seizures, confusion, fatigue, insomnia, loss of consciousness, coma, changes in heart rate, vision loss, anxiety, depression, and changes in mood are all symptoms

that can be induced by neurotoxins. Unfortunately, death often occurs quickly before diagnosis can be made. Types of neurotoxins include:

- ▶ **Anatoxin-a:** Mimics acetylcholine, causing continuous nerve firing
- ▶ **Saxitoxins:** Block sodium channels, leading to paralysis (also produced by marine dinoflagellates)
- ▶ **Aplysiatoxins:** Potent skin irritants and tumor promoters

Signs and Symptoms of Cyanotoxin Poisoning in Livestock and Companion Animals

Unfortunately, laboratory diagnosis of cyanotoxin poisoning can be problematic because liver lesions, serum chemistry, and complete blood counts associated with this condition can also be seen with other illnesses. For example, some poisonous plants and trace mineral toxicities have similar effects, symptoms, and lab results. During clinical presentations, most animals die within a few hours after exposure. Damage to liver cells leads to a decrease in circulating blood volume and shock due to blood loss into the damaged liver and embolism in the lungs. Clinical signs include nervousness, reluctance to move, recumbency, diarrhea, and pale mucous membranes, often within 30 minutes of exposure (Fig. 4). Animals that survive often develop sun sensitivity of the skin due to liver damage.



Figure 4. Death can occur within 30 minutes to hours in companion animals and livestock that ingest water containing cyanotoxins, particularly those cyanotoxins that are neurotoxic. Do not allow companion animals or livestock to swim in or drink from waters that you suspect are contaminated with a cyanobacteria bloom.

Cyanotoxin poisoning in livestock and companion animals can manifest as a range of symptoms depending on the specific toxin and the animal species. Common signs that livestock producers and pet owners should be aware of include vomiting, diarrhea, weakness, drooling, and in later stages of neurotoxicity, difficulty breathing. Some toxins can cause tremors, seizures, or even paralysis. In severe cases, death can occur within minutes to hours of exposure. Specific signs and symptoms of cyanotoxin poisoning in animals may include:

- ▶ **Gastrointestinal:** Vomiting, diarrhea, and possibly bloody or black diarrhea
- ▶ **Neurological:** Weakness, staggering, tremors, seizures, difficulty breathing, muscle rigidity, paralysis, and even respiratory failure
- ▶ **Other:** Lethargy, pale gums, jaundice (yellowing of skin and eyes), excessive drooling, and skin irritation
- ▶ **Death:** Cyanotoxins can be fatal, especially in cases of neurotoxin poisoning, which can cause death within minutes to hours of exposure.

Factors Influencing the Severity of Cyanotoxin Poisoning

The specific cyanotoxin involved is important, as different cyanotoxins have different effects and levels of toxicity. Higher concentrations of toxins will likely cause more severe symptoms. Different animal species may have varying sensitivities to cyanotoxins. The route of exposure is also important, as ingestion, skin contact, or inhalation can all be routes of exposure. If you suspect your companion animal or livestock has been exposed to cyanotoxins, seek immediate veterinary care. Treatment may include supportive care, like IV fluids, oxygen, anti-seizure medication, and potentially, other treatments depending on the specific symptoms.

Patient Examinations and Diagnostics for Veterinarians

Patient Examination

Veterinarians need a good history before submitting tissues to a veterinary diagnostic laboratory. Submitting gastric or rumen contents is always

indicated. Submit all available tissues: liver, kidney, heart, lung, spleen, and brain. If possible, submit the entire carcass to the Texas Veterinary Medical Diagnostic Laboratory. Submit a concise case history along with the carcass, as well as water samples and any known or suspected toxic plants (Fig. 5).



Figure 5. It is important to remove access to water sources from all livestock and companion animals if a cyanotoxin bloom is suspected or animals demonstrate signs or symptoms of cyanotoxin poisoning. Remove access to animals from the water source immediately and provide them with another freshwater source. It is recommended to rinse animals thoroughly to prevent further exposure, especially to dermatotoxins.

Clinical Signs

Observe for symptoms that vary depending on the type of cyanotoxin involved, including:

- ▶ **Neurotoxins:** Rapid onset (within 60 minutes), muscle tremors, rigidity, seizures, difficulty breathing, excessive salivation, vomiting, diarrhea, paralysis, and potentially death
- ▶ **Hepatotoxins:** Onset within 4 hours, vomiting, diarrhea (possibly bloody), pale or yellow gums (jaundice), abdominal tenderness, loss of appetite, weakness, and signs of liver damage, such as low blood sugar and prolonged clotting times
- ▶ **Dermatotoxins:** Skin irritation, rash, itching, redness, and blistering
 - **Exposure history:** Ask the owner about potential exposure to contaminated water, especially if the animal was swimming in or

drinking from a pond, lake, or other body of water suspected of having a harmful algal bloom (HAB).

- **Physical examination:** Assess vital signs, particularly heart rate, respiration, and mentation. Look for any signs of organ damage, such as jaundice or abdominal tenderness.

Diagnostics

As previously mentioned, unfortunately, laboratory diagnosis of cyanotoxin poisoning can be problematic because liver lesions, serum chemistry, and complete blood counts associated with this condition can also be seen with other illnesses. For example, some poisonous plants and trace mineral toxicities have similar effects, symptoms, and lab results. Currently, there are no readily available clinical tests for the direct diagnosis of cyanotoxin poisoning in local veterinary clinics, and only a few specialized veterinary diagnostic laboratories in the U.S. have the capability to detect cyanotoxins in animal tissues.

General Laboratory Tests

- ▶ **General bloodwork:** Assess liver function, kidney function, electrolyte levels, and blood sugar. Elevated liver enzymes (e.g., ALT, AST, ALKP, and GGT) and bile acids can indicate liver damage.
- ▶ **Urine and fecal testing:** Analyze urine and feces for the presence of cyanotoxins or other indicators of poisoning. Urine testing for microcystin metabolites can be especially helpful in diagnosing past exposure.
- ▶ **Tissue samples:** Collect and test tissues, such as liver, kidney, heart, lung, spleen, and brain for the presence of specific cyanotoxins. If possible, submit the entire carcass to the Texas Veterinary Medical Diagnostic Laboratory along with a concise case history.
- ▶ **Imaging:** Consider a chest radiograph if respiratory symptoms are present.
- ▶ **Water sample testing:** Collect samples from the suspected water source for testing for cyanobacteria and cyanotoxins. Note that testing water samples does not confirm the presence of toxins in the animal, but it can provide valuable information about the potential exposure source.

- **Necropsy:** If the animal has died, a necropsy is highly recommended to assist in the diagnosis. Postmortem findings can include liver damage (e.g., necrosis and hemorrhage), renal tubular necrosis, and jaundice.

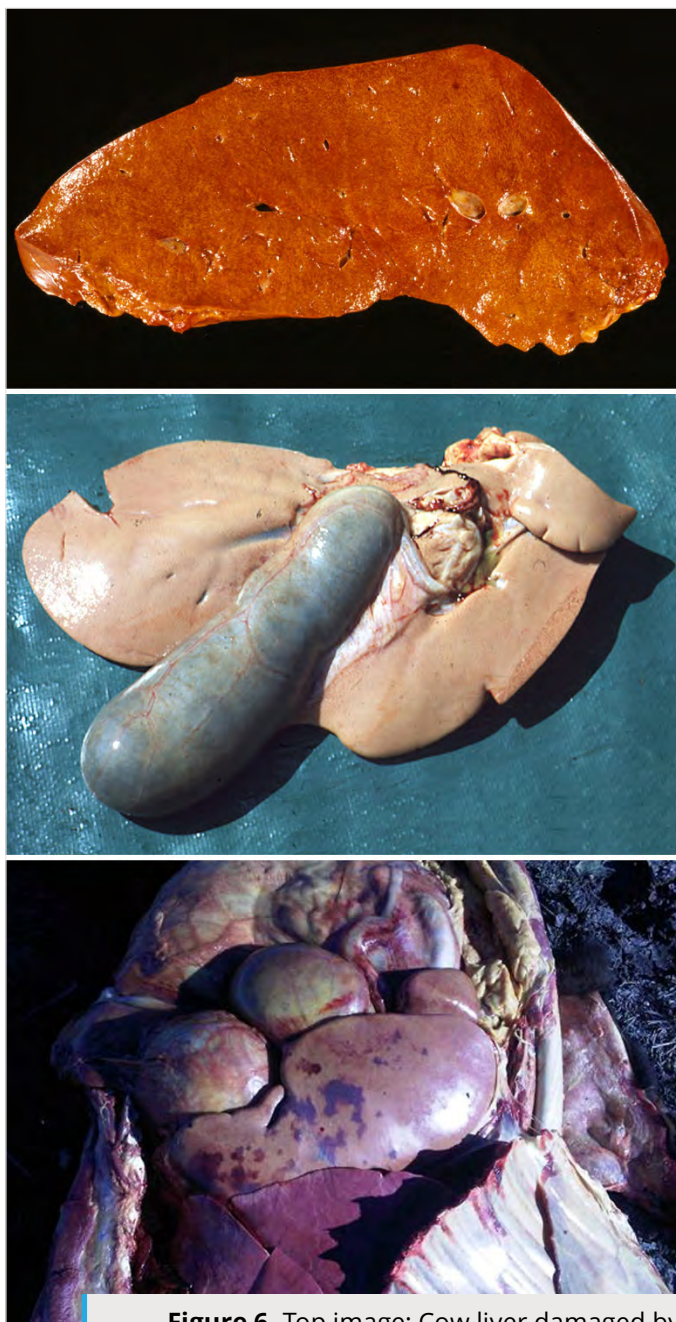


Figure 6. Top image: Cow liver damaged by cyanobacteria hepatotoxins; Middle image: Cow liver damaged by hepatotoxins with distended gall bladder; Bottom image: Cow liver showing pale coloration and hemorrhaging caused by hepatotoxins from cyanobacteria.

Important Notes

Currently, there are no readily available clinical tests for the direct diagnosis of cyanotoxin poisoning in veterinary clinics. Diagnosis is often based on a combination of clinical signs and exposure history. This is where a cyanotoxin test of the water source itself may prove useful to establish that toxins were, in fact, present and therefore could have been ingested. There are a few laboratories in the U.S., such as the Texas A&M Aquatic Diagnostics Laboratory and other university and commercial laboratories, that can detect various cyanotoxins in water to determine their presence. Specific cyanotoxin analysis in biological samples, such as liver, kidney, or nervous system tissue, typically requires specialized laboratories, which are even fewer than those that can detect cyanotoxins in water. While there is no antidote for cyanotoxin poisoning, supportive care is crucial, including intravenous fluids, liver support medications, and seizure control.

It is essential to prevent animals from accessing water bodies suspected of having HABs. As soon as an issue is detected or even suspected, all animals should be prevented from accessing the potentially contaminated water source until treatments have been applied and the water has been confirmed to be negative for toxins, or cell counts have been performed, and the cyanobacteria counts are well below the bloom threshold. If exposure is suspected, thoroughly wash the animal with clean water, provide a clean water source, and call the veterinarian immediately to make them aware of the situation. They may want to see the animal immediately, or they may want to be on standby with the correct supportive care if necessary. Reporting suspected cases of cyanotoxin poisoning to public health authorities and environmental agencies is vital for monitoring and prevention efforts.

How to Recognize Cyanobacteria Blooms

Early recognition of cyanobacteria blooms by aquaculturists, livestock producers, and pet owners is critical in both the management of the HAB and the protection of pets, livestock, wildlife, and even humans.

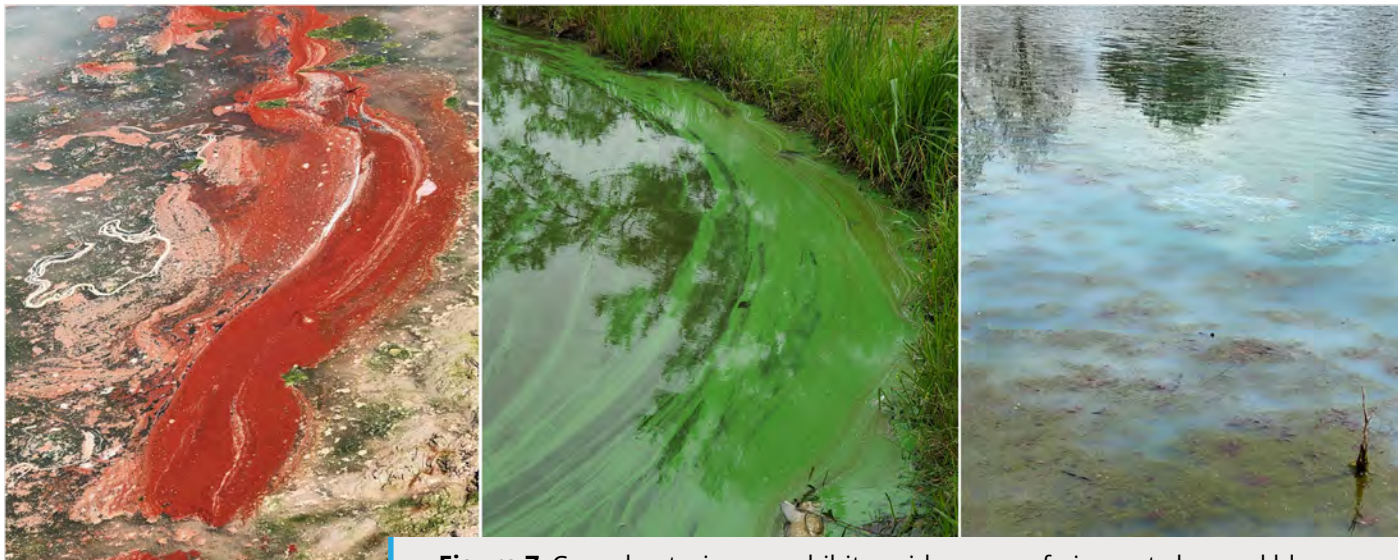


Figure 7. Cyanobacteria can exhibit a wide range of pigments beyond blue-green, including different shades of green, brown, yellow, turquoise, or even reddish hues.

How do I recognize cyanobacterium bloom or that I have cyanobacteria issues?

Due to the misleading nickname “blue-green algae,” many people have misconceptions about how to visually identify cyanobacteria blooms. While the iconic blue-green or turquoise surface scum is a well-known sign of a cyanobacteria bloom, this is just one of many possible appearances. Not only can cyanobacteria take on several different forms, ranging from single cells and colonies to filamentous structures, but they can also exhibit a variety of pigments beyond blue-green, including different shades of green, brown, yellow, or even reddish hues (Fig. 7). Therefore, color alone is not a reliable indicator of cyanobacteria presence. The absence of a typical blue-green coloration does not rule out the possibility of a bloom or the presence of toxins.

Pond owners should routinely inspect the water surface, particularly along the downwind shoreline where floating material tends to accumulate. Visual cues that may indicate a cyanobacterial bloom include surface films that resemble spilled paint in vivid colors (Fig. 8), such as turquoise, neon green, or red; clusters or specks that look like finely chopped grass clippings (Fig. 9); the appearance of an oily sheen; or floating globules and gelatinous mats in shades of dark blue, turquoise, or green. The “spilled paint” appearance or oily sheen are most often the most noticeable during the first 3 to 4 hours of daylight in the mornings. Most harmless

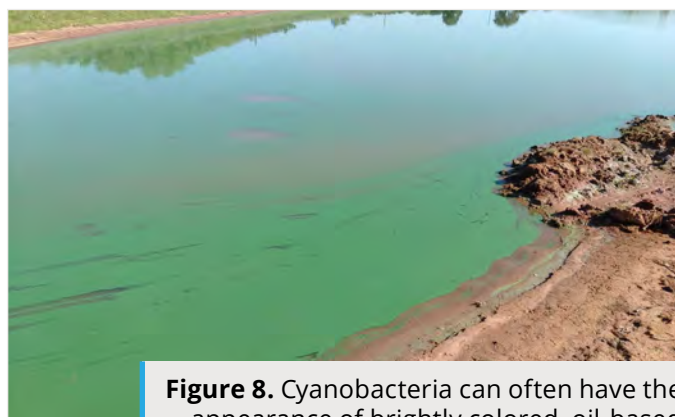


Figure 8. Cyanobacteria can often have the appearance of brightly colored, oil-based paint floating on the surface of the water.

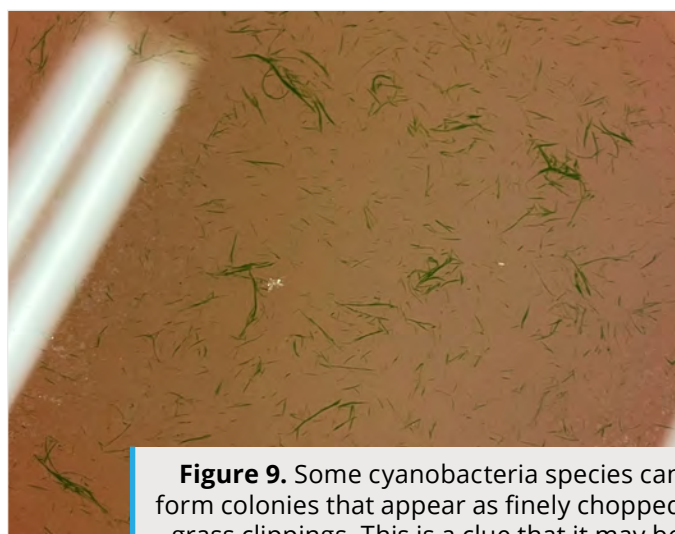


Figure 9. Some cyanobacteria species can form colonies that appear as finely chopped grass clippings. This is a clue that it may be cyanobacteria and not green algae.

species of green and brown algae are typically suspended evenly throughout the water column or are filamentous and form thick mats. Many species of cyanobacteria, however, can have gas vesicles that allow them to move vertically within the water to access light and nutrients, which is why the “spilled paint” or oily sheen may be most noticeable early in the morning after light levels have been low and the cyanobacteria come to the surface to obtain as much light as possible. This buoyancy control enables some species to form surface scums, which may appear as a nearly invisible sheen or which accumulate into thick, highly visible layers in areas where wind has concentrated them.

Importantly, the absence of visible surface scum does not guarantee that the water is safe. Some toxin-producing cyanobacteria are filamentous and can intermingle with common, harmless filamentous algae species that form dense, floating mats during the warmer months. These harmful forms can be nearly impossible to distinguish visually and require microscopy for identification. Another warning sign is the presence of small, irregularly shaped, round or egg-like clumps of dark green to dark bluish-green algae (Fig. 10) that float on the surface or accumulate along windblown edges. A common culprit, *Microcystis*, which produces the potent liver toxin microcystin-LR, often begins as a thin surface scum and later forms these floating clumps as the bloom intensifies. Therefore, any unusual surface accumulation, especially during hot, stagnant conditions, should prompt caution and potentially further diagnostic testing.

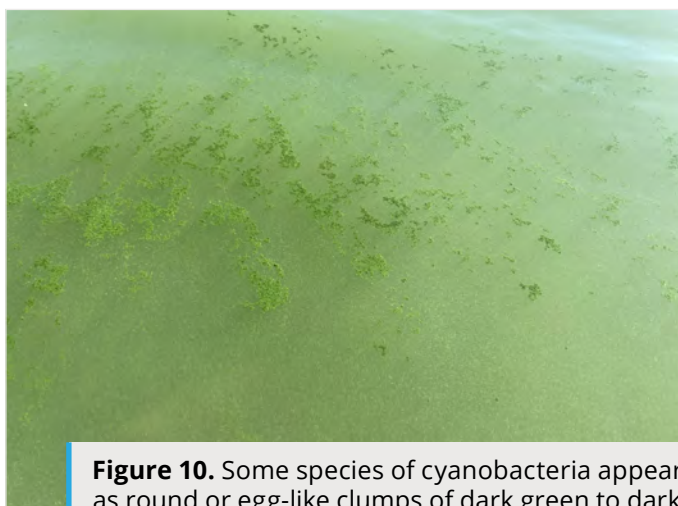


Figure 10. Some species of cyanobacteria appear as round or egg-like clumps of dark green to dark bluish-green algae that float on the surface or accumulate along windblown edges.

Preventative Management of Cyanobacteria Blooms

While reactive management strategies, discussed below in the Management of Existing or Ongoing Cyanobacteria Blooms section, can effectively reduce cyanobacteria blooms in the short term, they often fail to resolve the underlying causes, allowing blooms to recur. One of the primary drivers of cyanobacterial dominance is nutrient enrichment, particularly phosphorus and nitrogen, which fuel the blooms’ rapid growth. Although eutrophication is a natural aging process in aquatic systems, it is often accelerated by human activities such as urbanization, agriculture, and poor land management. Stormwater runoff can transport high levels of nutrients into waterbodies from sources like fertilizers, animal waste, leaf litter, and lawn clippings. These inputs not only support the excessive growth of algae and cyanobacteria but also contribute to oxygen depletion when these organisms die and decay, resulting in the buildup of organic-rich muck that further perpetuates nutrient recycling.

Effective mitigation requires a combination of in-pond treatments and watershed-based best management practices. Nutrient control is foundational and can include phosphorus-binding agents, beneficial bacteria, AC Aqua (an aquatically formulated soluble humic acid product; most terrestrial formulated humic acid products are not fully soluble in water), buffer strips, and artificial mixing through aeration. Phosphorus-binding agents, such as aluminum sulfate (alum) or lanthanum-modified bentonite, chemically inactivate soluble phosphorus by binding it into insoluble compounds that settle and become locked in sediments. While calcium-based products (e.g., calcium carbonate) can also precipitate phosphorus as calcium phosphate, they typically require higher application rates due to their lower binding affinity.

Bio-manipulation is another key strategy, which involves altering the biological components of the system to favor beneficial species of green algae. This can include adding AC Aqua, which binds phosphorus and also provides a carbon source for beneficial phosphorus cycling or phosphorus-storing bacteria, supplementing competitive phosphorus cycling bacteria to suppress cyanobacteria, artificial mixing through aeration, promoting green algae growth

through selective fertilization, using aquatic dyes to reduce light penetration, or introducing competitive rooted aquatic plants to uptake nutrients.

Beneficial nitrifying bacteria, often formulated in commercial blends, provide another layer of nutrient control by biologically consuming organic matter and promoting decomposition. These bacteria reduce the buildup of organic sediments and help cycle nutrients out of the system. The application is usually added directly into the water column, and product performance is temperature-dependent. Most strains become active when water temperatures exceed 60 to 65 degrees F, though product labels should be consulted for specific guidance.

Based on recent research conducted at Texas A&M University, AC Aqua has shown promising potential as part of an integrated nutrient management strategy for controlling HABs, particularly those caused by cyanobacteria. Similar to other nutrient binders, when applied to pond systems, AC Aqua can rapidly bind and precipitate available phosphorus in the water column and settle into the pond sediment, effectively locking the phosphorus out of the water column where it cannot be utilized by bloom-forming species. Additionally, the water-soluble humic acid in AC Aqua serves as a carbon source that stimulates the growth of naturally occurring or supplemented nutrient-cycling bacteria. These beneficial microbes use carbon to multiply rapidly, further sequestering phosphorus while also establishing a microbial community that competes directly with cyanobacteria for available nutrients.

It is important to note that humic acid is only soluble in water at a high pH. Most humic acid products available on the market for soil, lawn, and crop application are simply added to water at neutral to slightly acidic pH, and most of the humic acid is insoluble. When pouring these products through a sieve, large amounts of insoluble clumps resembling coffee grounds are retained. These clumps are ineffective during an aquatic application and simply sink to the bottom, becoming bound to sediment. Additionally, dry or liquid terrestrial “humic acid” products intended for agriculture are an umbrella term, covering humic acid, fulvic acid, humin, or most likely, a combination of

all three. These terrestrial products differ, and all these compounds vary in solubility and carbon content. It is important to choose an aquatically formulated, soluble humic acid when making aquatic applications for the management of algal blooms. One final note is that AC Aqua or other aquatically formulated humic acid products are only effective at water temperatures above 65 degrees F when phosphorus cycling and polyphosphate-accumulating bacteria are actively growing and reproducing. At temperatures below 60 degrees F, most of these bacteria in pond systems are dormant due to the cooler temperatures, and the addition of carbon sources such as AC Aqua will not be effective to support bacterial growth.

Artificial mixing through aeration can be used as a tool for managing cyanobacteria by disrupting thermal stratification and increasing dissolved oxygen levels throughout the water column. With many bloom-forming cyanobacteria species having the advantage in regulating their buoyancy to access resources, aeration interferes by providing vertical mixing. As a result, buoyant cyanobacteria are pushed downward and dispersed throughout the water, making it more difficult for them to maintain surface dominance. This reduces their access to optimal growth conditions and can limit bloom intensity, while simultaneously enhancing oxygenation and promoting the growth of beneficial aerobic microorganisms.

Selective fertilization, when done correctly, can play a strategic role in managing cyanobacteria by promoting the growth of beneficial phytoplankton, such as green algae, which compete with cyanobacteria for nutrients and light. By maintaining a healthy green algal population, fertilization limits the ability of nitrogen-fixing cyanobacteria to dominate, especially when small amounts of nitrogen are added to prevent nitrogen limitation. Proper nutrient balance based on total nitrogen and total phosphorus available in the water column helps maintain a stable algal community and prevents shifts toward harmful bloom-forming species.

Aquatic dyes, also known as pond dyes, can help manage cyanobacterial blooms by reducing light penetration, which shades the pond bottom and

limits the energy available for photosynthesis. This can suppress conditions that favor cyanobacteria. However, it is a non-selective approach that also inhibits the growth of beneficial algae, including phytoplankton that form the base of the aquatic food web. As a result, the use of dyes in ponds managed for fishing is generally not recommended unless supplemental food sources, such as commercial fish feed or forage species, are provided to support fish growth and maintain a balanced ecosystem.

Establishing and maintaining an emergent buffer zone is another low-cost, sustainable strategy that addresses the problem at its source by preventing excess nutrients from entering the pond. The shoreline, particularly the emergent zone—the vegetated area extending from just below the waterline to the upland bank—plays a vital role in cyanobacteria management by acting as a natural buffer that intercepts and filters excess nutrients like phosphorus and nitrogen before they enter the pond, ultimately fueling HABs. For guidance on how to establish and manage an effective buffer zone, refer to the publication [Creating a Buffer for Texas Ponds](#).

Finally, implementing best management practices (BMPs) throughout the surrounding watershed, such as reducing pasture or turf fertilization, controlling or limiting livestock access, stabilizing shorelines, and improving stormwater management, can help limit both point and nonpoint source nutrient inputs.

Management of Existing or Ongoing Cyanobacteria Blooms

While many strategies have been historically utilized to manage cyanobacteria blooms once they occur, the gold standard of recent decades has been copper-based and chelated copper algaecides. These include algaecides with aquatic labels that contain copper sulfate, copper ethylenediamine, and copper triethanolamine. Typically, copper is dosed at between 0.8 and 1 ppm (mg/L) of active compound to kill the cells, and the treatment is followed up 24 to 48 hours later with a phosphorus nutrient binder such as Phoslock, EutroSORB, aluminum sulfate, or AC Aqua. Less often, aquatically labelled herbicides containing alkylamine salts of endothall or sodium carbonate peroxyhydrate are used. It is more common to tank mix a copper-based algaecide with

herbicides containing alkylamine salts of endothall than to use herbicides containing alkylamine salts of endothall on their own.

The problem with the algaecide method is that it creates water that is even more toxic. After the algaecide is applied and the cells begin to die, they rapidly begin to lyse, releasing all of the toxin that was stored inside the cell. So, before the algaecide, there may have been water that was not toxic, but now is, or there may have been water that was slightly toxic and is now highly toxic after all the stored cyanotoxins are released. This can trigger fish kills and prompt other aquatic life to die off. It also puts pets and livestock in even greater danger of ingestion exposure and toxicity. Long-lasting in the environment, it may take more than a month for the water to become safe again.

An alternative strategy is to use an algaecide treatment followed immediately by a potassium permanganate treatment. Cyanotoxins typically must be oxidized to break down. The problem is that most oxidizers alone, such as bleach (sodium hypochlorite) or bromine, are extremely toxic to fish, can damage the gills and other membranes, and may result in a fish kill. It is for this reason that potassium permanganate is used as the primary means to oxidize cyanotoxins. Normally, 4 ppm (mg/L) of potassium permanganate is required to adequately oxidize the cyanotoxins. However, this concentration is dangerous to fish in many situations. Therefore, the potassium permanganate treatment is broken up into two separate treatments. To properly manage a cyanobacteria bloom, safeguard fish from a fish kill, and detoxify the water for livestock. The correct treatment order should be to first treat with 0.8 and 1 ppm (mg/L) of a copper-based algaecide, and immediately apply 2 ppm (mg/L) of potassium permanganate. Wait 2 to 6 hours based upon biological load (reapply when water turns from purple to lighter pink), and apply the second treatment of potassium permanganate at a rate of 2 ppm (mg/L). Follow up with nutrient binders or other forms of nutrient management 24 to 48 hours later to help prevent the cyanobacteria from reblooming after the release of all the stored nutrients from the dead algal cells.

An alternative management option based on recent research at Texas A&M University is to apply an aquatically formulated soluble humic acid product,

like AC Aqua. As previously stated, AC Aqua has shown promising potential as part of an integrated nutrient management strategy for controlling HABs, particularly those caused by cyanobacteria and golden algae. Similar to other nutrient binders, when applied to pond systems, AC Aqua can rapidly bind, precipitating available phosphorus in the water column and settling into the pond sediment. This process effectively locks the phosphorus out of the water column, where it cannot be utilized by bloom-forming species. Additionally, the soluble humic acid in AC Aqua serves as a carbon source that stimulates the growth of naturally occurring or supplemented nutrient-cycling bacteria. These beneficial microbes use carbon to multiply rapidly, further sequestering phosphorus, while also establishing a microbial community that competes directly with cyanobacteria for available nutrients. However, this is a slower-acting management practice, as the addition of AC Aqua has demonstrated the ability to slow the growth rates of *Microcystis* and *Prymnesium* blooms within 1 week. The populations begin to decline between 3 and 4 weeks after addition, with cell populations collapsing below bloom thresholds within 5 weeks.

AC Aqua presents a low-cost, easy-to-apply method of managing cyanobacteria blooms, but it is slow-acting and is best utilized as a preventative in ponds that are historically known to have or are in danger of forming cyanobacteria blooms. It is important to note that AC Aqua is not an algaecide or pesticide and does not directly kill the algal cells present. AC Aqua is a phosphorus binder and promotes the growth of competitive phosphorus-binding and phosphorus-storing bacteria, thus limiting available nutrients for cyanobacteria blooms.

Water Safety

How long do toxins last?

Cyanotoxins are persistent in the environment and can persist in water bodies for varying periods, from a few days to several months, depending on environmental conditions (Fig. 11). Factors like wind, sunlight, water temperature, rainfall, and specific toxin type influence the duration and presence of toxins. The majority of cyanotoxins will remain detectable for at least 3 weeks after an algaecide treatment has been applied to kill the cyanobacteria. However, others can persist for longer periods.

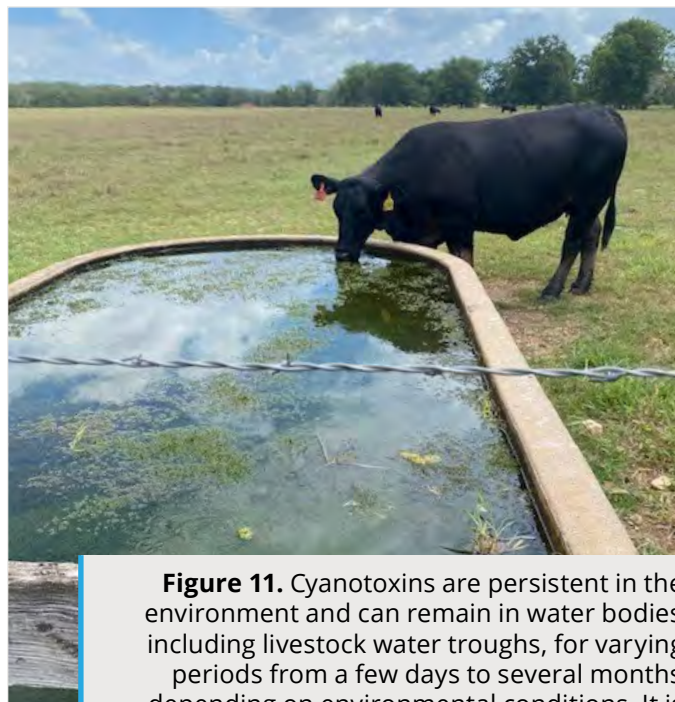


Figure 11. Cyanotoxins are persistent in the environment and can remain in water bodies, including livestock water troughs, for varying periods from a few days to several months, depending on environmental conditions. It is important to note that even livestock watering tanks and other water sources can become contaminated with cyanobacteria and should be monitored and cleaned regularly.

When is it safe to return livestock, companion animals, or fish to the water source?

The only way to ensure the safety of the water for the return of livestock or companion animals after a cyanobacteria bloom is to have a cyanotoxin test performed and return negative results, or to use a 4 ppm potassium permanganate treatment (broken into two separate treatments, 2 to 6 hours apart if fish are present) to oxidize the toxin. The Texas A&M AgriLife Extension's [Aquatic Diagnostics Laboratory](#) can perform water testing for 11 common hepatotoxins in water sources. Cell counts are not useful in determining the safety of water for the return of livestock or companion animals, as there may be little to no cyanobacteria cells in a sample, yet the toxin may remain in the water. If a cyanotoxin test is not performed and a negative result obtained, or if potassium permanganate is not applied to oxidize the toxin, the general recommendation is to wait a minimum of 5 weeks after an algaecide treatment before returning livestock or companion animals to the water source or restocking fish.

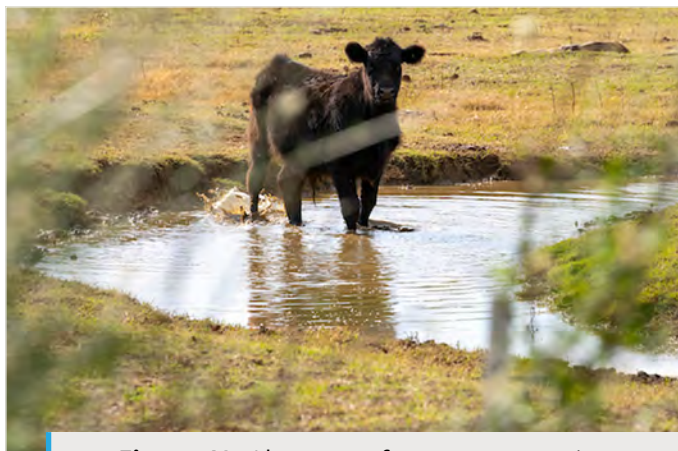


Figure 12. Always perform a cyanotoxin test to ensure it is safe to return livestock to an area where a suspected or confirmed cyanotoxin bloom has occurred. Cyanotoxins are persistent in the environment and can remain at toxic levels for weeks after a bloom has been treated. The Texas A&M AgriLife Extension's Aquatic Diagnostics Laboratory can perform water testing for 11 common hepatotoxins in water sources.

Conclusion

Cyanobacteria and cyanotoxin poisonings can be scary concepts to consider. However, by educating oneself of the signs and risks of a cyanobacteria bloom, knowing the proper detection diagnostics, including how to use cell counts versus cyanotoxin testing, understanding proper prevention, mitigation, and treatment of cyanobacteria blooms, and most importantly, quickly restricting access of animals to suspected water sources can prevent disaster from happening to companion animals or livestock. There is still a significant amount of new information being generated each year about cyanotoxins and related long-lasting health effects in humans and animals at both lethal and sub-lethal concentrations. The fact remains clear that the number of HABs, and especially toxic cyanobacteria blooms, has been on

the rise for the last decade, so evidence points to the issue of cyanotoxin poisoning becoming much larger in the future. Becoming educated on the topic and knowing the signs, combined with effective preventative management practices, is the best way to avoid an issue for livestock or companion animals. Cyanobacteria have been on this planet for billions of years, and they are not going away anytime soon, but they do not have to be the boogeyman hiding in a pond if one is educated on the issue and acts fast when a suspected cyanobacteria issue arises.

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