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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.

Contents

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.

occurred after the patient was given intra-cutaneous injections of the lake extract. The scum was shown to be comprised primarily of cyanobacteria.

Incidents of acute reactions to cyanotoxins following swimming have also been in reported in the United States by Billings (1981) and Carmichael et al. (1985), as well as in Canada (Dillenberg and Dehnel, 1960), Argentina (Pizzolon et al., 1999), the United Kingdom (Turner et al., 1990), the Netherlands (van Hoof, 1994), Australia (Williamson and Corbett, 1993) and Finland (Rapala et al., 2005). Symptoms following water skiing (Carmichael et al., 1985), sailboarding (Pearson et al., 1990), jet skiing (Giannuzzi et al., 2011) and fishing (Soong et al., 1992; Trevino-Garrison et al., 2015) have also been reported.

Incidents of cyanotoxin poisoning from recreational pursuits highlight how exposure routes may operate simultaneously to produce reactions in humans (Codd et al., 1999). For example, army recruits in England presented with a range of symptoms including malaise, sore throat, blistered mouth, dry cough, fever, abdominal pain, vomiting and diarrhoea (Turner et al., 1990). Two were hospitalised with pneumonia. The symptoms began after they had undertaken a canoeing and swimming exercise (including barrel rolls) in a lake containing an extensive cyanobacterial bloom. Bacterial and viral tests for waterborne pathogens were negative but the cyanotoxin microcystin-LR was detected in samples collected from the lake. Although exposure routes were not determined by Turner et al. (1990), Pearson et al. (1990) noted that the identification of a cyanotoxin, combined with the epidemiological and clinical observations, had led investigators of the incident to 'infer that the illness experienced by the soldiers was associated with contact with, and ingestion and/or inhalation of, toxic blue-green algae' (p. 48). Codd et al. (1999) similarly suggested that the incident involved oral and probably inhalation, as well as skin contact, exposure. Giannuzzi et al. (2011) also document an incident in which a jet-skier experienced gastrointestinal symptoms progressing to respiratory distress and liver damage after prolonged exposure to water containing a cyanobacterial bloom. High levels of microcystin-LR were detected in water samples. While the authors did not specify which route(s) of exposure were responsible for the observed symptoms, they did note that 'direct contact with the bloom through immersion, oral ingestion … and inhalation' (p.2169) was reported by the patient.

Unlike animals, death from exposure to cyanotoxins is rare in humans. In 1988, the deaths of 88 people were reported during a severe outbreak of gastroenteritis affecting more than 2000 residents in Bahia, Brazil (Teixeira et al., 1993). No infectious agents were identified, nor were heavy metals or agricultural contaminants found in the water supply. It was however, noted that the timing of the incident coincided with cyanobacterial blooms in the reservoir providing water to the affected population. The authors concluded that toxins produced by cyanobacteria could be a possible explanation for the epidemic. There is also an unconfirmed report of 100 mortalities attributable to cyanotoxin exposure in Lake Embu, Kenya, in 2001 (Codd et al., 2005).

The first confirmed human deaths from cyanotoxins occurred from intravenous exposure in a dialysis clinic in Brazil in 1996 (Azevedo et al., 2002; Carmichael et al., 2001; Dunn, 1996; Jochimsen et al., 1998; Pouria et al., 1998). Patients receiving treatment began to complain of visual disturbances, nausea and vomiting, progressing to acute liver failure which resulted in 76 deaths. Of these, 52 were confirmed to have been exposed to lethal levels of hepatotoxic microcystins. At the time of the incident, water was delivered via truck to the clinic from the city's reservoir, where it was treated using the in-house system. Microcystins were detected in water samples from the city's reservoir, the delivery truck, the water-holding tank and treatment devices at the clinic, as well as in serum and tissue samples taken from affected patients.

More recently, five teenage boys became ill with gastrointestinal symptoms after swimming in an algae-scum covered pond at a golf course in Wisconsin, USA. Approximately 48 h after exposure, one of the boys suffered a seizure and died of heart failure. Stool and blood samples tested positive for cyanobacteria and, despite the unusually long length of time between exposure and death, the coroner concluded that anatoxin-a, a neurotoxin, was most likely the underlying cause of death (Behm, 2003; Weirich and Miller, 2014).

Exposure through contaminated drinking water, recreational activities and haemodialysis account for the majority of human illnesses attributable to cyanotoxins, although several incidents have been documented following exposure through occupational activities such as water testing (New South Weales Blue-Green Algae Task Force, 1992; Pearson et al., 1990). Respiratory irritation has also been reported by home owners after watering lawns with water from a reservoir where a cyanobacterial bloom had recently been treated (Backer, 2002).

3. Literature review

This review presents a comprehensive summary of references of adverse health effects in animals and humans following acute exposure to toxins produced by cyanobacteria. Reviews by Schwimmer and Schwimmer (1964, 1968); Carmichael (1992); Landsberg (2002) and Stewart et al. (2006b) were used as a starting point for identifying discrete incidents. Codd et al. (2005) was also a useful reference for providing a summary of reported cyanotoxin exposures for each continent. Where available, the primary references were sourced and the following details were recorded: when the incident occurred, where it occurred, how many were affected, and the presenting symptoms and health outcomes. The species affected and exposure modes were recorded for animal and human cases respectively. If determined, the dominant species of cyanobacteria present at the time of the incident and the associated toxin were also noted.

Incidents reported in scientific, veterinary and medical journals were included, as well as those appearing in conference proceedings and 'grey literature' (technical reports and government publications). Unless directly referenced in these types of publications, incidents reported in newspapers and online sources were not included in this review. Similarly, publications written in languages other than English were excluded unless they had been referenced in another journal article. References in all publications were examined to identify other potential incidents for inclusion.

Every effort was made to collect detailed information on when, where, who, and how the incident occurred. However, the level of detail available varied significantly. While some reports provided a very detailed account of the incident, other incidents were briefly described using phrases such as "several livestock deaths", "many were affected" or "mass mortality". In some instances a year was not specified, while in others the term "recently" or a period such as "the 1990s" was used. Details were recorded exactly as described in the source material. Where more than one report described a single event, the most comprehensive report(s) detailing the health effects were chosen. It should also be noted that some inconsistencies were found in the literature, mostly relating to the year in which incidents occurred, what type of animals were involved and how many individuals (humans or animals) were affected. Where possible, details were obtained from the primary reference describing the incident and, as such, descriptions in this review may differ from those reported in other reviews and articles.

The results of the review are presented in Appendix A. A summary of the number of incidents occurring from 1800 to 2010 – by location, species affected and exposure mode – is presented in Table 1.

The information gathered in this review demonstrates that cyanobacterial blooms present an ongoing risk to human health and animal welfare worldwide. Every decade since the 1920s has seen incidents of acute cyanotoxin poisonings reported in the literature, with the number of reported incidents for animals and humans combined increasing ten-fold over this period. Of these, animals account for nearly 80% of incidents reported in the literature. By location, approximately

Table 1
Reported incidents of mortality and morbidity attributable to acute exposure to cyanotoxins.

	1800–1900	1901–10	1911–20	1921–30	1931–40	1941–50	1951–60	1961–70	1971–80	1981–90	1991–2000	2001–10	Total
Animals													
Number of incidents	10	–	8	11	12	27	25	24	45	70	79	95	406
Location													
– Australia/New Zealand	2	–	–	–	–	1	1	3	6	2	12	4	31
– Canada	–	–	3	1	5	14	12	10	17	3	–	1	66
– Europe	4	–	2	2	1	2	5	3	5	33	26	12	95
– Rest of World	–	–	–	–	–	2	1	1	3	2	6	7	22
– South Africa	–	–	–	1	–	1	–	–	4	3	13	2	24
– United States	4	–	3	7	6	7	6	7	10	27	22	69	168
Species affected[a]													
– Birds	1	–	1	1	4	16	5	3	–	11	8	12	62
– Dogs	1	–	–	1	–	5	7	5	7	21	31	72	150
– Livestock & poultry	7	–	6	8	5	13	15	13	17	27	24	7	142
– Fish & aquatic organisms	4	–	2	2	3	5	8	5	20	20	19	13	101
– Other mammals	–	–	–	1	1	6	3	–	2	8	5	6	32
– Other	–	–	–	1	1	–	–	–	2	1	1	3	9
Humans													
Number of incidents	3	–	–	2	8	9	9	12	13	18	16	25	115
Location													
– Australia	1	–	–	–	–	–	–	–	1	2	5	–	9
– Canada	–	–	–	–	–	–	4	1	–	–	–	–	5
– Europe	2	–	–	–	–	–	1	–	1	13	6	4	27
– Rest of World	–	–	–	–	–	–	1	11	2	2	4	4	24
– United States	–	–	–	2	8	9	3	–	9	1	1	17	50
Exposure mode													
– Drinking/domestic use	1	–	–	2	–	–	1	11	3	6	3	–	27
– Recreational activities	1	–	–	–	7	9	8	1	8	6	6	19	65
– Haemodialysis	–	–	–	–	–	–	–	–	1	–	1	1	3
– Occupational	–	–	–	–	–	–	–	–	–	2	1	2	5
– Other[b]	1	–	–	–	1	–	–	–	1	4	5	3	15

Excludes: incidents where a year wasn't specified or couldn't be determined; incidents recorded over open or extended periods of time where numbers affected in each year could not be determined (e.g. Carmichael and Li, 2006; Hayman, 1992; Mez et al., 1997; Wilde et al., 2014), and the following epidemiological studies (Aro et al., 1980; Backer et al., 2008; Backer et al., 2010; El Saadi et al., 1995; Lévesque et al., 2014; Muittari et al., 1980; Pilotto et al., 1997; Pilotto et al., 1999; Stewart et al., 2006a).

[a] The sum of the animal species affected for a given period may be greater than the total number of reported incidents. This occurs because multiple species of animals are often affected in a single incident.

[b] Includes incidents where no mode of exposure is specified, or where a combination of exposure modes (e.g. drinking water and recreational activities) is specified.

two-thirds of reported incidents have occurred in Europe and the United States. In terms of species affected, dogs and livestock account for the majority of reported incidents. When humans have been acutely exposed to cyanotoxins, recreational activities were responsible for approximately half of reported incidents.

Caution should be exercised, however, before using these results to claim that the actual number of cyanotoxin-related incidents is rising, that incidents involving a certain species or exposure mode are increasing, or that incidents occurring in a certain location occur more frequently. Since anecdotal reports in newspapers or from online sources were excluded from this review, the results underestimate the true number of poisoning incidents occurring each year. The lack of awareness of cyanobacterial blooms as a potential health hazard also means that many cases are likely misdiagnosed or go unreported.

Several other factors may also affect the number of incidents reported in the scientific literature. It is likely that early poisoning incidents in Australia, Canada, the United States and South Africa increased awareness of the issue and influenced the research agenda in these countries. Elsewhere, incidents may not be reported until one of sufficient severity occurs to initiate research on the issue. Papers with a specific research focus may also 'skew' the perceived severity or frequency of one aspect of problem. For example, Backer et al. (2013), looked at canine cyanotoxin poisonings in the United States from the 1920s to 2012. Due the large number of incidents documented in this paper, dogs and the United States account for the highest number of reported incidents by species type and location respectively. Hence care should be taken when interpreting these results or drawing specific conclusions.

The when, where, who and how aspects of reported poisoning incidents are summarised in Table 1. Unfortunately due to data limitations it is difficult to estimate how many animals and humans have been affected in these incidents. Of the 443 animal incidents included in this review, the number of animals affected was 'not specified' in nearly one-third of all studies. Inexplicit descriptors such as "many", "few", "large" and "massive" also make it difficult to determine with any degree of accuracy how many animals may have been poisoned.

Despite these limitations, an attempt at some general observations about the number of animals affected by cyanotoxin poisoning has been made. In Fig. 1, an estimate of the number of animals affected in each incident from 1900 onwards is shown. Since explicit numbers proved problematic, broad categories were used: ns (not specified), <10, 10–49, 50–99, 100–999 and >1000. For fish, phrases such as "many" and "large" were allocated to 100–999; "major", "mass", "massive", "immense" or "tonnes" were allocated to >1000. For other animals, 'several' or 'few' were assigned to <10.

Several observations can be made from Fig. 1. The number of birds affected varies considerably. Incidents where a low number of birds are affected often coincide with livestock poisonings, whereas incidents where a high number of birds are affected are more likely to be associated with large bird colonies such as flamingos. Livestock and poultry incidents show the most variation in the number of animals affected. Dog poisonings, although frequent, are usually limited to low numbers. Where the number of fish is specified it tend to be very high, with typically thousands of fish affected. It should, however, be noted that not all fish kills associated with cyanobacteria are necessarily the result of toxin exposure. Fish mortalities associated with cyanobacterial blooms have also been attributed to oxygen depletion (Barica, 1978; Mackenthun et al., 1948) and gill irritation (Rodger et al., 1994). Hence, the exact cause of fish mortalities in the presence of a cyanobacterial bloom is often not determined.

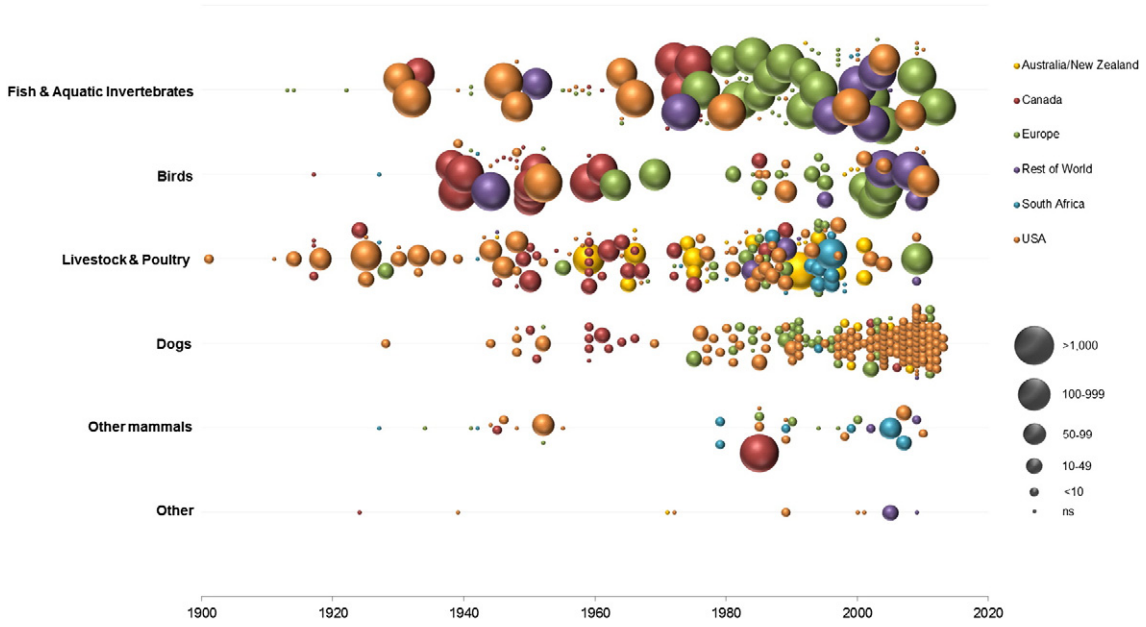

Fig. 1. Number of animals affected by cyanotoxin poisoning, 1900 to present. Excludes: incidents where a year wasn't specified or couldn't be determined, and incidents recorded over open or extended periods of time where numbers affected in each year could not be determined (e.g. Carmichael and Li, 2006; Mez et al., 1997; Wilde et al., 2014).

A similar representation of the number of humans affected by cyanotoxins is shown in Fig. 2. Here, any reference to "several" and "a number" were assigned to <10, and "many" were assigned to 10–49. Fig. 2 shows that contamination of a drinking water supply generally results in a large number of a people being affected. Recreational exposure, on the other hand, tends to affect a lower number of people, but incidents of this type are more prevalent.

4. Concluding remarks

Understanding the potential impacts of toxic cyanobacterial blooms is vital for effectively managing them. The general observations drawn from this review can be used to better inform policy decisions regarding the management of blooms and the risk of exposure to cyanotoxins through consumptive and recreational activities. For example, drinking water contamination is an infrequent occurrence, but if it does occur it has the potential to affect a large number of people. Conversely, recreational exposure appears to be a more frequent occurrence but the numbers affected in any given incident are typically much lower. Poisoning through haemodialysis is rare, but given the nature of the treatment (i.e. intravenous exposure) and the volume of water used, a contaminated supply can potentially affect a large number of individuals, and may also result in fatalities. With regard to animals, they are more likely to be fatally poisoned than humans. This is not necessarily because they are more susceptible to cyanotoxins but rather that they may not have access to treated water or there may be limitations on their ability to

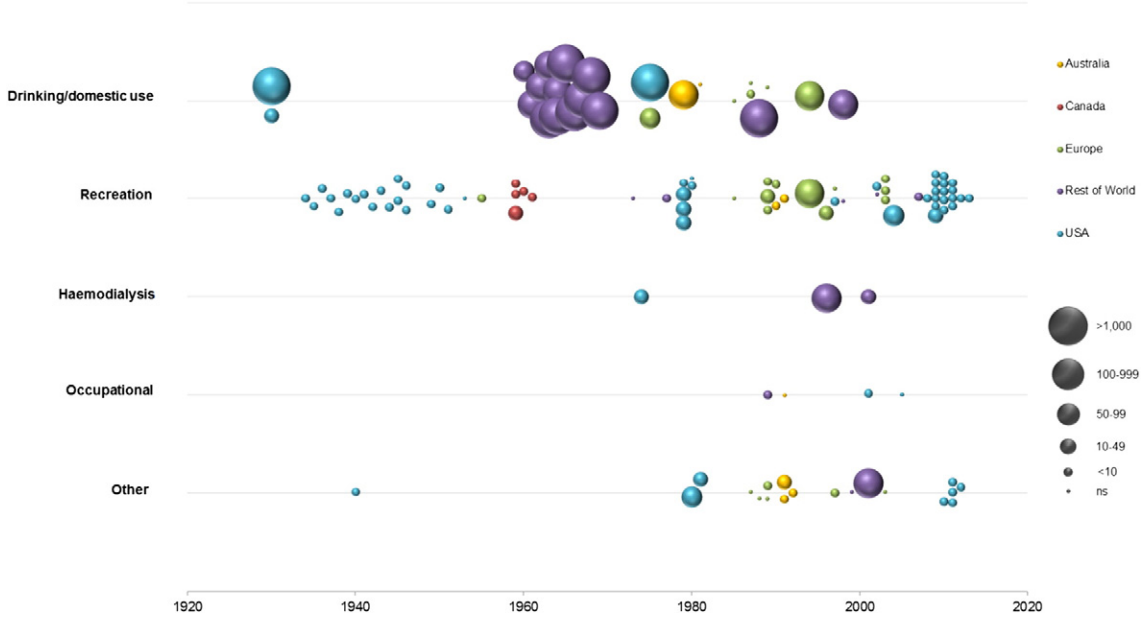

Fig. 2. Number of humans affected by cyanotoxin poisonings, 1920 to present. Excludes: incidents where a year wasn't specified or couldn't be determined; incidents recorded over open or extended periods of time where numbers affected in each year could not be determined (e.g. Hayman, 1992); and the following epidemiological studies (Aro et al., 1980; Backer et al., 2008; Backer et al., 2010; El Saadi et al., 1995; Lévesque et al., 2014; Muittari et al., 1980; Pilotto et al., 1997; Pilotto et al., 1999; Stewart et al., 2006a).

source alternative drinking water supplies when their primary source is contaminated (for example, livestock confined to a pasture with a single water source). Some animals may even be attracted to cyanobacteria (Codd et al., 1992; Steyn, 1943), while others have demonstrated an inability to discriminate between contaminated and alternative supplies when both are available (Carbis et al., 1994; Lopez Rodas and Costas, 1999). Humans, by comparison, are less likely to drink from or swim in water that contains visible and malodorous algal blooms.

The true incidence of acute poisonings in animals and humans from exposure to cyanotoxins is not known. However, this collation of anecdotal and case reports demonstrates that cyanobacterial blooms present an ongoing risk to human health and animal welfare worldwide. Given that research suggests that climate change and increased eutrophication of freshwater systems will lead to an increase in the frequency, severity and geographic distribution of cyanobacterial blooms (Paerl and Huisman, 2008; Paerl and Huisman, 2009; Paerl and Paul, 2012), the risk to humans and animals is not likely to reduce over time.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.envint.2016.02.026.

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