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Current approaches to cyanotoxin risk assessment and risk management around the globe

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ABSTRACT

Toxic cyanobacteria became more widely recognized as a potential health hazard in the 1990s, and in 1998 the World Health Organization (WHO) first published a provisional Guideline Value of 1 μg L^{-1} for microcystin-LR in drinking-water. In this publication we compare risk assessment and risk management of toxic cyanobacteria in 17 countries across all five continents. We focus on the three main (oral) exposure vehicles to cyanotoxins: drinking-water, water related recreational and freshwater seafood. Most countries have implemented the provisional WHO Guideline Value, some as legally binding standard, to ensure the distribution of safe drinking-water with respect to microcystins. Regulation, however, also needs to address the possible presence of a wide range of other cyanotoxins and bioactive compounds, for which no guideline values can be derived due to insufficient toxicological data. The presence of microcystins (commonly expressed as microcystin-LR equivalents) may be used as proxy for overall guidance on risk management, but this simplification may miss certain risks, for instance from dissolved fractions of cylindrospermopsin and cyanobacterial neurotoxins. An alternative approach, often taken for risk assessment and management in recreational waters, is to regulate cyanobacterial presence – as cell numbers or biomass – rather than individual toxins. Here, many countries have implemented a two or three tier alert level system with incremental severity. These systems define the levels where responses are switched from Surveillance to Alert and finally to Action Mode and they specify the short-term actions that follow. Surface bloom formation is commonly judged to be a significant risk because of the elevated concentration of microcystins in a scum. Countries have based their derivations of legally binding standards, guideline values, maximally allowed concentrations (or limits named otherwise) on very similar scientific methodology, but underlying assumptions such as bloom duration, average body size and the amount of water consumed while swimming vary according to local circumstances. Furthermore, for toxins with incomplete toxicological data elements of expert judgment become more relevant and this also leads to a larger degree of variation between countries' thresholds triggering certain actions. Cyanobacterial blooms and their cyanotoxin content are a highly variable phenomenon, largely depending on local conditions, and likely concentrations can be assessed and managed best if the specific conditions of the locality are known and their impact on bloom occurrence are understood. Risk Management Frameworks, such as for example the Water Safety Plan concept of the WHO and the 'bathing water profile' of the European Union are suggested to be effective approaches for preventing human exposure by managing toxic cyanobacteria from catchment to consumer for drinking water and at recreational sites.

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1. Introduction

Cyanobacteria cause problems worldwide, and the major cause for the global occurrence of nuisance blooms is eutrophication of surface water, in particular through excessive use of fertilizer and manure in agriculture as well as through sewage discharges. Climate warming also seems to play a role (Paerl and Huisman, 2009), whether through direct effects of warming or earlier, prolonged or higher water column stability (Carey et al., 2012). Eutrophication and climate may act together in supporting cyanobacterial blooms (Brookes and Carey, 2011; Carey et al., 2012), although the evidence for synergistic interactions seems strongly dependent on trophic state and the cyanobacterial taxa involved (Rigosi et al., 2014). Blooms have been reduced successfully in a large number of lake restoration programs, which almost invariably include abatement of nutrient loading in the catchment (Schindler, 2006; Schindler et al., 2008), sometimes in combination with additional measures like biomanipulation to interrupt the hysteresis of the turbid stable state (Jeppesen et al., 2007). In some cases internal measures like artificial mixing of lakes have been successful in removing nuisance blooms even in the absence of nutrient reduction (Visser et al., 1996).

Toxic cyanobacteria started to be more widely recognized as a potential health hazard in the 1980s, a number of case studies were published attributing illness to cyanobacterial toxins (see Kuiper-Goodman et al., 1999; Chorus et al., 2000), and numerous cases of animal deaths along water courses afflicted with cyanobacterial blooms were calling public attention to the issue. Progress in the elucidation of the chemical structures of a number of cyanotoxins and in the availability of chemical detection methods suitable for routine analyses picked up speed in the mid 1980s, and by the late 1990s a wider understanding of both their modes of action and their occurrence was available (Chorus and Bartram, 1999). The accumulating data suggested that, among the chemicals found in water or used for drinking or recreation, cyanotoxins may well be among the substances occurring most frequently at potentially harmful concentrations. In 1998 the World Health Organization (WHO; see Box 1 for all abbreviations) first published a provisional drinking-water Guideline Value of 1 $\mu g\,L^{-1}$ for one very common cyanotoxin, microcystin-LR (MCYST-LR), in its Addendum to Volume 2 of the Guidelines for Drinking-water Quality (see Chorus and Bartram, 1999). Since then, the number of countries which have addressed the cyanotoxin hazard has increased and further countries are currently discussing the most appropriate regulatory approach for their respective conditions. The primarily hepatotoxic microcystins – a family of more than 80 different congeners, commonly measured and expressed as total MCYST-LR equivalents – are probably the most widespread and best studied group of cyanotoxins (Dittmann et al., 2013; Ferrao-Filho and Kozlowsky-Suzuki, 2011; Ibelings and Havens, 2008; Kozlowsky-Suzuki et al., 2012). Data on the occurrence of other cyanotoxins are increasingly becoming available, particularly for cylindrospermopsin (CYN), neurotoxins like saxitoxin (STX) or anatoxins (ATX) (Metcalf et al., 2008; Seifert et al., 2007; van Apeldoorn et al., 2007; van der Merwe et al., 2012) and information on new classes arising (e.g. jamaicamides, Neilan et al., 2013). Regulations and guidelines, however, have been struggling with the multitude of cyanobacterial toxins that might occur, be it other microcystins or different classes of toxins, particularly as for most of them, toxicological data are insufficient for the derivation of concentration limits.

To some extent toxin levels respond to environmental conditions so that the toxin content per cell may vary several fold (Neilan et al., 2013; van der Merwe et al., 2012; Wiedner et al., 2003); also the proportion of different MCYST congeners may change with changes in the environment (Tonk et al., 2005).

Box 1. Abbreviations

ATX	Anatoxin(s)-a/a(s)
BMAA	Beta-N-methylamino-L-alanine
Chl-*a*	Chlorophyll-*a*
CYN	Cylindrospermopsin
ELISA	Enzyme linked immunosorbent assay
EU BWD	European Union Bathing Water Directive
EU DWD	European Union Drinking Water Directive
EU WFD	European Union Water Framework Directive
GDWQ	WHO Guideline Values for Drinking Water
GV	Guideline value
HACCP	Hazard analysis and critical control points
(H)AL	(Health) Alert level
i.p.	Intraperitonial (injection in body cavity)
IARC	International agency for research on cancer
MCYST	Microcystin(s)
N(L)OAEL	No (lowest) observed adverse effect level
NOD	Nodularin
OATP	Organic anion transporting polypetides
PHRMP	Public Health Risk Management Plans
(P)MAC	(Provisional) Maximum concentration
(P)MAV	(Provisional) Maximum value
PST	Paralytic shellfish toxins
RMF	Risk management framework
S	Standard value
STX	Saxitoxin(s)
TDI	Tolerable daily intake
TWQR	Target water quality range
UF	Uncertainty factors
US	EPA USA Environmental Protection Agency
WHO	World Health Organization
WSP	Water Safety Plan

Maximal cyanotoxin concentrations in a given waterbody, however, largely depend on the concentrations of cyanobacterial biomass – modified by the ratio of toxic to non-toxic strains, currently or previously present. In particular, concentration via scum formation (i.e. the accumulation of floating cyanobacteria at the lake surface during periods of calm weather) may increase toxin levels by orders of magnitude Therefore the amount of cyanobacteria observed can serve as a basis for alert level frameworks and risk assessment well before, or even without toxin analysis. Accordingly, some countries are implementing alert level frameworks and risk-based approaches on basis of cyanobacterial cell numbers or biovolume in their national guidance or regulations, sometimes complementary to regulating maximum cyanotoxin concentrations.

In principle, regulatory approaches differ for the main three exposure routes to cyanotoxins, i.e. oral, pulmonary and dermal. Dermal symptoms caused by freshwater cyanobacteria are typically mild and self-limiting, thus requiring some public education and guidance, but not necessarily regulation. Concern regarding pulmonary exposure to date is based on two early studies, i.e. one exposing guinea pigs experimentally (Falconer and Humpage, 2005) and one evaluating atypical pneumonia of army cadets submersed during their training (Lawton and Codd, 1991). However, more recent studies confirming this exposure route to be relevant are lacking, and uptake through aspiration usually also involves swallowing, thus at least partially occurring via the oral pathway. Accordingly, regulations and guidelines to date focus on the main vehicles of oral exposure, i.e. ingestion of toxins via drinking-water, recreation or consumption of fish, molluscs and crayfish from freshwater bodies, which we term 'freshwater seafood'. The literature on exposure through drinking-water (e.g. Falconer and Humpage, 2005; Hitzfeld et al., 2000; Zamyadi et al., 2012) is more extensive than that for other possible exposure vehicles, notably recreational exposure (Backer et al., 2010; Chorus et al., 2000) and uptake via food (Ibelings and Chorus, 2007). The