Communications

Coastal and inland cyanobacterial blooms (blue-green algae) – hazards to human and animal health

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Abstract

The aim of this communication is to increase the awareness about the hazards presented to animal and human health by cyanobacterial toxins, signs of poisoning by the toxins, samples to be taken for analysis and of sources of information on therapy. Heightened awareness is advocated in view of the increasing occurrence of cyanobacterial blooms and scums in Polish waters and of reports of animal intoxications attributed to cyanobacterial toxins in neighbouring and other countries around the Baltic Sea.

Mass growths (blooms) and scums of cyanobacteria (formerly known as blue-green algae) are an annual characteristic of many Polish water bodies, including freshwater lakes, reservoirs and ponds, slow-flowing rivers, estuaries, coastal lagoons and the open waters of the Baltic. Cyanobacterial blooms and scums present hazards to animal and human health because all of the species which characteristically form the blooms and scums are capable of producing potent toxins (Carmichael, 1992; Bell and Codd, 1994). Animal fatalities, bird- and fish-kills associated with cyanobacterial blooms and scums, or attributed to cyanobacterial toxins, have been reported from around the world for more than a century. Animal deaths, including cattle, sheep, horses, pigs and dogs and a variety of wildlife have occurred after drinking water containing toxic cyanobacterial blooms and scums (Beasley *et al.*, 1989). In the case of dogs, poisonings also occur after ingesting scum material during coat cleaning after entering water containing scum, or after eating shoreline crusts of drying cyanobacterial scum (Edler *et al.*, 1985; Codd *et al.*, 1992).

In Europe, toxic cyanobacterial blooms and scums have been reported from at least 20 countries, including Poland. However, cyanobacterial blooms do not always produce toxins. From national and state bloom toxicity surveys in *e.g.* Finland, Norway, Sweden, the UK and Australia, it has been established that around 50% of blooms and scums at the least are toxic in bioassays. This high probability of positive toxicity has led to the policy that a cyanobacterial bloom or scum should be assumed toxic, unless found to be otherwise. Although sporadic animal poisoning due to cyanobacterial toxins is reported, a problem of under-recognition, incomplete case investigation and lack of reporting exists.

This communication aims to increase the awareness of policy makers about the hazards presented to animal and human health by cyanobacterial toxins, signs of poisoning by the toxins, samples to be taken for analysis and of sources of information on therapy. Heightened awareness is advocated in view of the increasing occurrence of cyanobacterial blooms and scums in Polish waters and of reports of animal intoxications attributed to cyanobacterial toxins in neighbouring and other countries around the Baltic *e.g.* Germany, Denmark, Sweden, and Finland (see Nehring, 1993).

Common potentially toxic cyanobacteria in Polish freshwater include the unicellular *Microcystis* genus and the filamentous genera *Anabaena*, *Oscillatoria*, *Aphanizomenon* and *Nodularia*. The filamentous genera also occur in estuarine and coastal waters. Bloom- and scum-forming cyanobacteria produce chlorophyll, a range of blue pigments and sometimes red pigments. They may therefore discolour the water green, blue-green or brown, producing corresponding scums. Decaying cyanobacterial scums vary widely in colour from green or blue-green, through pale-blue/white and dark blue streaked with red, to dark brown.

Toxin properties, modes of actions and signs of poisoning

The alkaloid neurotoxins anatoxin-a and homoanatoxin-a are postsynaptic neuromuscular blocking agents, resulting in death by respiratory arrest. The alkaloid toxins saxitoxin and neosaxitoxin are sodium channel-blocking agents causing paralysis. The guanidinium methyl phosphate ester neurotoxin anatoxin-a(s) is an irreversible inhibitor of acetylcholinesterase. Signs of cyanobacterial neurotoxicosis in animals at peracute doses can appear rapidly with severe respiratory distress, convulsions, or death within about 15 to 45 minutes after drinking bloom or ingesting scum material. Signs associated with cvanobacterial neurotoxicoses include muscle fasciculations, lethargy, collapse, cyanosis, opisthotonos in birds, and convulsions. Anatoxin-a(s) in addition can promote hypersalivation, lachrymation, ataxia, tremors, dyspnoea and diarrhoea. The hepatotoxins include over 50 variants of a cyclic heptapeptide group microcystin and a smaller number of cyclic pentapeptides (nodularins). Microcystins and nodularing are slower acting: they are accumulated in the liver where they bind irreversibly and inhibit key regulatory enzymes (protein phosphates). The toxins may also accumulate in other tissues. Signs of poisoning include weakness, lethargy, pallor, cold extremities, bloody diarrhoea, respiratory distress and coma, with death occurring due to cardiovascular collapse within a few hours to several days. The liver appears swollen, mottled and dark, with marked lobular haemorrhages in mammals. Indications of liver dysfunction due to hepatotoxins may include increased bile acid levels, with elevated alanine aminotransferase, gamma glutamyl transferase, aspartate aminotransferase and alkaline phosphate activities in the serum. A dilated heart, blood in the pericardium, haemorrhage in the spleen and in the intestines may also occur.

Cyanobacteria also produce lipopolysaccharide endotoxins, which may result in gastroenteritis accompanying the above syndromes. Photosensitisation with blistering, peeling and oedema of exposed white skin of the ears and eyelids after ingesting cyanobacterial material has also been recorded. For further details of diagnosis see Beasley *et al.* (1989).

Collection and treatment of material for investigation of suspected cyanobacterial toxicoses

For the investigation of cyanobacterial poisoning, bloom or scum samples should be collected as soon as possible at the site of the incident. This is necessary because the toxicity of cyanobacterial blooms and scums can vary within a few days, and scum formation and distribution is dependent upon weather conditions. Scums may form during periods of calm, stable conditions, *e.g.* between June and November, and be concentrated on a shoreline by a gentle onshore wind. This may concentrate the cyanobacterial toxins more than a million-fold to present a lethal oral dose in much less than the daily water requirements of an animal. However, if wind speed increases or direction changes, the scum can be dispersed in a few hours. Bloom and scum samples should be collected for microscopic identification of the cyanobacteria and for toxin analysis by appropriate laboratories. Details of quantities needed and sample storage are available from the first author. Cyanobacterial toxin analytical methods are available elsewhere (Codd *et al.*, 1994).

In addition to environmental samples, it is recommended to collect the following material from intoxicated animals during therapy or at post-mortem: if blue-green material (*e.g.* dried scum) is found adhering to the muzzle or legs, this should be sampled for microscopy and toxin analysis. Samples of stomach washings, or of stomach or rumen contents should be collected for microscopy for cyanobacterial cells and for toxin analysis (Edwards *et al.*, 1992).

Prognosis and therapy

Recovery from cyanobacterial neurotoxicosis and hepatotoxicosis is possible. Emetics, gastric lavage, sedatives, activated charcoal and supportive fluid therapy and ventilation have been successful (*e.g.* Beasley *et al.*, 1989; Gunn *et al.*, 1992). Therapy for anatoxin-a(s) toxicosis may be possible using a cholinergic blocker *e.g.* methyl atropine nitrate (Beasley *et al.*, 1989).

Prevention of human and animal intoxications

Water with cyanobacterial blooms must be closed for public use (drinking water supply, recreation) until results of analyses confirm no toxicity.

The most effective way to prevent animal poisonings is to protect animals from ingesting cyanobacterial blooms and scums. This is achieved by increasing the awareness of farmers, animal owners and waterbody authorities of the hazards presented by cyanobacterial blooms and scums. Animals should be prevented from drinking from or entering cyanobacterial scums as a minimum policy. If an alternate water supply is not available for drinking, then owners should be advised to allow animals to drink on the upwind side of a waterbody away from the direction of scum accumulation.

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