

Avian Vacuolar Myelinopathy (AVM)

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Overview

Avian Vacuolar Myelinopathy (AVM) was first documented in bald eagles (*Haliaeetus leucocephalus*) and American coots (*Fulica americana*) at DeGray Lake, Arkansas in 1994. Since then, the disease has been documented in freshwater bodies across the southeastern United States. Clinical signs of AVM in birds include a general lack of coordination while walking, swimming and flying, and the inability to remain upright in the water (Figure 1). This often-fatal disease is most frequently observed during the fall and winter months on or near water bodies with invasive aquatic vegetation. Affected birds are prone to predation, drowning, starvation and injury.



Figure 1. American coots with AVM have difficulty swimming and flying.

AVM diagnosis

The only way to confirm AVM in birds is by histological examination of the brain tissue in dead birds. The characteristic vacuolar lesions appear as open spaces in the brain (Figure 2). They vary in severity and location, but are most frequently observed in the optic lobe and cerebellum. The lesions develop due to fluid build-up between the layers of the myelin sheath surrounding nerve cells. This is the “white matter”; a part of the brain made of axons that connect cells so nerves can communicate.

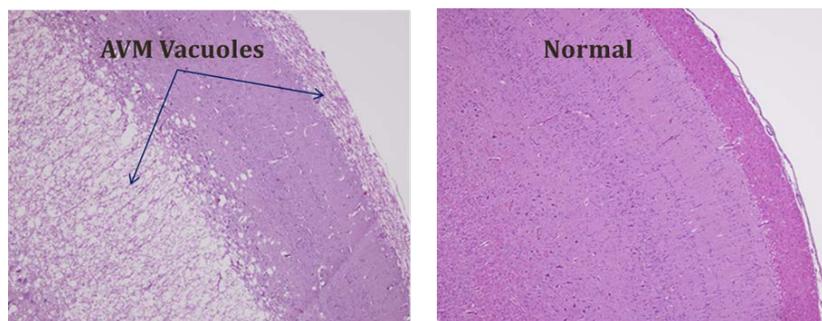


Figure 2. AVM vacuoles form within the optic lobe of the brain of affected birds (left) in contrast to normal brain tissue (right).

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Cause of disease

Although many aspects of the disease process are unknown, research studies established a link with invasive aquatic vegetation colonized by an epiphytic cyanobacterium (blue-green algae; Figure 3). Based on morphology and genetic information, this cyanobacterium was determined to be a new genus and species, and named *Aetokthonos hydrillicola* (eagle-killer living on hydrilla). Field and laboratory studies demonstrated that ingestion of aquatic vegetation, with *A. hydrillicola* growing on it, produces the characteristic clinical symptoms and brain lesions. Many species of cyanobacteria produce toxins that cause disease in wildlife and humans, including neurological impairment. Studies are currently underway to isolate and characterize this novel toxin.

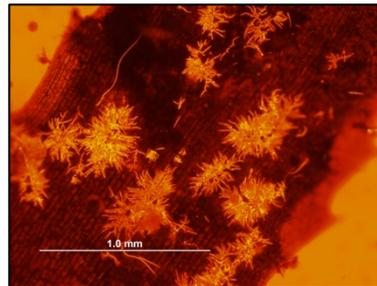
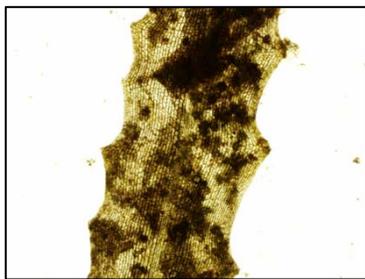


Figure 3. Hydrilla leaf with epiphytic *Aetokthonos hydrillicola* under light microscopy (left) and fluorescent microscopy (right).

AVM Locations

More than 70 eagle deaths were attributed to AVM in Arkansas reservoirs from 1994-1996. From 1998-2014, additional eagle deaths and waterbirds with the characteristic vacuolar AVM lesions (AVM+) have been verified in 20 reservoirs from Texas to North Carolina, and central Florida (Figure 4.)

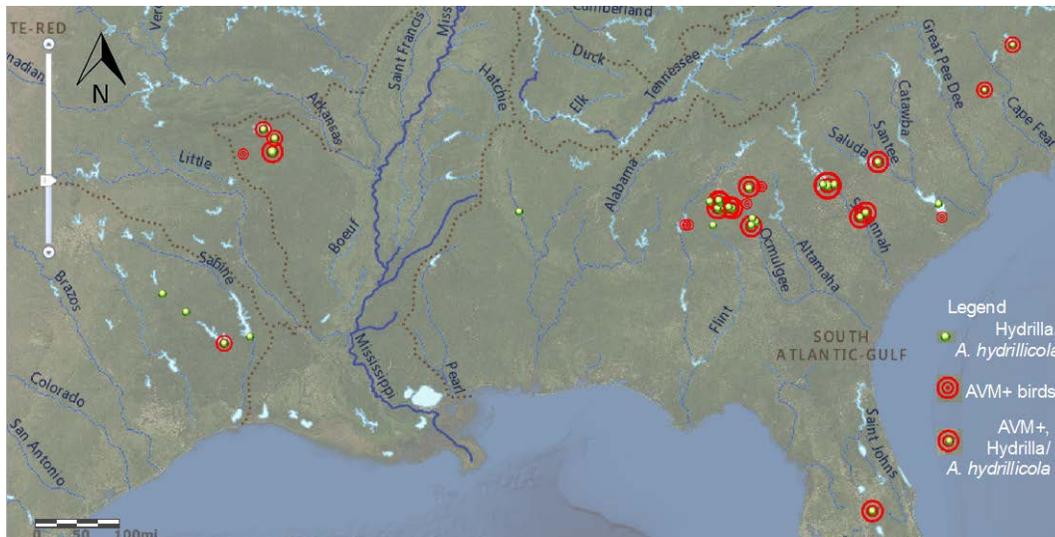


Figure 4. Reservoirs where birds with vacuolar lesions have been confirmed (AVM+ birds), An additional 30 sites with recent hydrilla/*Aetokthonos hydrillicola* infestations pose AVM risks to wildlife.

How is AVM spread?

Research with AVM-symptomatic birds confirms that the disease is not contagious, nor does the toxin bioaccumulate in animal tissues. AVM disease is acquired directly by ingestion of aquatic vegetation with the *A. hydrillicola* growing on it. The vegetation and cyanobacterium can be transferred to new locations by people, downstream transport, and wildlife (Figure 5). Neurologically impaired coots and waterfowl make easy targets for birds of prey. Predators and scavengers are likely exposed to the AVM toxin through ingestion of the gut contents of prey (Figure 6).



Figure 5. Clumps of the invasive plant hydrilla on a boat trailer from an AVM positive reservoir.



Figure 6. Hydrilla (A) covered with neurotoxic cyanobacteria, *Aetokthonos hydrillicola* (B) is readily consumed by coots (C) that become easy prey and a source of mortality for wintering and nesting eagles on these AVM reservoirs (D).

Treatment

There is no known cure for AVM at this time. All symptomatic eagles have died within days after capture. Veterinary care given to impaired coots that were removed from an AVM reservoir resulted in reduced neurological impairment, but the brain lesions persisted.

AVM Risk to other wildlife

Vacuolar lesions were documented in fish and aquatic turtles fed hydrilla collected from known AVM sites. It is currently unknown whether mammals are susceptible to AVM. Impaired and dead beavers have been found during the fall-winter on AVM sites, but test results were inconclusive. Pigs and mice that were fed hydrilla from an AVM site did not become impaired or develop brain lesions. Herbivorous fish did develop vacuolar brain lesions after consuming hydrilla/*A. hydrillicola*, but had no detectable neurological impairment. Laboratory trials confirmed birds of prey developed AVM brain lesions after



Figure 7. Red fox on coot bait at camera trap.

consuming the gut contents from coots that were collected from an AVM site. A recent camera trap study that was designed to photograph additional wildlife species that might consume coots at an AVM site documented raccoons, fox, crows and hawks feeding on dead coots (Figure 7).

Control Options

There are several management strategies currently being tested to control invasive aquatic plants associated with AVM. Aquatic herbicides are being investigated as possible solutions, but they are neither cost effective nor feasible on large lakes. Sterile, triploid grass carp were evaluated as a management option, since they have been an effective biological control method for unwanted aquatic vegetation. They were effective at eliminating hydrilla at an AVM reservoir and the AVM toxin did not transfer through these herbivorous fish to birds in a feeding trial. Implementation of this effective long-term control method for hydrilla is still problematic because of the time required to reduce hydrilla density initially and potential effects on beneficial aquatic plants.

Potential Impact

Hydrilla continues to spread (31 States in 2014) and new AVM sites continue to be identified annually. With every new AVM positive location, more avian deaths are documented and the potential for other species to be affected by the disease increases as well. AVM research is a great example of a cutting edge interdisciplinary collaboration to solve issues involving human-wildlife-disease interactions. The ongoing investigations incorporate aspects of aquatic ecology and wildlife disease, as well as human dimensions and how they factor into the spread of the disease. This ongoing research is critical to updating current reservoir management strategies for the benefit of wildlife and people.

Can I still eat waterfowl and fish from these AVM lakes?

Based on current findings, there is no direct evidence that human consumption of game meat collected from AVM positive lakes is a health concern. The Southeastern Cooperative Wildlife Disease Study, University of Georgia, advises, *“AVM is not believed to affect mammals, but consumption of any animals displaying abnormal behavior or other signs of disease is not advisable.”*

How can I help reduce the spread of AVM?

Take the time to thoroughly wash vehicles, equipment, boats, and trailers after spending time on the water to reduce the likelihood of transferring aquatic plants and animals. Report unusual behavior, sick, and dead wildlife to the local resource manager or state wildlife agency. Discuss AVM with fellow outdoor enthusiasts and get the word out on invasive plant containment procedures for boating equipment.

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Editor

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Further reading

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