Cyanobacterial poisoning in livestock, wild animals and birds – an overview

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Abstract

Toxic cyanobacterial poisoning of livestock was first reported in the scientific literature in 1878, when sheep, horses, dogs and pigs were seen to die within hours of drinking from an Australian estuarine lake affected by a bloom of the brackish water cyanobacterium *Nodularia*. Throughout the 20th century, cyanobacteria-related poisonings of wild and stock animals and birds have been described on all continents. Some mass mortality events involving unrelated fauna in prehistoric times have also been attributed to cyanotoxin poisoning; if correct, this serves as a reminder that toxic cyanobacteria blooms predate anthropogenic manipulation of the environment, though there is probably general agreement that human intervention has led to increases in the frequency and extent of cyanobacteria blooms. Many of the early descriptions of cyanobacteria poisoning were anecdotal and circumstantial, though the sheer number of such reports lends weight to the suspicion that the observations were often accurate. Early necropsy findings of hepatotoxicity were subsequently confirmed by experimental investigations. More recent reports supplement clinical and post-mortem findings with investigative chemistry techniques to identify cyanotoxins in stomach contents and tissue fluids.

Reports of cyanotoxin poisoning can be dramatic in terms of the number of animals affected or the rapid progression of illness and death, with mass events involving thousands of animals, and large animals succumbing within minutes of exposure. The exposure route for wild and domestic animal poisonings is presumably exclusively via oral consumption of cyanotoxin-affected water or filamentous benthic cyanobacteria.

The diagnosis of acute intoxication and mortality due to ingestion of cylindrospermopsin or the hepatotoxic cyanotoxins (microcystins or nodularin) may be assisted by necropsy findings of characteristic pathology, particularly in the liver. Sub-acute intoxication over days or weeks may be more difficult to determine, with concomitant degenerative and reparative changes seen in the liver. Monogastric animals are reportedly less sensitive to microcystins than ruminants. Photosensitisation secondary to acute cholestatic hepatotoxicity may be seen on unpigmented body parts (e.g. nose, tongue, udder). Poisoning by neurotoxic cyanotoxins (anatoxin-a or the saxitoxin group) is determined by the clinical picture, supplemented by specialist laboratory findings of neurotoxins in source water, stomach contents and/or tissues; necropsy findings are unremarkable.

Differential diagnoses, particularly with respect to hepatotoxic forage, should be considered. For example, pyrrolizidine alkaloids are produced by plants in the Compositae, Leguminosae and Boraginaceae families; pyrrolizidine intoxication of livestock occurs on all continents. Clinical signs of acute toxicity include liver failure and ascites; secondary photosensitivity may be seen in cases of chronic poisoning.

Morbidity and mortality resulting from cyanotoxin-affected waters is difficult to predict in the field, as cyanobacteria blooms can be concentrated and moved by the wind, and many other physical and biological parameters that influence toxin release and degradation are in play. Information and education strategies to assist farmers with techniques to mitigate cyanobacteria blooms over the medium to long term and to manage blooms in the short term (e.g. by restricting stock access and providing alternate water supplies) should be maintained and expanded. There may be room for improvement in the reporting and dissemination of information about cyanotoxin-related events, as exemplified by the Animal Health Surveillance Quarterly Report from Australia. A research strategy that remains to be conducted is a comprehensive analysis of the economic cost to farmers and the tourism industry due to cyanobacteria, tourism presumably being adversely affected by reports and sightings of wildlife and pet animal deaths.