DEADLY DIATOMS: THE LATEST ON HARMFUL ALGAL BLOOMS

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BIOTOXINS AND ALGAL TOXINS:

Biotoxins are poisons that are produced by cells of living organisms. A well-known example is the suite of toxins (types A to G) produced by the anaerobic bacterium *Clostridium botulinum*, one of the most toxic substances known. Outbreaks of avian botulism, most often caused by type C toxin, have occurred in North America since the early 1900's and are responsible for killing millions of birds. Mycotoxins like aflatoxin and fusariotoxin, produced by fungi, are other examples of biotoxins. Of all of the known biotoxins, we probably understand the least about harmful algal blooms (HABs), yet the marine waters of the US and many countries around the world are increasingly impacted by HABs. Increased efforts over the past decade have lead to great advances in our understanding of HABs and how they impact wildlife health. Most naturally occurring marine biotoxins that impact wildlife health also impact human health. Consequently the study of biotoxins in wildlife also is helping to improve and protect human health.[1] Four of the more common culprits are examined (Table 1), all of which are passed up through the food chain and bioaccumulate in prey species that are then ingested by marine birds and mammals.

BREVETOXIN

Brevetoxins are lipid soluble neurotoxic ethers and nine brevetoxins have been isolated from the dinoflagellate *Karenia brevis*, all with varying degrees of toxicity. The mechanism of toxin action is sodium channel activation with toxin binding to site 5 on the voltage gated sodium channel, resulting in channel opening at normal resting potential and prolongation of their open state, leading to uncontrolled Na^+ influx into the cell. Clinical signs can range from acute death to severe cerebellar ataxia as documented in Double-crested Cormorants (*Phalocrocorax auritis*). [2]

Karenia brevis blooms occur in the Atlantic Ocean off of the southeastern United States where they tend to be seasonal, beginning in late summer and early fall and lasting into January or beyond. In addition to causing human health problems, blooms have been documented to cause morbidity and mortality in Bottlenose dolphins (*Tursiops truncatus*), Manatees (*Trichechus manatus latirostris*), Double-crested Cormorants, Lesser Scaup (*Aythya affinis*), Red-breasted Mergansers (Mergus merganser), and multiple sea turtle and fish species.[3]

In Florida, documented brevetoxin-caused mass mortality events killed Manatees in 1996, 2002, 2003, and 2005 and Bottlenose dolphins in 1999–2000, 2004, and 2005–2006.[3] Some mortality events occurred at the same time as *Karenia brevis* blooms while in other cases there was a lag between the *K. brevis* bloom and detected mortality or in one case, no bloom was ever detected. Evidence suggests that the route of lethal exposure in both species seems to be ingestion and the concurrent or delayed mortality is related to the complex patterns of trophic transfer where the toxin moves through various invertebrate and vertebrate hosts before being ingested by Manatees or Dolphins. Consequently, the occurrence large brevetoxin mortality events in Manatees and Dolphins depends on a multitude of factors, including where and when a bloom occurs, how long it persists and what route the toxins travel in the food chain.

DOMOIC ACID:

The neurotoxin domoic acid (DA) is produced by diatoms in the genus *Pseudo-nitzschia* and has a high affinity for glutamate receptors. In humans, it causes loss of short-term memory and is called amnesic shellfish poisoning. The disease in marine mammals has been best studied in California sea lions (*Zalophus californianus*) where there are two distinct clinical syndromes: acute domoic acid toxicosis and a more chronic disease characterized by epilepsy.[4] Acutely, the neurotoxin causes degeneration and neuronal necrosis within the neuropil of the hippocampus, amygdala, pyriform lobe and other limbic structures. Clinically signs include ataxia, head weaving, seizures or coma in acute cases. Severity ranges, but clinical signs usually last about a week followed by recovery, if treated, or death. Chronically the toxin causes mild nonsuppurative inflammation and loss of laminar organization in affected areas. Clinical signs in chronic cases include seizures, periods of marked lethargy and inappetence, vomiting, muscular twitching, central blindness blepharospasm and abnormal behavior. Despite treatment with diazepam, lorazepam and phenobarbital, seizures often increase in frequency and severity, resulting in status epilepticus and spontaneous death in some animals, or euthanasia due to poor prognosis for release in others. In addition to neurologic signs, a degenerative cardiomyopathy and reproductive failure (through mortality of pregnant females, abortion and premature parturition of pups) also can

be associated with DA exposure in sea lions. Like brevetoxins, DA is transmitted to marine birds and mammals via trophic transfer of the toxin, often by planktivorous fish like Northern Anchovies (*Engraulis mordax*). Interestingly, it has been proposed that at times, sea lions stranding with DA toxicity might be a more sensitive and reliable indicator of the presence of domoic acid-producing *Pseudo-nitzschia* than traditional water testing.

Other marine mammals that have been impacted by DA include southern sea otters (*Enhydra lutris nereis*), long-beaked common dolphins (*Delphinus capensis*) and gray whales (*Eschrichtius robustus*). Domoic acid has been detected in marine mammals such as Northern right whales (*Eubalaena glacialis*), pygmy *(Kogia breviceps)* and dwarf *(Kogia sima)* sperm whale, humpback whales (*Megaptera novaeangliae*) and blue whales (*Balaenoptera musculus*) confirming exposure but not morbidity or mortality. Acute DA poisoning also has killed large numbers of Brown Pelicans (*Pelecanus occidentalis*), Brandt's cormorants (*Phalacrocorax penicillatus*) and Marbled Murrlets (*Brachyramphus marmoratus*) in California.

SAXITOXIN

Saxitoxins are potent neurotoxins produced by multiple dinoflagellates including approximately 11 species of the genus *Alexandrium* as well as by *Gymnodinium catenatum* and *Pyrodinium bahamense* var. *compressum* and several cyanobacteria, however the majority of mortality reports in wildlife have been associated with *A. tamarense* and *A. catenela*. Mass mortality of marine birds and mammals due to saxitoxin have been reported sporadically and in most cases, picivorous birds were affected after consuming fish contaminated by saxitoxin.[5]

In 1942, over 2,000 dead sea birds, including at least 8 species, stranded on the coast of Washington State coincident with an *Alexandrium catenella* bloom. Six human cases of paralytic shellfish poisoning also were reported. A similar event was reported near the Farne Islands in northeast England in 1968 when 636 seabirds were found dead, including over 80% of the local breeding Shag (*Phalacrocorax aristotelis*) population. Clinical signs exhibited prior to death included loss of equilibrium, paralysis, pupilary constriction and vomiting. Gross lesions included intestinal hemorrhage and organ congestion. Concurrently, 78 cases of paralytic shellfish poisoning occurred in humans from eating toxin mussels. A 1972 epornitic from Maine to Massachusetts killed over 1600 black ducks (*Anas rubripes*) and another 620 birds (representing 13 species). In 1987 saxitoxin was believed to kill 14 humpback whales in Cape Cod Bay, Massachesetts. The animals all were in good condition but had been exposed to saxitoxin by consuming Atlantic Mackerel (*Scomber scombrus*) that contained saxitoxin in their viscera demonstrating the transfer of this toxin through fish. In 1997, the death of over 117 Mediterranean Monk seals (*Monachus monachus*) that died along the coast of West Africa was connected to saxitoxin ingestion and a concomitant bloom of *Alexandrium minutum* and *Gymnodinium catenatum*, however in this epizootic, the role of morbillivirus couldn't be ruled out.

AKASHIWO SANGUINEA

In November and December of 2007, a bloom of the surfactant-producing dinoflagellate *Akashiwo sanguinea* caused widespread seabird mortality in Monterey Bay, California.[6] Over 700 live-stranded or freshly dead marine birds were collected. While most were Northern Fulmars (*Fulmarus glacialis*), 13 other species also were found including Surf Scoters (*Melanitta perspicillata*), Pacific and Red-throated Loons (*Gavia* spp.), and Clark's and Western Grebes (*Aechmorphorus* spp.). All birds found had variable patches of a slimy pale yellow-green material on their feathers.

It was discovered that the material fouling the bird's feathers was proteinaceous foam derived from the cellular breakdown of the dinoflagellate *Akashiwo sanguinea*. The foam coated bird's feathers and destroyed their waterproofing ability in a manner similar to fouling by petroleum oil, which lead to hypothermia, hypoglycemia and even death. Other than muscle palor (due to anemia), atrophy of the pectoral muscles and lack of significant subcutaneous adipose tissue there were no common lesions. A few birds showed gross or microscopic evidence of acute hemorrhage into the lungs and patchy fibrin deposition in air sacs that was thought to be consistent with oxidative damage to respiratory epithelium, similar to but less severe than that reported from birds exposed to aerosolized products from overheated polytetrafluorethylene (Teflon)-coated pans. It was hypothesized that this could have been due to transient exposure to an aerosolized component of the surface slime. Live-stranded birds responded well to rinsing, rehydration, warming and nutritional supplementation using standard treatment protocols developed for rehabilitating birds oiled with petroleum products. Most birds regained body mass within a week or two and were fit for release suggesting that the protein fouling their feathers was non-toxic or minimally toxic.

The frequency, amplitude and duration of HABs had increased substantially within Monterey Bay since 2004 and continue to increase globally. Jessup and others hypothesized that the harmful effects of dinoflagellate blooms could become more common. Interestingly, an *Akashiwo sanguinea* bloom caused another larger marine bird mortality event in Oregon and Washington in 2009.[7] Hundreds of birds were found stranded with similar presenting conditions to those reported in the Monterey Bay event of 2007. Instead of

Northern Fulmars, Red-throated Loons (*Gavia immer*), Western Grebes (*Aechmophorus occidentalis*), and Common Murres (*Uria aalge*) represented the greatest proportion of examined birds, likely reflecting a difference in species present during the time of the bloom.

Algal species	Toxin	Human Disease	Wildlife species affected
Akashiwo sanguinea	Surfactant	None known	Multiple seabird species
Alexandrium spp.	Saxitoxin	Paralytic shellfish poisoning	Multiple seabird species, Humpback whales, Mediterranean Monk seals
Karenia brevis	Brevetoxins	Neurotoxic shellfish poisoning	Bottlenose dolphins, Florida manatees and multiple bird, sea turtle and fish species
Pseudo-nitzschia spp.	Domoic acid	Amnesic shellfish poisoning	California sea lions, Gray whales, Long-beaked common dolphins, Southern sea otters, Brand's cormorants, Brown pelicans and Marbled Murrlets

Table 1: Examples of marine algal species documented to cause mortality in wildlife

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