Toxic Cyanobacteria in Water:

A guide to their public health consequences, monitoring and management

---

Edited by Ingrid Chorus and Jamie Bartram

E & FN Spon
An imprint of Routledge

London and New York

First published 1999 by E & FN Spon, an imprint of Routledge
11 New Fetter Lane, London EC4P 4EE

Printed and bound in Great Britain by
St Edmundsbury Press, Bury St Edmunds, Suffolk

British Library Cataloguing in Publication Data
A catalogue record for this book is available from the British Library

Library of Congress Cataloging in Publication Data
A catalog record for this book has been requested.

ISBN 0-419-23930-8

Also available from E & FN Spon

Agricultural Pollution
M. Redman

Determination of Organic Compounds in Natural and Treated Waters
T.R. Crompton

Box 5.2 Epidemiological evidence for low-level cyanobacterial hazard

The epidemiological data of Pilotto *et al.* (1997) can be used as a basis for guideline derivation for acute, non-cumulative health effects which are more likely to result in discomfort rather than serious health outcomes. These data encompass the health effects on humans of intact cyanobacterial cells and colonies and thus include effects of currently unknown substances and bacteria associated with cyanobacterial colonies. The effects measured were eye irritation, ear irritation, skin rash, as well as vomiting, diarrhoea, cold/flu symptoms, mouth ulcers and fever. An elevated "Odds Ratio" for symptoms (3.44) was shown by the people who were in water contact for more than one hour, at above 5,000 cyanobacterial cells per ml. Similar Odds Ratios were seen for symptoms in people bathing in water with 5,000-20,000 cells per ml (2.71) and above 80,000 cells per ml (2.90).

5.1.4 Recreational water exposure

Three potential routes of exposure to cyanotoxins can be distinguished: direct contact of exposed parts of the body, including sensitive areas such as the ears, eyes, mouth and throat, and the areas covered by a bathing suit (Pilotto *et al.*, 1997); accidental swallowing (Turner *et al.*, 1990); and inhalation of water. Cases of illness from accidental swallowing and inhalation of *Microcystis* have been reported (see section 4.1) and provide direct evidence of harm to recreational water users from cyanobacterial blooms in the recreational water bodies.

Health effects observed in the prospective epidemiological study of Pilotto *et al.* (1997) occurred at low cyanobacterial cell densities. These related clearly to the cyanobacterial cell population, but not to the concentration of microcystins (see Box 5.2). Thus, this hazard appears to be due to additional, or other unidentified, cyanobacterial metabolites or compounds from associated bacteria, even at moderate levels of exposure.

Intake through oral ingestion or inhalation

Incorporation of toxins through swallowing, contact with nasal mucosa, or by inhalation are likely to be important routes of exposure to cyanotoxins during water-contact sports. Well-documented evidence from one animal experiment (Fitzgeorge *et al.*, 1994) and one case of multiple human illness (Turner *et al.*, 1990) indicates that inhalation and resorption through nasal and pharyngeal mucous membranes may present a high risk in water sports involving intensive submersion of the head (jumping from diving boards, sailboarding, canoe capsizing, competitive swimming) and inhalation of aerosols (water skiing).

Experimental results indicate a hazard of cumulative liver damage by repeated microcystin intake (Fitzgeorge *et al.*, 1994, see section 4.2.1), as can occur during a holiday with daily bathing at a recreational site with a high density of microcystin-containing cyanobacteria. Sub-acute liver injury is likely to go unnoticed, because signs of liver injury are only apparent after severe injury. In addition, the dose-response curve for liver injury from microcystins is relatively steep. There may be little evidence of acute liver damage when levels are close to those that could lead to severe acute toxicity, and

thus exposure at such levels is likely to be continued by people if they are uninformed of the risk (e.g. for consecutive days of a holiday or hot spell), thereby increasing the risk of cumulative liver damage.

Risks of ingestion are particularly high for children playing in shallow near-shore water where scums tend to accumulate. Because the hazard of microcystin uptake is directly related to the levels of toxins in the water (cell-bound as well as dissolved) and the volume of water ingested or inhaled, the range in these levels needs to be recognised in deriving guidelines for recreational water safety.

Direct contact

Allergic and toxic dermal reactions of varying severity are known from cyanobacteria as well as from freshwater algae, but have not been documented extensively. Bathing suits, and particularly diving suits, tend to aggravate such effects by accumulating cyanobacterial cells, thereby enhancing the disruption of cells and hence the liberation of cell contents onto the wearer's skin. Reports from the USA have recorded allergic reactions from recreational exposure, and the cyanobacterial pigment phycocyanin was shown to be responsible in one case (Cohen and Reif, 1953). Severe dermatitis, resembling skin burns, has been reported from marine bathing in the presence of cyanobacteria dislodged from rocks, particularly after storms in tropical seas (see section 4.2.8).

5.2 Safe practices

The placing of barriers that reduce exposure to a cyanotoxin hazard is an important measure and involves identifying "critical control points" and implementing measures for their monitoring and control. In the case of cyanobacteria, critical control points might include, for example, noting the tendency of a water body to develop blooms, scums or mats. Monitoring schemes need to be developed that are capable of detecting proliferation of cyanobacteria (linked to a programme of appropriate actions) and drinking water treatment technology needs to be in existence that is capable of preventing human exposure if cyanobacteria occur in source waters.

5.2.1 Drinking water

A drinking water supply safe from cyanotoxins will either draw upon a resource which does not harbour cyanotoxins (e.g. groundwater or surface water which does not support cyanobacterial growth), or have treatment in place that is likely to remove cyanobacterial cells (without causing their disrupture) as well as removing cyanotoxins. However, in many circumstances a potential cyanotoxin hazard can be managed effectively without the necessity of advanced treatment processes, through water resource management techniques (see Chapter 8) and removal of intact cells (see Chapter 9). The critical control points for safe practices are indicated in Table 5.1.

Most of the reported incidents of human injury that have raised awareness of the importance of cyanotoxins in drinking water have involved the inappropriate treatment of water supplies, such as the use of copper sulphate in dealing with an established bloom of cyanobacteria.

A very effective approach to safe practices may involve changing the drinking water source. In a number of regions, surface waters are used for reasons of easy access and tradition, although groundwater of high quality is available. Exploring options of improving practices of drinking water abstraction with low technological input (such as drilling wells, or using bankside filtration) may lead to health benefits. In China, a high prevalence of endemic primary liver cancer was related to several factors: hepatitis B, aflatoxins in the diet, and drinking surface water polluted with cyanobacteria likely to contain microcystins. Changing the drinking water source from shallow, eutrophic ponds and ditches to groundwater was a major element of a package of measures which showed some success in improving health (Box 5.3).

5.2.2 Recreational waters

Recreational water use is likely to be a major route of exposure to cyanotoxins in some parts of the world. Whereas similar approaches to resource protection apply as for drinking water, there are very few further management options available once cyanobacteria proliferate or accumulate in a recreational water. Because adequate surveillance is sometimes difficult and management options, except precluding or discouraging use, may be scarce, a large share of the responsibility for safe practices lies with the users of a bathing site. The provision of adequate information to the public becomes, therefore, a major responsibility of public authorities.

The growth of cyanobacteria in lakes and rivers used for recreational purposes has been well recognised as a public nuisance. Water blooms of cyanobacteria may be associated with unpleasant odours and the offensive appearance of lake shores, especially when scums of the organisms accumulate and decay. Areas with extensive cyanobacterial scums or accumulated detached mats on bathing beaches may be avoided by swimmers and other water users because of the obviously unpleasant environment, particularly if locally anaerobic water conditions or cyanobacterial toxins cause fish-kills, further emphasising the unattractiveness of water contact. In temperate climates, cyanobacterial dominance is most pronounced during the summer months, when the demand for recreational water is highest. In some regions, cyanobacteria have been abundant for more than a generation and visitors have accepted this water quality as "normal" for their region. Multiple anecdotal observations of children playing with scum material have been reported.

Table 5.1 Critical control points for assessing the intrinsic safety of a drinking water supply which may contain cyanobacterial cells and/or toxins

Control point/issue	Comments
Source water type	The health risk associated with cyanobacterial contamination of groundwaters is generally negligible. An exception may occur where infiltration galleries are strongly influenced by eutrophic surface waters
Occurrence of cyanobacteria in source water and tendency for bloom formation	Many surface water sources do not support cyanobacterial growth. In others, cyanobacteria may occur occasionally at low population densities. In reservoirs and lakes with very low nutrient concentrations (total phosphorus < 10 µg P l^{-1}) or rivers and reservoirs with a hydrodynamic regime unfavourable for cyanobacteria (continuous high flows especially during summer, or deep vertical mixing), other phytoplankton species may regularly out-compete cyanobacteria. A water source which does not have a history of cyanobacterial growth or bloom formation is generally considered to present a low cyanotoxin risk, regardless of treatment type. Where bloom formation is well characterised in terms of annual cycles, the health risk may similarly be low if control measures are in place for times of bloom formation. If regular monitoring of source phytoplankton is in place, waters presenting no significant cyanotoxin risk are easily identified (see Table 6.2)
Likelihood of cell lysis in transport or treatment	Throughout cyanobacterial growth, most cyanotoxins are cell-bound. Removal of intact cyanobacterial cells therefore largely removes cyanotoxins (see section 3.4). Neurotoxins may be an exception under some circumstances. When cyanobacterial cells die and decay (lyse), toxins are released. Lysis can occur naturally or be caused by chemical treatment, hydraulic and pumping regimes in different treatment steps, and by long transport pipes for raw water. Thus, abstraction and treatment systems which lead to cell lysis present an increased risk of cyanotoxin release.
Treatment systems capable of toxin removal	Methods, such as adsorption to some types of granular activated carbon, and oxidation, can be effective in cyanotoxin removal. However, conditions of operation are critical for success. If processes are operated only periodically during cyanobacterial growth or reservoir treatment, monitoring of plant functioning must be adequate to ensure cyanotoxin removal. Substantially less is known about removal of neurotoxins and cylindrospermopsin than about microcystins, thus toxin monitoring of treatment steps and finished water is especially important if potentially neurotoxic or cylindrospermopsin-producing cyanobacteria proliferate

Box 5.3 Primary liver cancer and cyanotoxins in China

Primary Liver Cancer is one of the most common cancers in China. In 1994 and 1995, it accounted for 24 mortalities per 100,000 population in some rural counties and cities; in these areas it was ranked with stomach cancer as the two most important causes of cancer death.

The uneven geographic distribution of liver cancer was conspicuous, and "hot spots" could be related to drinking water supply, e.g. in some clearly delineated areas of Nandong District, in Jiangsu Province (particularly in Rudong, but also in Haimen and Qidong), in Nanhui (suburb of Shanghai) and Fusui (Guangxi).

- In Rudong, Nanhui and Fusui people had blocked the drainage system, causing stagnation of the water used for the drinking supply.

- In areas of Qidong-Haimen, with mortality rates 20 per 100,000, people drank water from the Yangtze River, but in areas with mortalities of 100 per 100 000, pond and ditch water was used.

- Primary liver cancer mortalities 10 per 100,000 were found in areas where water from deep wells were used for drinking.

Epidemiological study of the mortality showed strongest correlation with hepatitis B incidence, a lesser correlation with aflatoxins in the diet, and a third correlation with drinking of pond and ditch water. No correlations were found with insecticides. Samples of pond and ditch water showed microcystin present in both endemic liver cancer areas and in areas with lower liver cancer rates. Children in some endemic areas were fed corn paste and drank pond or ditch water from infancy. Further, up to 43 per cent carry the hepatitis-B virus from infection by their HBsAg positive mothers. The evidence suggests that aflatoxins from corn and microcystins from drinking water act together with the hepatitis B virus in causing and promoting primary liver cancer.

In order to alleviate this situation, attempts have been launched over the past 20 years to change the staple food and drinking habits of the people. Efforts began with the methods of harvest, following the motto *"quick to reap, quick to store, at a moisture content 12.5 per cent"*, aimed at the reduction of fungal contamination. For some time, the government bought corn and exchanged it for rice to reduce aflatoxin exposure, but this function has now been transferred to a private initiative in the market economy. Recently, it has been estimated that more than 95 per cent of the population eats rice rather than corn.

Even prior to the recognition of microcystins as possible promoters of endemic primary liver cancer, the connection to poor quality surface water for drinking was observed and programmes for construction of deep wells were begun. At present, 80 per cent of the population in some of the afflicted regions have changed their water source to deep well water, and the incidence of liver cancer has dropped consistently.

The mottoes for prevention of primary liver cancer now are:

"control of water - control of crops - prevention of hepatitis"

For additional discussion, see section 4.1.2.

Health impairments from cyanobacteria in recreational waters must be differentiated between the chiefly irritative symptoms caused by unknown cyanobacterial substances (as described in Box 5.2), and the more severe hazard of exposure to high concentrations of known cyanotoxins, particularly microcystins. A single guideline therefore, is not appropriate. Rather, a series of guidelines associated with incremental severity and probability of adverse effects has been defined at three levels as described below.

1. Relatively mild and/or low probabilities of adverse health effects

For protection from health outcomes not due to cyanotoxin toxicity, but due to the irritative or allergenic effects of other cyanobacterial compounds, a guideline level of 20,000 cyanobacterial cells per ml (corresponding to 10 µg l^{-1} of chlorophyll *a* under conditions of cyanobacterial dominance) can be derived from the prospective epidemiological study by Pilotto *et al.* (1997) (see Box 5.2). Whereas the health outcomes reported in this study were related to cyanobacterial density and duration of exposure, they affected less than 30 per cent of the individuals exposed. At this cyanobacterial density, 2-4 µg l^{-1} of microcystins may be expected if microcystin-producing cyanobacteria are dominant, with 10 µg l^{-1} being possible with highly toxic blooms (regional differences in microcystin content of the cells may be substantial). This level is close to the WHO provisional drinking water guideline value of 1 µg l^{-1} for microcystin-LR (WHO, 1998) which is intended to be safe for life-long consumption. Thus, health outcomes due to microcystin are unlikely and providing information for visitors to bathing sites with this low-level risk is considered to be sufficient. Additionally, it is recommended that the authorities are informed in order to initiate further surveillance of the site.

2. *Moderate probability of adverse health effects*

At higher concentrations of cyanobacterial cells, the probability of irritative symptoms is elevated. Additionally, cyanotoxins (usually cell-bound) may reach concentrations with potential health impact. To assess risk under these circumstances the data used for the drinking water provisional guideline value may be applied. Swimmers involuntarily swallow some water while bathing and the harm from ingestion of bathing water will be comparable with that from a drinking water supply with the same toxin content. A swimmer can expect to ingest up to 100-200 ml of water in one session, sail-board riders and water skiers would probably ingest more.

A density of 100,000 cyanobacterial cells per ml (which is equivalent to approximately 50 µg l^{-1} of chlorophyll *a* if cyanobacteria dominate) is a guideline for a moderate health alert in recreational waters. At this density, 20 µg l^{-1} of microcystins are likely, if the bloom consists of *Microcystis* and has an average toxin content per cell of 0.2 pg, or 0.4 µg microcystin per µg chlorophyll *a* (up to 50 µg l^{-1} of microcystin are possible) but toxin levels may approximately double if *Planktothrix agardhii* is dominant. This toxin concentration is equivalent to 20 times the WHO provisional guideline value for microcystin-LR in drinking water, but would result in consumption of an amount close to the TDI for an adult of 60 kg consuming 100 ml of water while swimming (rather than 2 litres of drinking water). However, a child of 15 kg consuming 250 ml of water during extensive playing could be exposed to 10 times the TDI. The health risk will be

increased if the person exposed is particularly susceptible (e.g. because of chronic hepatitis B). Therefore, cyanobacterial densities likely to cause microcystin concentrations of 20 µg l^{-1} should trigger further action.

Non-scum-forming species of cyanobacteria, such as *Planktothrix agardhii,* have been observed to reach cell densities corresponding to 200 µg l^{-1} of chlorophyll *a* or even more in shallow water bodies. Transparency in such situations will be less than 0.5 m when measured with a Secchi disk (see Chapter 11). *Planktothrix agardhii* has been shown to contain a very high cell content of microcystin (1-2 µg per µg chlorophyll *a)* (see Figure 3.5) and therefore toxin concentrations of 200-400 µg l^{-1} can occur without scum formation.

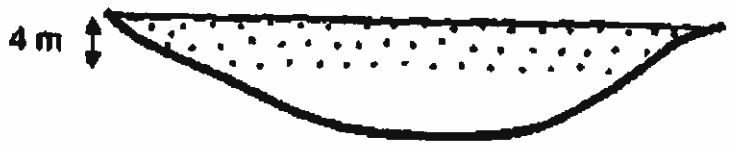
An additional reason for increased alert at 100,000 cells per ml is the potential of some frequently occurring cyanobacterial species (particularly *Microcystis* spp. and *Anabaena* spp.) to form scums (see Figure 5.1). These scums may increase local cell density and thus toxin concentration by a factor of 1,000 or more in a few hours, thus rapidly changing the risk from moderate to high (see next subsection) for bathers and others involved in body-contact water sports.

Cyanobacterial scum formation presents a unique problem for routine monitoring carried out at the usual time intervals of one or two weeks, because such monitoring intervals are unlikely to detect hazardous maxima. Because of the potential for rapid scum formation at a cyanobacterial density of 100,000 cells per ml or 50 µg l^{-1} chlorophyll *a* (from scum-forming cyanobacterial taxa), intensification of surveillance and protective measures are appropriate at these levels. Daily inspection for scum formation (if scum-forming taxa are present) and measures to prevent exposure in areas prone to scum formation are the two main options.

Intervention is recommended to trigger effective public information campaigns educating people on avoidance of scum contact. Furthermore, in some cases (e.g. with frequent scum formation), restriction of bathing may be judged to be appropriate. An intensified monitoring programme should be implemented, particularly looking for scum accumulations. Health authorities should be notified immediately.

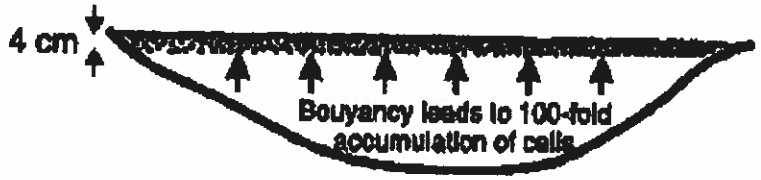
Figure 5.1 Schematic illustration of scum-forming potential changing the cyanotoxin risk from moderate to very high

Lake profile



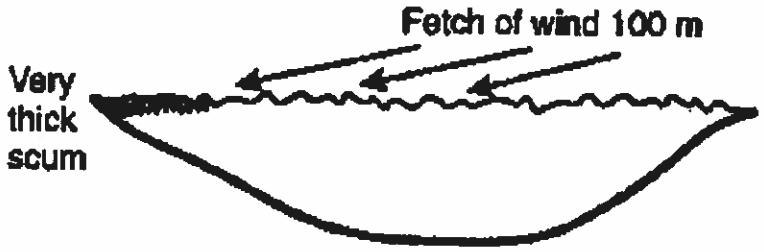
Moderate risk level:

- 50 µg l^{-1} chlorophyll *a*
- or 100,000 cells l^{-1}
- possibly 20 µg l^{-1} of microcystin in top 4 m of water body



100-fold accumulation to high risk level scum:

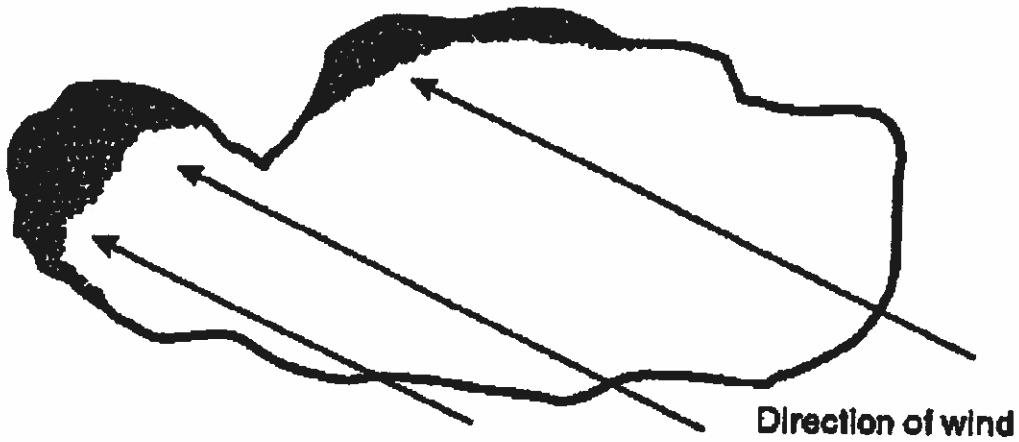
- 5,000 µg l^{-1} chlorophyll *a*
- or 10,000,000 cells l^{-1}
- possibly 2,000 µg l^{-1} of microcystin in top 4 cm of water body

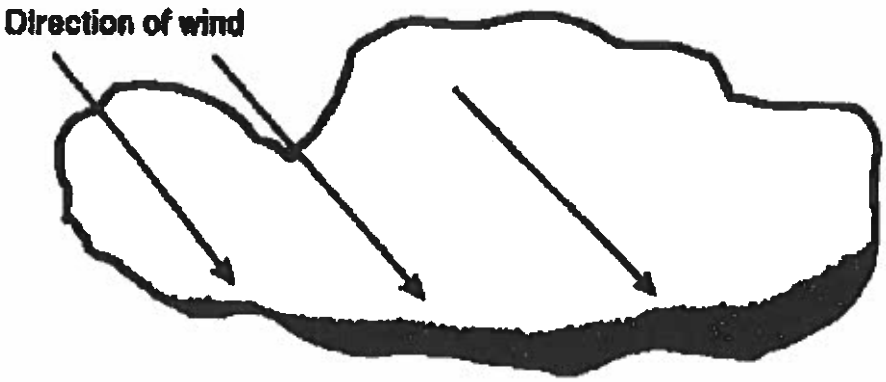


1,000-fold accumulation to very high risk level shore scum if wind sweeps scums from 100 m into 10 m:

- 50,000 µg l^{-1} chlorophyll *a*
- or 100,000,000 cells l^{-1}
- possibly 20,000 µg l^{-1} of microcystin concentrated in one bay of the water body

Lake plan





3. High risk of adverse health effects

Abundant evidence exists for potentially severe health hazards associated with scums caused by toxic cyanobacteria (see section 4.1). No human fatalities have been unequivocally associated with oral ingestion of scum, even though numerous animals have been killed by consuming water containing cyanobacterial scum material (see section 4.1). This discrepancy can be explained by the fact that animals would drink higher volumes of scum-containing water, compared with the small amounts of scum accidentally ingested by humans during bathing (resulting in a lower dose).

Cyanobacterial scums can represent a thousand-fold to million-fold concentration of cyanobacterial cell populations. It has been calculated that a child playing in a *Microcystis* scum for a protracted period and ingesting a significant volume could receive a lethal exposure, although there are no reports that this has actually occurred. Based on evidence that the oral LD_{50} of microcystin-LR in mice is 5,000-11,600 µg kg^{-1} bw (see section 4.2), for a child of 10 kg the ingestion of 2 mg of microcystin or less could be expected to cause liver injury, because concentrations of up to 24 mg l^{-1} of microcystins have been published from scum material (see section 3.2). Substantially higher enrichment of scums (up to gelatinous consistency) is occasionally observed, and accidental ingestion of smaller volumes of these could cause serious harm. Anecdotal evidence indicates that children, and even adults, may be attracted to play in scums. The presence of scums caused by cyanobacteria is a readily detected indicator of a high risk of adverse health effects for those bathers who come into contact with the scum. Immediate action to control scum contact is recommended for such situations.

The approach outlined in this section, however, does not cover all conceivable situations. Swimmers may be in contact with benthic cyanobacteria after a storm breaks off clumps of filaments, or cyanobacterial mats naturally detach from the sediment and are accumulated on the shore (Edwards *et al.*, 1992). Some marine beaches have been reported to have widespread problems due to a benthic marine cyanobacterium, *Lyngbya majuscula*, growing on rocks in tropical seas and causing severe blistering when trapped under the bathing suits of people swimming following a storm (Grauer, 1961). This response may be due to acute toxicity; *Lyngbya* can produce irritant toxins. Measures of cyanobacterial population cell density as outlined in Table 5.2, will not detect these hazards. Instead, this type of hazard calls for critical and well-informed observation of bathing sites, coupled with a flexible response.

It is difficult to define "safe" concentrations of cyanobacteria in recreational water in relation to allergenic effects or skin reactions, because individual sensitivities vary greatly. Aggravation of dermal reactions due to accumulation of cyanobacterial material and enhanced disruption of cells under bathing suits and wet suits may be a problem, even at all densities below the guidelines described above. Further information related to monitoring and management of recreational waters is available in Bartram and Rees (1999).

5.3 Other exposure routes

5.3.1 Renal dialysis

Renal dialysis patients are at great risk when water used for dialysis contains contaminants such as cyanotoxins. For these patients large volumes of water (120 litres) are used and the route of exposure is similar to the i.v. route, which allows for a much greater uptake of toxin than following oral ingestion. One serious incident, including a number of deaths arising from exposure through this route, has already been described in section 4.1.

The WHO *Guidelines for Drinking-water Quality* (WHO, 1993) do not consider the especially high quality of water needed for dialysis treatment, intravenous therapy or other clinical uses. The treatment processes used at conventional surface water treatment plants (such as coagulation, clarification and sand filtration) are normally effective in removing cyanobacterial cells, but are not effective in removing or destroying dissolved cyanotoxins, especially from water supplies with a high organic content and cyanobacterial dominance (see Chapter 9). Consequently, clinics and hospitals with special water needs, such as for dialysis treatment or for transfusions (intravenous administration), may need to provide additional water treatment to remove the cyanotoxins. Such treatment ranges from granular activated carbon filtration, followed by reverse osmosis, to more elaborate treatment including membrane filtration (e.g. 25 µm pore size filter). The extent of treatment necessary depends on the quality of the municipal water supply.

Continuous monitoring of performance and equipment is essential to ensure adequate quality of the water. On-site water treatment systems in clinics and hospitals require rigorous monitoring and regular maintenance, including back-flushing of filters and recharge of activated carbon, according to manufacturers' specifications. It is important that manufacturers' specifications should be assessed for their adequacy for maintaining performance under local conditions. Activated carbon, for example, may be exhausted for its ability to remove cyanotoxins long before it reaches saturation for removal of other organic compounds, and some manufacturers may be unaware of this.