Volume 3 Datasheets – Micro-organisms

Part 1.3: Cyanobacteria

2019

### Note

The cyanobacteria are discussed together in this single datasheet.

The cyanotoxins are covered individually in Part 2: Datasheets for Chemical and Physical Determinands, 2.4: Cyanotoxins.

# Cyanobacteria

## Maximum Acceptable Value

There are no drinking-water MAVs for cyanobacteria in the DWSNZ. MAVs (provisional) have been established for some cyanotoxins.

The DWSNZ (2000) had a MAV of less than 1 potentially toxic cyanobacterium present in 10 mL of sample (of drinking-water). There was also a MAV of less than 1 toxic alga present in 10 mL of sample. These MAVs were dropped from DWSNZ 2005 because:

* cell numbers in drinking-water are difficult to relate to cyanotoxin concentrations
* counting cells at the 100 per litre level is not very precise
* cyanotoxin concentrations in drinking-water are more likely to relate to the number of cells in the raw water than in drinking-water
* cell numbers in the raw water can vary greatly in a short time
* cell numbers in the source water can vary greatly with horizontal and vertical distribution in water bodies
* these variations can be quite rapid, depending on wind direction, light and other factors.

### Background

In the past the DWSNZ and *Guidelines* have referred to toxic algae and blue-green algae. The species used for early nomenclature were blue-green in colour; hence, a common term for these organisms was blue-green algae. However, owing to the production of different pigments, there are a large number that are not blue-green, and they can range in colour from blue-green to yellow-brown to red. They are now called cyanobacteria. It is the cyanotoxins (which are chemicals that they exude) that cause the health effects.

Chapter 9 of the *Guidelines* discusses water supply issues related to managing cyanobacteria and cyanotoxins generally and compliance with the DWSNZ more specifically. Sections 9.5.1 and 9.5.2 of the Guidelines discuss sampling, identifying and counting cyanobacteria, and various Alert Levels.

The size of cyanobacterial cells makes a large difference as to how many cyanobacterial cells are likely to represent a public health problem because the cyanotoxin level produced is thought to be approximately proportional to cell volume. It is for this reason the *New Zealand Guidelines for Cyanobacteria in Recreational Fresh Waters* – *Interim Guidelines (2009)* recommend biovolume determinations as well as cell counts to estimate the risk to public health from recreational water and this approach is considered to be appropriate for drinking-water sources as well.

Health warnings have traditionally been issued when cyanobacterial densities exceed a threshold of 15,000 cells per mL. In recent years cell concentrations of pico-planktonic cyanobacteria (<2 μm) have become increasingly prevalent and at times exceeded this threshold resulting in the unnecessary issuing of health warnings. Biovolume takes into account the variability in size of different species and is therefore a better indicator of potential health risk than cell concentrations. Calculation of biovolume requires time-consuming measurement of individual cells. A list of standardised volumes for cyanobacteria in the Rotorua lakes would greatly assist ENVBOP in incorporating biovolume thresholds into their current monitoring programme. Cawthron Institute and University of Waikato were asked by Environment Bay of Plenty to assemble a list of biovolumes for 10 problematic cyanobacteria of the Rotorua lakes. Cell biovolumes were calculated for the following species: *Anabaena lemmermannii, A. planktonica, Aphanocapsa holsatica, Aphanizomenon gracile, Aphanothece clathrata, Coelosphaerium kuetzingianum, Microcystis* sp. (small), *Microcystis* sp. (large), *M. wesenbergeii* and *Snowella lacustris* (Wood et al 2008).

In Australia there has been an attempt to relate cell numbers to toxin levels. The *Australian Drinking-water Guidelines* (2011) have suggested a tiered framework involving an initial level to health authorities, and an alert level. Cell concentrations in the raw water relate to toxin concentrations in finished water. The cell concentrations given represent the cell concentrations that are thought to contain sufficient toxin to reach the Australian drinking-water guideline concentration.

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| --- | --- | --- |
|  | **Notification levels** | **Alert levels** |
| *Anabaena circinalis* | 6,000 cells/mL;biovolume 1.5 mm3/L | 20,000 cells/mL;biovolume 5 mm3/L |
| *Cylindrospermopsis raciborskii* | 4,500 cells/mL;biovolume 0.2 mm3/L | 15,000 cells/mL;biovolume 0.6 mm3/L |
| *Microcystis aeruginosa* | 2,000 cells/mL;biovolume 0.2 mm3/L | 6,500 cells/mL;biovolume 0.6 mm3/L |
| *Nodularia spumigena* | 12,000 cells/mL;biovolume 2.7 mm3/L | 40,000 cells/mL;biovolume 9.1 mm3/L |

Since 2001, several more species have been identified as toxic including *Anabaena bergii,* *Raphidiopsis curvata, Oscillatoria formosa and Aphanizomenon flos-aquae* for example but these are not included in the Australian Drinking Water Guidelines. In the absence of toxicity data, Australian water suppliers are encouraged to contact relevant health authorities when these organisms are detected. NHMRC, NRMMC (2011) notes that a change of nomenclature has been proposed for *Anabaena* to *Dolichospermum*.

### Sources to drinking-water

The cyanobacteria are a group of photosynthetic bacteria that occur throughout the world. Blooms of cyanobacteria are common in natural surface waters and impoundments. Some species of freshwater cyanobacteria may accumulate on the surface as unsightly blue-green scums. Many species are planktonic (occur suspended in the water column) while others are benthic (attached to surfaces or sediments).

Some species of cyanobacteria produce potent toxins, which are broadly classified according to their mode of action as hepatotoxins (microcystins, nodularin and cylindrospermopsin), neurotoxins (saxitoxins, anatoxin-a, anatoxin-a(s) and homoanatoxin-a), skin irritants, broadly termed endotoxins (being skin irritants, these are not covered in the DWSNZ), and other toxins. The cyanotoxins are discussed in more detail in individual datasheets.

Cyanobacterial blooms form in water bodies that provide conditions suitable for their growth and which are less favourable for competing algal or plant species. Blooms often occur when adequate levels of essential inorganic nutrients are available, and in water that is slow moving or stagnant. Different temperature ranges suit different species, and some species have the ability to fix atmospheric nitrogen. This gives those species an advantage in waters that are poor in inorganic nitrogen. Blooms can occur at any time of the year, but often occur in late spring, summer or early autumn in the more temperate regions. Optimum growth is controlled by a combination of factors including nutrient availability, light, temperature, water column stability and grazing pressure from zooplankton such as *Daphnia* sp (eg, Hietala et al 1997).

Cyanobacteria growths seem to have been more common in recent years, prompting some people to suggest that this is a result of increasing pollution, or nutrient runoff. This may be incorrect. Cyanobacteria can grow prolifically in “pollution-free” Antarctica, and have been found growing in mountainous regions in New Zealand. If the incidence of cyanobacterial growth is increasing in New Zealand, it could well be due to a reduction of nutrients reaching natural waters. If nitrogenous wastes are diverted from natural waters, thereby causing nitrogen to become a limiting nutrient, cyanobacteria with their nitrogen-fixing ability, would be advantaged. For example, *Cylindrospermopsis raciborskii* has been found to have a high uptake affinity and storage of phosphorus, dominating in reservoirs and lakes when the phosphate concentrations are below detection limits (Burford and Davis 2011).

The buoyancy of some species of cyanobacteria is regulated through the production of carbohydrates during photosynthesis. Photosynthesis causes cells to become denser, so they sink. Respiration uses the carbohydrates and this causes the cells to become less dense and they migrate up to the light, and sometimes to the surface to form the scums. This characteristic allows some cyanobacteria to use nutrients confined to cooler, deeper and often anoxic water, or the light near the surface. Mixing, caused by convection that occurs in reservoirs that are not temperature stratified, or in water bodies that are physically mixed or turbulent like fast flowing rivers, provides conditions less favourable to the growth of cyanobacteria than other algae. Diatoms can often out-compete cyanobacteria in these conditions by providing these otherwise non-motile species with access to both the nutrients and light they need for growth.

Heavy rain events can increase nutrient levels in run-off water. This can promote the development of biological growths. Cyanobacteria can respond quickly to changes in conditions, so cyanobacterial blooms may result, if other conditions are right. Heavy rain events also increase sediment levels in the receiving water; some cyanobacteria have been shown to cope with low light conditions, giving them a competitive advantage in dirty water.

Toxic and non-toxic strains of the same species can be found together in a bloom (Skulberg et al 1993, AWWA 1995, Codd and Bell 1996, Orr et al 2004). Variations in relative concentrations of toxic and non-toxic cells along with differences in the potency of the various toxins they contain means the toxicity of bloom material cannot be determined by microscopic examination. Additionally, changes in toxicity can occur temporally and spatially within a water body (Hrudey et al 1994) due to movement of cells, changes in species dominance and in rates at which toxins are produced. The unpredictability of toxicity of blooms renders them potentially dangerous and suspect at all times (Ressom et al 1994). Prevention of cyanobacterial blooms is therefore the key to the control of toxins in source water (WHO 1998).

Many environmental factors have been implicated in the production of toxins by cyanobacteria. Orr and Jones (1998) and Long et al (2001) showed that the rate of microcystin production by nitrogen-limited *M. aeruginosa* was exactly the same as the rate of cell division. This finding held true for other species of microcystin-producing cyanobacteria including *Oscillatoria agardii* and *Anabaena flos-aquae* irrespective of the environmental variable. Hence, for microcystin, a change in toxicity of a bloom is controlled by changes in the biomass of toxic strains within the bloom.

Cawthron (2015) notes that the rivers with observed *Phormidium* issues are primarily non-alpine rivers on the lower-lying parts of the dry, eastern side of New Zealand. These are also often areas with shallow aquifers that are part of an increasingly allocated water supply, often used to support intensive agriculture. Outcome-driven thinking would require that we consider possible ways in which these common features could promote *Phormidium* growth. Based on our current understanding, the most likely processes are:

* water abstraction (both direct and indirect via groundwater abstraction) and flow modification. These can affect median flow, velocity and flood frequency
* run-off of nutrients, fine sediments and other contaminants (ie,herbicides, hormones, pesticides) from intensive agriculture, and to a lesser extent forestry and urban development
* habitat modification including: changes in riparian zones, removal of shading, and channel modification through removal of gravel for construction or flood protection.

Tables 9.1 and 9.2 in Chapter 9 of the *Guidelines* show some of the species found internationally and in New Zealand that produce toxins, the nature of the toxin produced, and where the species was found. This list is continually increasing, and should not be regarded as definitive. The tables provide a guide to those trying to determine whether a cyanobacterial species found in a water may be a toxin producer. Toxins are not species-specific, and toxins produced by one species in one location may be produced by a different species elsewhere.

There have been no records of a single cyanobacterial strain producing more than one type of toxin although some strains produce multiple microcystins, and others produce multiple saxitoxins.

Individual chemical datasheets are provided for each of the following cyanobacterial toxins: anatoxin-a, anatoxin-a(s), cylindrospermopsin, endotoxins, homoanatoxin-a, microcystins, nodularin, and saxitoxins.

### Forms and fate in the environment

Cyanobacterial toxins are either membrane-bound or occur freely within the cells. In laboratory studies, most of the toxin release occurs as cells age and die and passively leak their cellular contents (Orr and Jones 1998), although active release of toxins can occur from young growing cells (Pearson et al 1990) and from *C. raciborskii*. Microcystins and anatoxins can be degraded by bacteria (Jones and Orr 1994; Bourne et al 1996; James et al 1998) or adsorbed by soils (Morris et al 2000).

### Method of identification and detection

Cyanobacteria are members of the eubacteria group of prokaryotes. They have a typical bacterial intracellular structure except for extensive thylakoid membranes that contain the structure, enzymes and pigments necessary for photosynthesis. They can occur as single cells, as filaments (chains of cells) or colonies. Some species are able to fix atmospheric nitrogen, and some produce gas vacuoles that confer buoyancy enabling those species to migrate vertically in the water column (NHMRC and NRMMC 2004).

Although they typically grow aerobically like plants, and fix carbon dioxide in the light during photosynthesis, some can also grow heterotrophically (ie, using existing organic carbon sources for respiration). This may enable those cyanobacterial species to survive below the euphotic zone and the oxycline (ie, near the bottom of deep storages in low light and in near-anaerobic conditions).

The common problem genera are: *Microcystis*, *Oscillatoria/Planktothrix,* *Anabaena, Anabaenopsis, Aphanizomenon, Nodularia*, *Cylindrospermopsis* and *Phormidium*. Definitive identification is often difficult, and considerable variation in morphology can occur within a species. Detailed descriptions are given in Chorus and Bartrum (1999). Some are described in part 10 of APHA (2005).

Cyanobacteria are usually identified using high-powered light microscopes, and cells are counted using specialised chambers. The structure of colonies also presents problems in counting. For example, *Microcystis* can form colonies of a few cells or several thousands of cells that are difficult to separate for counting, and *Anabaena* forms long tangled filaments.

### Removal methods

#### Prevention of bloom formation

Preventing blooms from developing in the first place is the best option for reducing the risk to drinking-water supplies from cyanobacterial toxins. Management strategies such as total catchment management to minimise or control nutrient inflows into water bodies, along with in-reservoir interventions such as destratification to reduce water column stability can minimise nutrient release from sediments and mitigate conditions that promote growth of cyanobacteria (adapted from NHMRC and NRMMC 2004). Benthic cyanobacteria may proliferate in rivers during periods of stable low flows; they may be able to be removed by flushing from an upstream impoundment, if available. An Australian publication (May 2009) *A Practical Guide to Reservoir Management* Research Report 67 by CRC for Water Quality and Treatment includes cyanobacteria (<http://www.waterquality.crc.org.au/publications/report67_Practical_Guide_Reservoir_Management.pdf>).

One way to prevent blooms is to try to understand what encourages them to grow. McAllister (2014) studied the environmental factors that promoted *Phormidium* blooms in Canterbury rivers. Sites with regular *Phormidium* blooms were generally dominated by larger substrate (boulder and cobble). Sites without *Phormidium* blooms were dominated by smaller substrate (sand/silt, fine gravel and gravel). All sites had low dissolved reactive phosphorus concentrations. There were differences in dissolved inorganic nitrogen concentrations but these did not relate to probability of bloom formation. *Phormidium* was observed in a range of water temperatures, between
4–20°C. A distinct pattern existed at some sites between flushing flows (three times median flow) and *Phormidium* percentage cover, with more frequent flushing flows resulting in decreased *Phormidium* percentage cover. However, the general flushing rule that three times the median flow is sufficient to remove all *Phormidium* mats was not applicable in all of the Canterbury rivers studied. A large flushing flow of 22 times the median occurred at Pareora at the huts on 28 January 2011 and did not remove all the *Phormidium*. *Phormidium* had no specific preference for water velocity and depth, but occurred at a range of depths (0.03–0.59 m) and point velocities (0–1.4 ms-1). It appears that water quality is a weak predictor of *Phormidium* blooms; substrate stability and flow may be the most important factors controlling the dynamics of *Phormidium* in Canterbury rivers.

#### Use of algicides

Algicides have been used widely in some areas to control cyanobacterial blooms (eg, Kuiper-Goodman et al 1999; Chorus and Mur 1999), sometimes with unforeseen negative consequences.

Algicides disrupt cells and this can release cell-bound toxins into the surrounding water. The water itself then becomes toxic and can remain so for long periods. Conversely, the toxins released from thick surface scums can be diluted into the rest of the water body and quickly fall to concentrations that are below guideline or detection levels (Jones and Orr 1994).

Algicides can also have ecological consequences. They can promote shifts in dominance towards more resistant strains and species. This occurred in California where control, by application using repeated does of copper sulphate (CuSO4), of an *Oscillatoria* bloom to reduce taste and odour problems, caused a shift to a copper tolerant species of *Phormidium*. The *Phormidium* prevailed for longer, and caused year round taste and odour problems (Izaguirre 1992). Copper-based algicide use may also negatively affect populations of environmental microbes that contribute to biodegradation of cyanotoxins in water sources (Smith et al 2008). Use of CuSO4 can also cause build-up of toxic Cu residues in sediments (Prepas and Murphy 1988). Use of copper-based algicides is now restricted or banned in many jurisdictions but is still permitted in New Zealand, although rarely used. The application of copper-based algicides is most likely to be effective if used before bloom formation, when cyanobacterial cell numbers and toxin levels are low (Smith et al 2008).

Use of algicides is not recommended as a prima facie method for control of cyanobacteria and should only be considered the option of last resort. If used, algicides must be used strictly in accordance with local environment and chemical registration regulations. They tend to prevent the onset of a bloom rather than dispel a bloom; that implies a certain degree of previous knowledge is needed for its success.

#### Abstraction of water for treatment

Intake valves in storage reservoirs usually have multiple depths. Deeper levels will invariably have lower cyanobacterial cell concentrations so selective withdrawal of water from a different depth can minimise the intake of toxic cells to water treatment plants. The level from which to withdraw water will also depend on other water quality criteria because deeper water may be anoxic, and may be high in dissolved Fe and Mn. The cyanotoxins cylindrospermopsin and deoxycylindrospermopsin, which are released from the cyanobacterial cells during cell growth, have been shown to concentrate in sub-surface water layers and this should be considered when determining water abstraction levels when these toxins may be present (Everson et al 2009).

#### Treatment of drinking-water

This is covered in Chapter 9 of the *Guidelines*.

### New Zealand significance

Cyanobacterial blooms have been recorded in New Zealand (eg, Viner 1987, Carmichael et al 1988, Rinehart et al 1988, Hamill 2001) and many of these have tested positive for toxins (Stirling and Quilliam 2001; Wood and Stirling 2003). Occurrence of toxic blooms in New Zealand water bodies continues to increase with blooms being recorded in lakes including the Rotorua lakes, and constructed reservoirs. They have appeared in the Waikato River. Species recorded in New Zealand that produce toxins appear in Table 9.2 of the Guidelines.

The following notes were taken from Cawthron (2015).

#### *Nostoc*

*Nostoc* is usually found in fast-flowing relatively clean water, upland streams and rivers, and grows attached to rocky substrates. Thick gelatinous mats of *Nostoc commune* were observed on the eastern shore of Lake Taupo in 2003. These accumulated along the shoreline following a storm event that dislodged colonies from rocks. Analysis using liquid chromatography-mass spectrometry (LC-MS) detected high concentrations of microcystins (708 mg/kg dry weight), predominantly the variants MC-RR (535 mg/kg) and DMeRR (142 mg/kg). Water samples collected close to the shoreline also contained microcystins, demonstrating toxins were being released. *Nostoc* spp. are common in many New Zealand rivers and streams.

#### *Oscillatoria* sp.

Following rapid deaths of dogs at the Waikanae River (Wellington) in 1998, the toxicity of a benthic mat dominated by *Oscillatoria* sp. was investigated using a mouse bioassay and high performance liquid chromatography with fluorescence detection (HPLC‑FLD). The presence of natural degradation products of anatoxin-a was confirmed. Further sudden deaths of dogs were reported at the Mataura River (Southland) in 1999 and 2000. Benthic *Oscillatoria* sp. mats were collected and mouse bioassays confirmed their high toxicity (death within five minutes). Detailed taxonomic identification of the causative species was not undertaken. Oscillatoriales are notoriously difficult to identify based on morphology alone, and it is likely that the *Oscillatoria* sp. documented in this study was *Phormidium*.

#### *Phormidium*

Since 1999, at least 17 dogs have been reported to have died after coming into contact with *Phormidium*-dominated mats in the Wellington region (GWRC 2016). The most frequent and expansive *Phormidium* blooms tended to occur in the larger, gravel-bed rivers in Kapiti, Hutt and central Wairarapa, with the Hutt and Waipoua rivers being particularly problematic. Morphological and molecular techniques identified the causative cyanobacterium as *Phormidium autumnale*. Liquid chromatography-mass spectrometry of the *Ph. autumnale*-dominated mats identified anatoxin-a, dihydroanatoxin-a, homoanatoxin-a and dihydrohomoanatoxin-a. Since this confirmation *Ph. autumnale*-dominated mats have been identified across New Zealand. When conditions are optimal, *Ph. autumnale* forms thick cohesive mats that may cover many kilometres of the riverbed. When a combined molecular and morphology approach was used to screen over 30 cultures isolated from *Phormidium*-dominated mats collected across New Zealand, *Phormidium autumnale* was the only species found to produce anatoxins. Both toxic and non-toxic strains were identified. Non-toxic strains lacked the presence of genes involved in the biosynthesis of anatoxins. *Phormidium* species are challenging to distinguish based on morphology alone. There are indications that *Phormidium-*dominated mats may contain multiple *Phormidium* species.

*Phormidium* blooms under a wide range of nitrogen concentrations but generally only at low soluble phosphorus concentrations (<0.01 mg/L). Water column nutrient availability appears to be a strong driver of why *Phormidium* blooms occur in some rivers in the region and not others. However, it does not explain why *Phormidium* blooms occur in some rivers in one year but not the next (GWRC 2016). Routine testing and research on *Phormidium-*dominated mats from around New Zealand has shown marked variations in the presence and concentrations of anatoxins and in the structural variants produced. This variability is partly due to differences in the relative abundance of toxic and non-toxic strains within a mat, and differences in the amounts of toxin each strain produces. *Phormidium-*dominated mats are commonly associated with cobble-bedded rivers in New Zealand; however, under stable flow conditions they also grow in rivers with fine substrate. Extensive mats have been identified on the bottom of lakes in the Rotorua region (those tested to date were non-toxic). In 2014 ingestion of mats dislodged from the bottom of a small farm pond, caused the death of a dog. Culturing, molecular and toxin analysis confirmed the causative species was also *Ph. autumnale*.

#### *Planktothrix* sp.

In November 2008 a dog died soon after ingesting benthic algal mat material from the Waitaki River (Canterbury). Based on morphology, the causative organism was putatively identified as *Phormidium* sp. Subsequent molecular analysis of cultures demonstrated it was a benthic strain of *Planktothrix* (a common planktonic toxin-producing genus in the Northern Hemisphere). Using LC-MS, microcystin-LR, [D-Asp3, Dha7] microcystin-LR, [D-Asp3] microcystin-LR, and minor proportions of [D-Asp3, ADMAdda5] microcystin-LhR were identified. This seems to be the only identification of this species in New Zealand, however, given its similar appearance to *Phormidium,* it is probably commonly misidentified.

#### *Scytonema cf. crispum*

Samples collected from the metaphyton of a drinking-water supply’s pre-treatment reservoir (Oamaru) and a small eutrophic lake (The Groynes, Christchurch) tested positive for saxitoxins using pre-column oxidation HPLC-FD. Cultures were established and morphological and molecular analysis identified the causative species as *Scytonema* cf. *crispum*. Initially saxitoxin was the only variant detected and this was found at concentrations of 65.6 mg/kg dry weight in the Groynes sample and 119.4 mg/kg in the culture (strain UCFS10). Subsequent surveys identified this species in additional lakes in the Canterbury region, and toxin analysis of environmental samples and cultures from these samples identified the variants: gonyautoxins
(GTX 1–5), neosaxitoxin, decarbamoyl saxitoxin and decarbamoyl gonyautoxins (dcGTX2/3). Unlike the other toxic benthic cyanobacteria described in this section, *S*. cf. *crispum* does not attach to a substratum but is free-floating in the metaphyton amongst aquatic plants, a habitat that is commonly overlooked in lake studies.

#### Unknown nodularin producer

A survey of periphyton (depth 6–7.5 m) in Lake Tikitapu (Rotorua) revealed widespread thick spongy benthic mats dominated by a mixed assemblage of cyanobacterial species from the orders Oscillatoriales*,* Nostocales and Chroococcales. All benthic mats tested contained nodularin (maximum 0.61 mg/kg dry weight. Genes involved in nodularin production were sequenced and had very low (≤ 89 percent) homology to those from other known nodularin producers, suggesting a new toxin-producing species. Attempts to identify the nodularin producer were unsuccessful. Genetic analysis has identified the presence of the same species in cyanobacterial-dominated mats in alpine tarns near Nelson. Nodularin was also detected in these mats using LC‑MS.

#### Tastes and odours

Cyanobacteria have been recognised as nuisance organisms in the drinking-water and aquaculture industries because some species produce potent odour compounds including geosmin and 2-methyl isoborneol (2-MIB). These compounds (which have datasheets) impart tastes and odours to water and fish flesh. Geosmin is released from cyanobacteria in dry soil when rain first falls and provides that distinctive odour associated with rain. Sensitive individuals can smell geosmin at concentrations approaching 10 ng/L. WHO (2011a) states that although other microorganisms, such as actinomycetes, also produce geosmin and 2-methylisoborneol, cyanobacteria are considered the major source of these compounds in surface waters.

There is no evidence of any correlation between toxin production and the production of taste and odour compounds. In many cases, different species are responsible (Falconer et al 1999).

### Health considerations

Cyanobacteria have been implicated in animal and human poisoning for over 100 years (Francis 1878; Zilberg 1966; Teixera et al 1993; Negri et al 1995; Hamill 2001) (WHO 1998).

The major route of human exposure is the consumption of drinking-water. Other minor exposure routes include accidental ingestion resulting from recreational exposure to contaminated water bodies and ingestion of food or food supplements that might be contaminated by cyanotoxins. An additional, minor route of exposure is through inhalation while taking showers. The WHO allocates 80 percent of ingestion of cyanotoxins to drinking-water (Falconer et al 1999). In Australia, the *Australian Drinking Water Guidelines* (NHMRC and NRMMC 2004) increased this to 90 percent for some cyanotoxins to reflect differences between Australia and the more generalised conditions defined by the WHO.

Microcystins and cylindrospermopsin present the greatest risk to human health because they have both acute and chronic effects, and occur commonly in fresh water systems. Nodularin is of comparable toxicity to the microcystins, but being produced by a saline species, is unlikely to occur in drinking-water.

The peptide toxins (microcystins and nodularin) accumulate preferentially in the liver following absorption from the gut. Following acute exposure, they cause a break down in liver ultrastructure by interfering with enzyme pathways in the liver, and death can occur in as little as 30 minutes from severe liver haemorrhage. Chronic exposure to both the microcystins and nodularin promotes tumour growth while nodularin has been confirmed as a carcinogen in its own right (Ohta et al 1994). These toxins can also affect other body organs after passing into the bloodstream from the liver.

Toxicity data relating to anatoxins is sparse, and completely lacking for human exposure. To date, intoxication by the alkaloid neurotoxins, including the anatoxins and saxitoxins, has only been shown to cause acute effects. Recovery from saxitoxin intoxication is usually complete. Data for saxitoxins is more complete with exposure data being derived from intoxication events resulting from consumption of shellfish contaminated from toxic marine dinoflagellates. Consumption of freshwater shellfish, such as mussels, contaminated by saxitoxins derived from freshwater cyanobacteria is an additional source of ingestion (Negri and Jones 1995).

#### General features of the cyanotoxins – summary

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| **Toxin group1** | **Primary target organ in mammals** | **Cyanobacterial genus or species (if species specific)2** |
| ***Cyclic peptides*** |  |  |
| Microcystins | Liver | *Microcystis, Anabaena, Planktothrix/Oscillatora, Nostoc, Hapalosiphon, Anabaenopsis* |
| Nodularin | Liver | *Nodularia spumigena* |
| **Alkaloids** |  |  |
| Anatoxin-a | Nerve synapse | *Anabaena, Planktothrix (Oscillatoria), Aphanizomenon, Phormidium* |
| Homoanatoxin-a | Nerve synapse | *Phormidium formosa (Oscillatoria formosa)* |
| Cylindrospermopsin | Liver3 | *Cylindrospermopsis raciborskii, Aphanizomenon ovalisporum, Umezakia natans* |
| Saxitoxins | Nerve synapse | *Anabaena circinalis, Aphanizomenon, Planktothrix, Cylindrospermopsis* |
| Lipopolysaccharides (LPS, eg, endotoxin) | Potential irritant; affects any exposed tissue | All gram negative bacteria including cyanobacteria, particularly *Lyngbya majuscula* |

1 Many structural variants may be known for each toxin group.

2 Not produced by all species of the particular genus. Where species name presented, only produced by that species.

3 Whole cells of toxic species elicit widespread tissue damage, including damage to kidney and lymphoid tissue.

Cylindrospermopsin is a hepatotoxic alkaloid found in five species of cyanobacteria. It exhibits an acute response and is thought responsible for an intoxication event in 1979 on Palm Island (North Queensland) where 10 adults and 140 children were admitted to hospital with a range of symptoms including an unusual hepatoenteritis, tender liver enlargement, constipation, vomiting and headache (Falconer 2001). This event became known as Palm Island Mystery Disease and resulted from the deliberate lysing (by CuSO4) of a bloom of what is thought to have been *C. raciborskii* in Solomon Dam. More recent studies indicate cylindrospermopsin has chronic toxicity as a carcinogen at sub-acute doses (Humpage and Falconer 2002). An AWQC Research Report (#32, March 2007) updates and summarises the Australian experience.

Lipopolysaccharides (LPS) are cell wall components of gram-negative bacteria, the group to which the cyanobacteria belong. Also referred to as endotoxins, LPS can cause both allergic and toxic responses, including skin irritant effects. Little is known currently about the cyanobacterial LPS although they are thought to be less toxic than LPS from other bacteria. The lack of knowledge regarding the occurrence and toxicity of cyanobacterial LPS prevent any guidelines being set. Recent data from Australia has linked exposure to blooms of the cyanobacteria *Lyngbya majuscula* to skin irritant effects (Osborne et al 2008; Osborne and Shaw 2008).

In order to set safe levels of toxicants or contaminants in food or drinking-water (called the Maximum Acceptable Value or MAV), it is first necessary to determine the dose level in humans that poses the maximum acceptable risk when taken over a lifetime. Studies involving animals (usually pigs, rats and mice) provide basic epidemiological data to determine either the Lowest Observable Adverse Effect Level (LOAEL) where some doubt exists about the exact maximum dose that causes no adverse effect, or the No Observable Adverse Effect Level (NOAEL) where the maximum dose that causes no adverse effect has been determined accurately. This is then converted to the average permissible daily intake based on a lifetime of exposure. After application of appropriate safety factors, this value is called the Tolerable Daily Intake (TDI), or sometimes the Acceptable Daily Intake (ADI).

#### Acute exposure

Acute exposure is a single (or brief) exposure that results in an immediate adverse health effect.

The recorded cases of gastrointestinal and hepatic illness that can be attributed reliably to cyanobacterial toxins in water supplies have all been coincident with either the breakdown of a natural cyanobacterial bloom, or with the artificial lysis of a bloom by application of algicides.

In the USA and Australia, several different cyanobacterial toxins have been implicated in human illness from municipal water supplies, often after algal blooms had been treated with copper sulphate (Bourke et al 1983; Falconer 1989, Ressom et al 1994). In most cases, the cyanobacteria and sometimes the toxins involved have been identified, but the levels of toxin associated with illness have not been established in any of the outbreaks.

The most lethal outbreak attributed to cyanobacterial toxins in drinking water occurred in Brazil, when a newly flooded dam developed a bloom of toxic *M. aeruginosa*. Approximately 2,000 gastroenteritis cases, 88 of which resulted in death were reported over a 42-day period (Teixera et al1993). Also in Brazil, 117 of 136 patients (86 percent) experienced visual disturbances, nausea, vomiting, muscle weakness and painful hepatomegaly, following routine treatment at a haemodialysis centre. Subsequently, 100 patients developed acute liver failure and 50 of these died.

Note that NHMRC, NRMMC (2011) states:

No human deaths have been recorded from ingesting the toxins of cyanobacteria but gastroenteritis may result from drinking water containing toxic species and extended exposure may lead to more serious impacts. Deaths have been attributed to the presence of microcystin in water used for renal dialysis in Caruara, Brazil (Jochimsen et al 1998).

#### Chronic exposure

Chronic exposure is one or more exposures to sub-acute doses that result in an adverse health effect over the lifetime of an individual.

Only the hepatotoxins have been demonstrated unequivocally to have chronic health effects at sub-acute doses.

ANZECC/ARMCANZ (2000) gives guideline values for livestock drinking water, and includes the following: “Algal blooms should be treated as possibly toxic and the water source should be withdrawn from stock until the algae are identified and the level of toxin determined. An increasing risk to livestock health is likely when cell counts of *Microcystis* exceed 11,500 cells/mL and/or concentrations of microcystins exceed 2.3 μg/L expressed as microcystin-LR toxicity equivalents. There are insufficient data available to derive trigger values for other species of cyanobacteria”. These guidelines were meant to have been updated in 2012.

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