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Review article

Acute animal and human poisonings from cyanotoxin exposure — A review of the literature

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ABSTRACT

Cyanobacterial blooms are a potential health hazard due to the ability of some species to produce toxins that are harmful to other living organisms. This review provides a comprehensive summary of anecdotal and case reports on acute poisonings in animals and humans attributable to cyanotoxin exposure in fresh- and brackish-waters. Approximately two-thirds of reported poisonings have occurred in Europe and the United States. Dogs and livestock account for the majority of reported cases involving animal exposure to cyanotoxins, while recreational activities are responsible for approximately half of reported incidents involving human exposure. Due to data limitations it is difficult to estimate the total number of animals and humans affected by cyanotoxins, however, some general observations regarding frequency and numbers affected are made. The review demonstrates that cyanotoxins have, and will likely to continue to have, potentially serious consequences for public health and animal welfare worldwide.

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1. Introduction

Cyanobacteria, also known as blue-green algae, are a common and naturally occurring component of fresh- and brackish- water environments. They are important primary producers and play a key role in ecosystem functioning and biodiversity, providing oxygen for aerobic micro-organisms and converting nitrogen into organic forms. However, under certain environmental conditions – high nutrient loads, low turbidity, warmth and sunlight – cyanobacteria can quickly multiply to form extensive, and often highly visible, blooms. Such blooms affect the colour, odour and taste of the water, creating aesthetic problems and impairing consumptive and recreational use.

Cyanobacterial blooms are also a potential health hazard due to the ability of some species to produce toxins that are harmful to other living organisms, including humans. These 'cyanotoxins' are stored in the cells of the bacteria and are released into the water when the cells rupture or die. Often the release of toxins occurs at the end of the bloom's natural lifecycle; hence, toxins can still be present in the water and pose a health risk even after the bloom has dissipated and is no longer visible.

The toxins produced by cyanobacteria vary in their toxicology and are typically classified according to the systems and organs they target in terrestrial vertebrates. Neurotoxins affect the nervous system, while hepatotoxins target the liver. At sufficiently high concentrations, these toxins can result in death, by respiratory arrest and liver failure respectively, in a few minutes to a few hours. Chronic exposure to low doses of some hepatotoxins has also been associated with tumour promotion (Chen et al., 2009; Fleming et al., 2002; Li et al., 2011; Lun et al., 2002;

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Ueno et al., 1996; Yu et al., 1989) and there is evidence to suggest that β-methylamino-L-alanine (BMAA), a neurotoxin produced by a variety of cyanobacteria species, may be a contributing factor in the development of neurodegenerative diseases (Cox et al., 2003; Holtcamp, 2012; Metcalf et al., 2008). Other types of toxins produced by cyanobacteria include cytotoxins, dermatotoxins and lipopolysaccharide endotoxins.

Exposure to cyanotoxins can occur either directly or indirectly. Direct exposure routes include ingestion through drinking water, dermal contact whilst bathing or swimming, inhalation of aerosolised particles whilst showering or participating in water sports, and intravenously through medical procedures. Indirect exposure can occur via the consumption of animal or plant products that have been exposed to cyanotoxins. Research has also shown that cyanotoxins can bioaccumulate such that their toxic effects may be magnified in food chains (Ettoumi et al., 2011).

Reports of animal poisonings attributable to cyanotoxins have been documented worldwide for more than a century. Over this period, a diverse range of animals have been affected – from dogs, cattle and fish, to flamingos, bats and bees. Adverse health effects experienced by humans following exposure to cyanotoxins have also been reported.

In this review I provide a comprehensive summary of anecdotal and case reports on acute animal and human illnesses attributable to cyanotoxin exposure in fresh- and brackish-water environments. I update and expand upon previous collations by Schwimmer and Schwimmer (1964); Schwimmer and Schwimmer (1968); Carmichael (1992); Landsberg (2002) and Stewart et al. (2006b), documenting more than twice the number of discrete poisoning incidents reported in these previous reviews.

Of the hundreds of reported incidents of cyanotoxin exposure, many are circumstantial, noting only a temporal and spatial association between the presence of a cyanobacterial bloom and unexplained, adverse health effects in animals or humans. Others are very detailed, outlining a full history of the incident and details of medical and diagnostic investigations. In this review, incidents are included where there is evidence to suggest that cyanobacteria may have been the causative agent or a contributing factor.

Unlike previous reviews, where only mortalities have been recorded for animal poisonings, in this review morbidity is also included. The rationale for this is that animals carry a value based on their contribution to productivity (e.g. livestock), enhancement of human wellbeing (e.g. companion animals) or biodiversity. Illness affects their ability to carry out these roles and, in some instances, could be just as, if not more, costly than mortality due to lost productive capacity or veterinary costs.

In reviewing these incidents, I show that toxins produced by cyanobacteria have, and will likely continue to have, potentially serious consequences for public health and animal welfare worldwide.

2. The adverse health effects of cyanotoxins

The potential toxicity of cyanobacterial blooms was initially brought to the attention of the scientific community through reports of animal poisonings by farmers and veterinarians. In 1878, Francis (1878) described sudden livestock and domestic dog deaths in South Australia, noting that they occurred following the ingestion of water from a lake containing a cyanobacterial bloom. Toxins produced by cyanobacteria were determined to be the cause of death following an experiment which involved dosing a sheep with cyanobacterial scum from the lake and observing its effects. The dose given to the sheep proved to be fatal, with post-mortem findings similar to those observed in other affected animals.

Reports of further poisonings, mostly livestock fatalities, followed from the United States (Arthur, 1887a; Arthur, 1887b; Cotton, 1914; Fitch et al., 1934; Nelson, 1903; Porter, 1887; Stalker, 1887) and Canada (Gilliam, 1925; Howard and Berry, 1933). These studies were mostly field reports giving an overview of the animals affected, poisoning signs, and the results of post-mortem examinations and pathology tests. In most cases, the ingestion of toxins produced by cyanobacteria were suspected, but not confirmed, as the cause of illness or death. The inability to determine the exact cause was due to a variety of reasons, including the absence of a visible bloom at the time the animals were observed to be affected, the failure to collect or a delay in collecting appropriate specimens for analysis, and a lack of awareness of cyanobacteria as a potential source of toxins. A specific diagnosis was further hindered by the inability at the time to detect and identify the toxins produced by cyanobacteria in water or tissue samples.

Since these initial reports, hundreds of cases of animal poisonings, suspected or confirmed, have been documented in the literature. Incidents involving livestock, dogs, birds and fish consistently feature, but other reports highlight the diverse range of species that can potentially be affected, and how pervasive the incidents are globally. For example, incidents have been reported involving bees in Australia (May and McBarron, 1973), giraffes in South Africa (Harding and Paxton, 2001), bats in Canada (Pybus et al., 1986), flamingos in Spain (Alonso-Andicoberry et al., 2002), yaks in Bhutan (Dahal, 2000), deer in Norway (Handeland and Østensvik, 2010) and sea otters in the United States (Miller et al., 2010).

In reviewing these incidents, it is evident that the toxins produced by cyanobacteria can act rapidly, sometimes within minutes, and in most cases they proved to be fatal for the animals affected. This rapidity and severity arises because cyanotoxins are some of the most potent toxins known. For example, the LD_{50} dose[1] for microcystin-LR is 50 μg/kg, compared 500 μg/kg for strychnine and 10,000 μg/kg for sodium cyanide (Carmichael, 1991). They also have a steep dose-response curve, meaning that signs of poisoning are generally not observable until an animal has been exposed to a near-lethal dose (Carmichael, 1991), and treatment options to date are limited (Roegner et al., 2013; Stewart et al., 2008).

The first notable incident involving humans and exposure to cyanotoxins occurred in 1930–31 in Charleston, West Virginia and other cities along the Ohio River, USA (Miller and Tisdale, 1931; Veldee, 1931). Following reports of heavy cyanobacterial growth in a side branch of the Ohio River, which was the source of the public water supply for the city of Charleston, gastrointestinal symptoms were reported, affecting an estimated 15% (approximately 9000) of the city's population. The same symptoms then began to appear in other cities which sourced public water from the Ohio River. After the cause of the illnesses could not be attributed to other infectious agents it was proposed that toxins produced by cyanobacteria might have been responsible for the epidemic.

Confirmed or suspected illness arising from cyanotoxins in drinking water supplies have since been reported in Zimbabwe (Zilberg, 1966), the Philippines (Dean and Jones, 1972), the United States (Lippy and Erb, 1976), Australia (Bourke et al., 1983; Byth, 1980; Falconer et al., 1983; Hawkins et al., 1985), Brazil (Teixeira et al., 1993) and Sweden (Annadotter et al., 2001). It is worth noting that most of these incidents involved populations receiving water through a treated reticulated supply system and that the release of toxins was often triggered by treatment of a cyanobacterial bloom in the supply reservoir with copper sulphate.

Recreational activities are also a common mode of exposure to cyanotoxins in humans. One of the earliest reported incidents was documented by Heise (1949) in the United States. A patient reported recurring episodes of asthma, conjunctivitis and nasal irritation after swimming in the same lake each summer. Of note was that the patient had swum in a nearby lake and other swimming pools previously without incident, and that the symptoms only presented from mid-August onwards. In 1944, some 'floating green scum' was collected from the shore of the lake and diluted extracts given to the patient produced an immediate skin reaction. Asthmatic symptoms and swelling also

[1] The amount required to kill 50% of the rodent test population in 24 h by intraperitoneal injection.