Chapter 36

Algal Toxins

Synonyms

Red tide toxins, phycotoxins

Periodic blooms of algae, including true algae, dinoflagellates, and cyanobacteria or blue-green algae have been reported in marine and freshwater bodies throughout the world. Although many blooms are merely an aesthetic nuisance, some species of algae produce toxins that kill fish, shellfish, humans, livestock and wildlife. Pigmented blooms of toxin-producing marine algae are often referred to as "red tides" (Fig. 36.1). Proliferations of freshwater toxin-producing cyanobacteria are simply called "cyanobacterial blooms" or "toxic algal blooms." Cyanobacterial blooms initially appear green and may later turn blue, sometimes forming a "scum" in the water (Fig. 36.2).

Although algal blooms historically have been considered a natural phenomenon, the frequency of occurrence of harmful algae appears to have increased in recent years. Agricultural runoff and other pollutants of freshwater and marine wetlands and water bodies have resulted in increased nutrient loading of phosphorus and nitrogen, thus providing conditions favorable to the growth of potentially toxic algae. The detrimental impact of red tides and cyanobacterial blooms on wetland, shore, and pelagic species has long been suspected but not often been substantiated because information on the effects of these toxins in fish and wildlife species is lacking and diagnostic tools are limited.

Photo by Peter Frank, Scripps Institute of Oceanography

Figure 36.1 *Aerial view of a large dinoflagellate bloom in near-shore ocean waters. The organism responsible for this bloom is not a toxin producer; however, toxic blooms may have a similar appearance.*

Cause

Some dinoflagellates and cyanobacteria produce toxins that can affect domestic animals and humans. Some of these toxins such as domoic acid, saxitoxin (paralytic shellfish poisoning or PSP toxin), brevetoxin, and cyanobacterial toxins (including anatoxins, microcystins, and nodularins) have been suspected, but they have rarely been documented, as the cause of bird mortality (Table 36.1). Marine algal toxins such as domoic acid, saxitoxin, and brevetoxin that bioaccumulate or are magnified in the food chain by fish and shellfish, and anatoxins from freshwater cyanobacteria, affect the nervous system; cyanobacteria that contain microcystins or nodularin cause liver damage.

The effects of some harmful algae are not related to toxin production but rather are related to depleted dissolved-oxygen concentrations in water caused by algal proliferation, death, and decay, or night respiration. Other harmful effects include occlusion of sunlight by large numbers of algae and physical damage to the gills of fish caused by the structure of some algal organisms. All of these effects can

Photo by Wayne Carmichael, Wright State University

Figure 36.2 *A cyanobacterial or blue-green algal bloom.*

Table 36.1 *Documented instances of wild bird mortality caused by algal toxins.*

Toxin	Algal species	Toxin type(s)	Migratory bird species affected	Route of exposure
Cyanobacterial	***Microcystis*** sp., ***Anabaena*** sp., ***Aphanizomenon*** sp., ***Nodularia*** sp., and ***Oscillatoria*** sp.	Hepatotoxins (microcystins and nodularin) Neurotoxins (anatoxin-a and anatoxin-a(s))	Unidentified ducks, geese, and songbirds, Franklin's gull, American coot, mallard, American wigeon	Oral (water)
Domoic acid (amnesic shellfish poisoning)	***Pseudonitzschia*** sp.	Neurotoxin	Brown pelican, Brandt's cormorant	Oral (food items)
Saxitoxin (paralytic shellfish poisoning)	***Alexandrium*** sp.	Neurotoxin	Shag, northern fulmar, great cormorant, herring gull, common tern, common murre, Pacific loon, and sooty shearwater	Oral (food items)
Brevetoxin	***Gymnodinium*** sp.	Neurotoxin	Lesser scaup	Oral (food items)

lead to mortality of aquatic invertebrates, aquatic plants, or fish and may produce an environment conducive to botulism. Other marine algal toxins (okadaic acid, neosaxitoxin, ciguatoxin, and *Pfiesteria* exotoxin) and cyanobacterial toxins (saxitoxin, neosaxitoxin, and cylindrospermopsin) have not yet been identified as causes of bird mortality events, but increased awareness and further research may establish a relationship.

Species Affected

Many bird and mammal species can be affected by algal toxins. Most reports of mortality in birds are of die-offs that occur in conjunction with a bloom. Sometimes algal toxins are found in potential food items; however, there have been very few instances in which the algal toxin has been isolated from the ingesta or tissues of affected birds. Domoic acid poisoning caused mortality in brown pelicans and Brandt's cormorants on the central California coast. Brevetoxin has been suspected as the cause of mortality in lesser scaup, and saxitoxin has been strongly suspected as the cause of mortality in sea birds (common terns, shags, great cormorants, northern fulmars, herring gulls, common murres, Pacific loons, sooty shearwaters, and others). Cyanobacterial toxicosis has been suspected in mortalities of free-ranging ducks, geese, eared grebes, gulls, and songbirds.

Distribution

Many of the organisms responsible for red tides are widely distributed and, in recent years, the organisms seem to be markedly spreading. Natural events such as hurricanes can disperse organisms, and it is suspected that some organisms may be transported long distances in ship ballast waters. Another factor that may encourage algal proliferation in both marine and freshwater systems is increased nutrient loading. Certain algae occur more commonly in some areas than others and it is useful to know which ones are problems in specific locations. Good sources of information about algal blooms are the State public health department or the State division of marine resources or marine fisheries.

Seasonality

There have not been enough confirmed instances of wild bird mortality caused by red tides and cyanobacterial blooms to establish seasonal patterns of occurrence.

Field Signs

Field signs reported are variable and they depend on the toxin involved. Domoic acid poisoning of brown pelicans caused neurologic signs that included muscle tremors, a characteristic side-to-side head movement, pouch scratching, awkward flight, toe clenching, twisting of the head over the back, vomiting, and loss of the righting reflex just before death. Brandt's cormorants that also were involved in this mortality event were easily approached and handled, but they did not exhibit the neurologic signs seen in the pelicans. Sea birds suspected of having been poisoned by saxitoxin exhibited paralysis and vomiting. Clinical signs observed in lesser scaup suspected of having been poisoned by brevetoxin included lethargy, weakness, reluctance or inability to fly, head droop, and excessive ocular, nasal, and oral discharge.

White Pekin ducklings that were experimentally exposed to brevetoxins exhibited lethargy, loss of muscle coordination or ataxia, spastic head movements, head droop to one side, and leg extension to the rear during rest. Clinical signs in muscovy ducks dosed with anatoxin-a(s) included excessive salivation, regurgitation of algae, diarrhea, tremors, reduced responsiveness and activity, incoordination, difficulty breathing, excessive thirst, congestion in foot webs, wing and leg weakness, and recumbency and intermittent seizures prior to death.

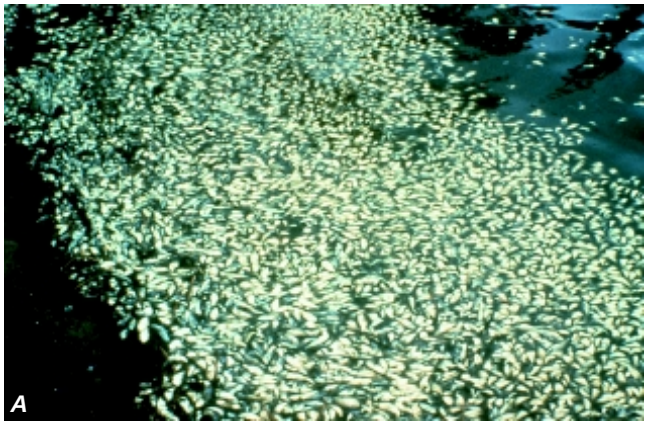
Gross Lesions

No characteristic or diagnostic gross lesions have been described for most types of algal toxin poisonings of wild birds. Many of the toxins, particularly the neurotoxins, have a chemical effect that does not produce a grossly observable lesion. Birds that ingest toxic blooms of *Microcystis* may have notable lesions of necrosis or tissue death and hemorrhage in the liver. These lesions have been reported in domestic mammals and birds, including ducks, that died as a result of exposure to a toxic *Microcystis* algal bloom or that were experimentally dosed with microcystin.

Diagnosis

Definitive diagnosis of algal toxicosis is difficult. Circumstantial evidence, such as the occurrence of a marine red tide or freshwater cyanobacterial bloom in conjunction with a die-off, and supportive clinical and pathologic findings, such as a lack of evidence of the presence of other types of toxins or infectious disease, are often used to reach a presumptive diagnosis. Analysis of the upper gastrointestinal tract contents or tissues of affected birds for algal toxins is possible but the tests are not yet widely available. In addition, there are no established toxic thresholds for wildlife species. Even when levels of particular toxins can be measured it may be difficult to assess their significance. Recently developed methods permit detection of microcystins in animal tissues and gastrointestinal contents by using enzyme linked immunosorbent assay (ELISA) technologies. Also, it is now possible to detect saxitoxin in urine and blood samples from affected animals by using highly sensitive neuroreceptor assays.

A sample of organisms from the bloom may be useful or necessary for diagnosis. Because of the ephemeral nature of blooms, collect algal samples during the die-off event as soon as possible after carcasses are found. Contact a diagnostic laboratory for advice on appropriate sample collection.



Photos by JoAnn Burkholder, North Carolina State University

Figure 36.3 (A and B) *These fish were killed by* ***Pfiesteria*** *sp., an organism that has caused neurological problems, including prolonged amnesia, in people exposed by aerosols in a laboratory. This organism has only been fully described recently, and it has not been reported to cause mortality in birds; however, it may be encountered by biologists investigating concurrent bird and fish kills.*

Control

Because it is difficult to identify algal toxins as the cause of wildlife mortalities, there has been little opportunity to consider control measures. Currently, there is much interest in algal toxins and their threat to human water and food supplies. Identification of the conditions that trigger harmful algal blooms may aid in developing strategies to prevent red tides or freshwater cyanobacterial blooms and associated wildlife mortality. Controlling nutrient loading through reduced fertilizer use, improved animal waste control, and improved sewage treatment may reduce the number, or likely locations, of toxic algal blooms. Careful monitoring and early detection of potentially toxic algal blooms could allow time to initiate actions to prevent or reduce bird mortality.

Human Health Considerations

Most red tide and toxic freshwater cyanobacteria are not harmful unless they are ingested. However, some organisms irritate the skin and others release toxic compounds into the water and, if aerosolized by wave action, these compounds may cause problems when people inhale them (Fig. 36.3). When investigating wildlife mortality that is occurring in conjunction with a known red tide or cyanobacterial bloom, contact the local public health department or a diagnostic laboratory for information on precautions you may need to take. As in the investigation of all wildlife mortality events, wear rubber or latex gloves when handling carcasses.

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Supplementary Reading

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