Chapter 13: Cyanobacterial toxin removal in drinking water treatment processes and recreational waters

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Abstract

Although federal drinking water regulations determine the quality of potable water, many specifics influence how each utility chooses to treatment water. Some of the specifics include source water quality, storage capacity, existing unit process, and space. An overview of the US recreational and drinking water regulations were discussed in context of cyanobacterial toxin removal and inactivation by ancillary as well as auxiliary treatment practices. Ancillary practice refers to the removal or inactivation of algal toxins by standard daily operational procedures where auxiliary treatment practice refers to intentional treatment. An example of auxiliary treatment would be the addition of powder activated carbon to remove taste and odor compounds. The implementation of new technologies as such ultraviolet disinfection and membrane filtration, to meet current and purposed regulations, can greatly affect the algal toxin removal and inactivation efficiencies. A discussion on meeting the current regulations by altering chemical disinfection, ozone, chlorine, chloramines and chlorine dioxide included their ancillary effects on the protection against algal toxins. Although much of the research has been on the efficiency of the removal and inactivation of microcystin LR and several microcystin variants, the discussion included other algal toxins: anatoxin–a, saxitoxins, and cyclindrospermopsin.

Introduction

Before the United States Environmental Protection Agency (USEPA) can develop guidelines or regulations concerning the permissible concentrations of algal toxins for drinking water, they must first determine if cyanoalgal toxins can be removed or inactivated through common drinking water treatment practices. In 2000, a group of experts, through a USEPA initiative, created a cyanoalgal toxin priority list that contained the microcystins, anatoxin–a, and cylindrospermopsin. Microcystins are cyclic peptide hepatotoxins with a conserved (2S, 3S, 8S, 9S)–3–amino–9–methoxy–2,6,8–trimethyl–10–phenyl–deca–4,6–dioc acid, frequently abbreviated as Adda, and variable amino acids. Since there are over 70 variants of microcystins, a short list of four variants, LR, YR, LA and RR, referring to the changes in the amino acids, was recommended based on prevalence and toxicity. Cyanoalgal toxins can be either found inside of the cell, "intracellular", or outside the cell, "extracellular". The efficiency of a drinking water unit process with respect to the removal of cyanoalgal toxins depends on the total concentration of the algal toxins, the form of the toxin, and whether it is intracellular or extracellular. Commonly, dissolved contaminants such as extracellular cyanoalgal toxins are most costly to remove because conventional treatment (flocculation, coagulation, sedimentation and filtration) is usually not effective and advanced treatment processes must be implemented unless the contaminant is oxidized through disinfection. This paper reviews the literature on cyanotoxin removal during drinking water treatment with the initial focus on the removal of extracellular toxins followed by the removal of intracellular toxins. Published treatment reviews include Yoo (1995), Chorus (1999), Westrick (2003), Svrcek (2004), and Newcombe (2004).

With more stringent drinking water regulations and decreases in surface water quality, drinking water treatment trains have become more complex with fewer utilities using conventional treatment (coagulation/sediment/filtration/chlorination) alone. New additions have included more traditional unit processes like powered activated carbon (PAC), pretreatment with oxidants other than chlorine, ballasted flocculation, as well as advanced treatment processes such as membrane filtration, ultraviolet photolysis, granular activated carbon (GAC) absorbers and ozone. The traditional oxidant/disinfectant, chlorine, has been replaced with, or supplemented by, ozone, potassium permanganate, chlorine dioxide, or chloramines to eliminate nuisance organisms (e.g., zebra mussels) from the intake, compounds that cause taste and odor problems, and more recently compounds that react with chlorine disinfectant to produce toxic chlorinated by products.

Extracellular Cyanotoxin Inactivation by Chemical Disinfection Processes

Usually the primary focus of the disinfection process is to inactivate pathogenic organisms. The disinfection process can also be used to degrade several nuisance compounds such as those that cause taste and odor problems and a few regulated organic contaminants such as, some pesticides and fungicides, and industrial waste products. However, the disinfectant may also react with organic compounds in the water to produce toxic or carcinogenic byproducts. With health risk concerns about the exposure to chlorinated disinfection byproducts; the traditional chlorination process is being replaced by or supplemented with chloramination, chlorine oxide, ozone, and UV disinfection. This section focuses on the effectiveness of disinfection oxidants at inactivating important cyanotoxins.

Commonly, disinfectant effectiveness is expressed in terms of the product of the disinfectant concentration (C) in mgL^{-1} and the contact time (T) in minutes; hence CT values are in units of mgL^{-1}min. The effectiveness of a disinfectant depends primarily on the water pH, the water temperature, the concentration of compounds in the water that can react with the disinfectant, and the target organism itself. Thus, CT values for a particular disinfectant will also vary according to these factors. Several federal regulations incorporate CT values to guide the inactivation of both microorganisms and chemical contaminants. An example is Table 1, which shows the CT values needed with chorine at different water temperatures and pH values to reduce the level of the protozoan pathogen *Giardia lamblia* by 99.9%. As the temperature increases, the CT values for 99.9% inactivation of *G. lamblia* decrease. On the other hand, as pH increases, the CT values also increase. Since many utilities use these tables to determine which disinfectant dose to use, it is relevant to compare CT values needed to control *G. lamblia* with those needed to control the most common cyanotoxin, microcystin.

The CT values for the inactivation of 10 ug/L and 50 ug/L microcystin LR (MCYLR) for a batch reactor are presented in Table 2 (Acero 2005). When comparing Tables 1 and 2, at pH values of 6 and 7, the chlorine CT values needed for 99.9% inactivation of *G. lamblia* would also decrease 50 ug/L and 10 ug/L microcystin below the World Health Organization's drinking water guideline of 1 ug/L MCYLR , except perhaps at warm water temperatures. However, at a pH above 8, several of the CT values associated with the initial doses of 50 ug/L and 10 ug/L are much larger than those for 99.9% inactivation of *G. lamblia*. The larger CT values for both *G. lamblia* and microcystin inactivation at pH 8 and 9 reflect the change of

the equilibrium from a strong oxidant, hypochlorous acid, to a weak oxidant, hypochlorite ion. Determining chlorine CT values for the inactivation of MCYLR gives the utilities a familiar tool which can easily be incorporated into treatment practices.

Two recent publications (Acero 2005 and Ho 2005) proposed chlorine inactivation mechanisms for several microcystins. Acero reported that MCYLR, microcystin RR (MCYRR) and microcystin YR (MCYYR) react with chlorine at a similar rate, suggesting that hydroxylation of the conversed Adda moiety is the likely site of deactivation. In contrast, Ho and coworkers reported that the reaction rates with chlorine for the four MCYs they studied differed, where MCYYR>MCYRR>MCYLR>MCYLA, suggesting the oxidation of various amino acids was the most important mechanism of deactivation. Regardless of the mechanism, both reports suggested that chlorination is an effective treatment for destroying the studied microcystins.

Table 1. Chlorine CT values for 99.9% (3-log) inactivation of G. lamblia cysts. Modified version (EPA Guidance Manual, 2003)

pH	CT values ($mgL^{-1}min$)			
	10°C	15°C	20°C	25°C
6	87	58	44	29
7	124	93	62	41
8	182	122	91	61
9	265	177	132	88

Table 2. Chlorine CT values for reducing microcystin concentration to 1 ugL-1 for a batch reactor.

pH	MCYLR	CT values ($mgL^{-1}min$)			
	(ug/L)	10°C	15°C	20°C	25°C
6	50	46.6	40.2	34.8	30.8
	10	27.4	23.6	20.5	17.8
7	50	67.7	58.4	50.6	44.0
	10	39.8	34.4	29.8	25.9
8	50	187.1	161.3	139.8	121.8
	10	110.3	94.9	82.8	71.7
9	50	617.2	526.0	458.6	399.1
	10	363.3	309.6	269.8	234.9

The inactivation of saxitoxins, cylindrospermopsin and anatoxin–a by chlorination has not been studied as thoroughly as the degradation of the microcystins by chlorination. Recent work suggests that the degradation of saxitoxins by chlorine is also pH dependent; however, in contrast with

the microcystins, higher pH values increase the inactivation rate of saxitoxin and thus decrease the CT values needed for inactivation. At pH 9, a CT value of 15 mgL^{-1}min (chlorine residual of 0.5 mgL^{-1} for 30 minutes) degraded the five studied saxitoxins by 90% (Nicholson, 2003). The saxitoxins order of reactivity towards chlorine was as follows, with the most toxic saxitoxin being the most susceptible:

GTX5=dcSTX>STX>GTX3=C2>GTX2

Cylindrospermopsin is very susceptible to oxidation by chlorine when the free chlorine residual is above 0.5 mgL-1 with a 30 minute contact time, CT values 15 mg min/L and at a pH above 6 (Senogles 2000), implying that cylindrospermopsin is more susceptible to chlorination than microcystins. These researchers suggested that the more effective degradation of both saxitoxins and cylindrospermopsin at higher pH values is probably due to both the saxitoxins and cyclindrospermopsin being in the unprotonated form. Anatoxin–a is not degraded by chlorination (Carlile 1994). Thus, different cyanotoxins react differently to chlorine. When a cocktail of cyanotoxins is present in a drinking water source, it is important to note that cylindrospermopsin and microcystin are more effectively inactivated at lower pH values, while saxitoxins are more effectively inactivated at higher pH values and anatoxin–a may not be degraded appreciably.

The toxicities of chlorination byproducts of microcystins, nodularin, saxitoxin and cylindrospermopsin have been examined. Microcystin and nodularin byproducts are not toxic by acute toxicity tests such as the mouse bioassay (Nicholson 1994), protein phosphatase inhibition assay, and mutagenicity (Tsuji 1997). Similarly, the byproducts from the chlorination of saxitoxins have been tested by mouse bioassay and have found not to be acutely toxic (Newcombe 2002a and Nicholson 2003). In contrast, the chlorination byproducts of cylindrospermopsin produce a low level of liver damage and genotoxicity (Shaw 2001). An oral 90 and 170 day toxicity study of chlorinated solutions with microcystin, saxitoxin and cylindrospermopsin showed that only cylindrospermopsin byproducts produced ill effects, i.e. fatty vacuolations of the liver in mouse (Senogles–Derham 2003). Chronic testing has not been performed for any of the algal toxins.

Other chlorine disinfection processes such as chloramines and chlorine dioxide have shown little promise towards degrading cyanotoxins. Chloramine doses, as high as 20 mgL^{-1} with 2 day exposures, did not degrade microcystins (Nicholson 1994). Although chlorine dioxide is used in several plants to remove taste and odor compounds, it is not recommended for treatment of microcystins since the dosage would not be cost

effective (Kull 2004). Although one might speculate that cylindrospermopsin, anatoxin–a, and saxitoxins are not susceptible to chloramine and chlorine dioxide treatment, studies are needed to validate this statement.

Ozone is an effective disinfectant that oxidizes organic compounds either directly as molecular ozone or indirectly through a hydroxyl radical. Microcystin and anatoxin–a are inactivated at ozone doses where a residual is maintained for several minutes, CT values of 0.1 ugL-1min and 0.3 ugL-1min, respectively. (Keijola 1988, Himberg 1989, Bruchet 1998, Hart 1998, Rositano 1998, 2001, Newcombe 2002b). An acute toxicity screen was negative for microcystin ozone byproducts. The saxitoxin family appears to have low to moderate susceptibility to ozone oxidation, with a CT value of 6.9 mg/L min not being effective (Rositano 2001, Newcombe 2002b). The author of this review speculates that ozone would be an effective treatment to inactivate cylindrospermopsin because ozone is highly reactive toward unsaturated bonds. The confounding factors of ozone treatment for cyanoalgal toxins are 1) oxidation of the dissolved carbon competes with the destruction of algal toxins and 2) sensitive to alkalinity and temperature.

Extracelluar Cyanotoxin Removal by Activated Carbon Adsorption

Two types of activated carbon are used in the drinking water industry: powdered activated carbon (PAC) and granular activated carbon (GAC). Common sources of activated carbon are coal, coconut, and wood. Several studies suggest that mesoporous (i.e., pore diameters between 2–50 nm), wood–based activated carbon is more effective at removing cyanotoxins than other types of activated carbon. PAC is usually used seasonally to remove taste and odor compounds and agricultural chemicals, or as an emergency barrier during industrial/commercial spills. Drinking water utilities can test the relative effectiveness of various PAC types for removing a contaminant(s) of concern by performing jar tests, and then purchase the carbon with the most capacity for their target contaminants. A series of jar tests suggest different microcystins have different adsorption efficiencies:

RR>YR>LR>LA (Cook 2002)

The efficiency of saxitoxins removal by PAC mirrors the order of toxicity, with saxitoxin STX being the most toxic:

STX>GTX>C (Newcombe 2004)

Very little work has been performed on the adsorption of anatoxin–a and cylindrospermopsin to PAC. More systematic studies are needed to determine which PAC type, dosage, and contact time are most appropriate. Although more research is needed to guide drinking water utilities on the effectiveness of PAC for anatoxin–a, cylindrospermopsin and mixtures of cyanotoxins, the author of this review still stresses the importance of site specificity and in–house jar testing to select the most effective activated carbon.

GAC can be used in filter beds along with other filter media or can be used as a stand–alone fixed bed adsorber. GAC filters are used primarily to remove toxic chemicals and occasionally to remove substances in the water that cause taste and odor problems. They lose their organic compound adsorption capabilities usually within a couple of months of use and any organic removal after that comes from biological activity on the filter. Commonly, the effectiveness and degradation of a GAC filter is measured by total organic carbon (TOC) in the filter effluent. When 70–80% of the TOC entering the filter is measured in the filter effluent, usually after at least three months of service, the GAC media is replaced or reactivated. Newcombe and co–workers (2002b) reported microcystin in the GAC effluent at 80% TOC breakthrough suggesting that GAC filters would not be an effective barrier for controlling microcystin, since GAC media is not replaced monthly. However, frequently GAC absorbers are designed to last several months before 80% TOC breakthrough occurs, and since the adsorber media is replace upon 80% TOC breakthrough, GAC adsorbers may be an effective microcystin and other algal toxin barrier. Newcombe and coworkers (2002b) also studied the effect on cyanotoxins of ozonating the water before GAC adsorber entry, using two unit processes as one barrier. This barrier will oxidize microcystin, cylindrospermopsin and anatoxin–a, and the GAC absorber would provide an additional barrier to saxitoxins not oxidized by this treatment. To the best of the author's knowledge, the removal of cylindrospermopsin and anatoxin–a by GAC adsorption has not been investigated.

Extracellular Cyanotoxin Inactivation by Advanced Drinking Water Treatment Processes

The absorbance of ultraviolet (UV) energy can break molecular bonds without chemical addition and is used to inactivate many pathogens in drinking water. Normally, the UV treatment process uses a low to medium pressure lamp with wavelengths between 200 and 300 nm. Commonly, in

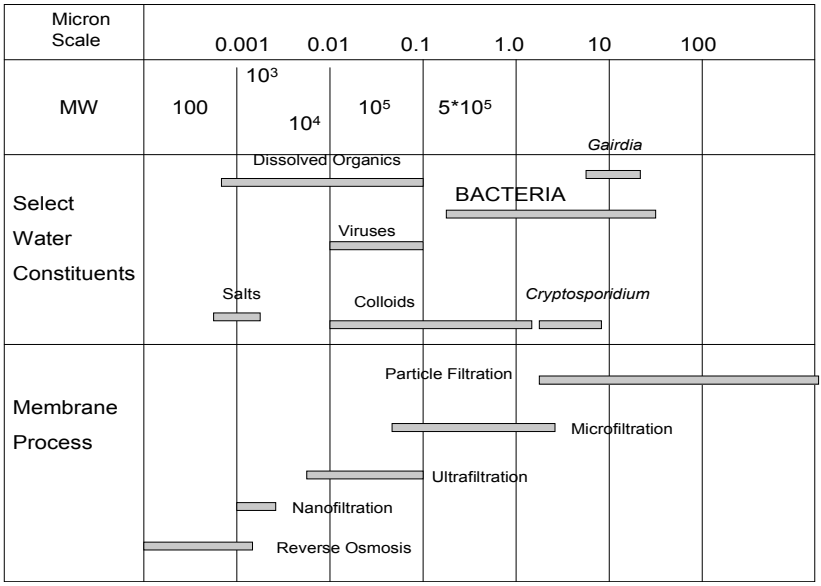
the water treatment industry, the UV dose is expressed in milli–joules/cm^2 (mJ/cm^2). A dose of 40 mJ/cm^2 for the inactivation of *Crytosporidium parvum* oocysts is considered economically feasible. Much work has been done on the photolytic destruction of microcystins, cylindrospermopsin, and anatoxin–a, but the UV doses that effectively degrade microcystin, cylindrospermopsin, and anatoxin–a range from 1530 to 20,000 mJ/cm2 (Tsuji 1995, Chorus 1999, Senogles 2000), or several orders of magnitude higher than that needed for *Cryptosporidium* oocyst. Thus, UV irradiation is not considered economically feasible for cyanotoxin degradation.

The use of titanium dioxide and UV photolysis together successfully degrades MCYLR and cylindrospermopsin (Lawton 1999, Feitz 1999, Cornish 2000, Senogles 2001, Sherpard 2002). Although this is not a cost efficient treatment process for utilities, it may be viable in point–of–use treatment (e.g., devices used in the home). The combination of hydrogen peroxide with either ozone or UV irradiation enhances the efficiency of microcystin destruction (Rositano 1998, Qiao 2005, respectively). With hydrogen peroxide at 0.1 mgL^{-1} and ozone at 0.2 mgL^{-1}, 1 mgL^{-1} of MCYLR was completely removed in 30 minutes (CT value of 6 $mgL^{-1}min$). However, more research on these combinations for the degradation of cyanotoxins is needed to assess their feasibility.

Extracelluar and Intracellular Cyanotoxin Removal by Filtration (other than GAC)

Semi–permeable membranes such as those used in reverse osmosis filters remove many contaminants from water, especially those whose size is larger than the membrane pores. Advances in membrane technology have made this process more versatile, dependable and economically feasible as a drinking water treatment process. Reverse osmosis and nanofiltration membranes remove all pathogens and also large molecules above the membrane pore size (100 Daltons for reverse osmosis, 200 Daltons for nanofilters). However, the membranes are not necessarily foolproof. When concentrations of the removed substance become sufficiently high traces of inorganic and organic compounds may pass through the membrane barrier. A reverse osmosis membrane study using between 10 ugL^{-1} and 130 ugL^{-1} of MCY LR, RR and nodularin removed greater than 95% of the toxin from waters with a range of salinities (Neumann 1998, Vouri 1997). A water with high conductivity such as brackish water may allow slightly greater breakthrough of cyanotoxins than otherwise (Vuori 1997).

Several nanofiltration studies report from 82% to complete microcystin removal (Fawell 1993, Muntisov 1996, Simpson 2002, Smith 2002).
Microfiltration and ultrafiltration use membranes with greater pore sizes than the membranes used in reverse osmosis and nanofiltration. Ultrafiltration is capable of removing intact bacteria (including the cyanobacteria), but not all dissolved organic compounds (Fig. 1). Microfiltration, with its larger pore size, may not remove all intact bacteria and is not effective in removing dissolved organic compounds. Since 50% to 95% of the cyanotoxin is intracellular, ultrafiltration and (to a degree) microfiltration are effective in removing intracellular cyanotoxins in drinking water supplies. Studies (Chow 1997, and Zhou 2001) suggest that both microfiltration and ultrafiltration either as a stand–alone treatment process or as a replacement to conventional filtration are excellent at removing intact cyanobacteria and their intracellular toxins. With regard to conventional water filtration (coagulation, sedimentation, and then filtration), a bench – top jar test apparatus and a full –scale pilot plant resulted in 70% and 99.9% removal, respectively, of *Microcystis* cells (Drikas 2001). This study suggests that conventional treatment is effective in removing intracellular cyanotoxins. These studies reported very little cell breakage during the cell removal process, implying that the process did not cause significant intracellular cyanotoxin release.



F**ig. 1.** A summary of membrane processes and their filtration characteristics. Modified version (Tech Brief 1999)

Through Plant Removal and Inactivation of Cyanotoxins: A Multi–barrier Approach

Toxin–producing cyanobacteria are abundant in surface waters and so they impose a degree of risk to drinking water utilities. The toxicity of microcystin LR has prompted the World Health Organization to publish a guideline value of 1.0 ug MCYLR/L for drinking water. Several studies have investigated the concentration of cyanotoxins in drinking water treatment plants. The most recent studies have been done by Carmichael (2001), Karner (2001), Hoeger (2005), and Wood (2005).

During two year study, 1996 to 1997, Carmichael (2001) measured toxins in source and finished (i.e., filtered) waters in 10 utilities in North America. Of the 677 samples tested, 539 (80%) were positive for microcystins when tested using the enzyme linked immunosorbent assay (ELISA). Of the positive samples, 4.3% were higher than the 1 ug/L WHO guideline. Only two finished water samples were above 1 ugL^{-1}, suggesting that during the study many of the utilities were adequately removing or inactivating microcystins.

Karner's (2001) study evaluated source and treated water from five Wisconsin drinking water plants with four plants located on one lake. The reported total concentration of microcystin (intracellular and extracellular toxin) was determined by ELISA. The source waters ranged from tenths of ugL^{-1} to 7 ugL^{-1}. The five plants demonstrated 1-3 log removal of microcystin. This study suggested that pretreatment alone (i.e., before filtration) with potassium permanganate, copper sulfate, and PAC reduced algal toxins as much as 61%. Karner reported the percent of removal, but did not mention the initial microcystin concentrations. There may be several site specific reasons that pretreatment worked for these plants; two of the utilities have at least 5.5 day water detention times before being distributed to the public and another plant used a lower dose of $KMnO_4$. These conditions could have played an important role in achieving the 61% decrease of microcystin. This study stresses the uniqueness of individual treatment trains.

Hoeger's study investigated microcystin removal through two drinking water plants. One plant had pre–ozonation (1.0 mgL^{-1}), rapid sand filtration, intermediate ozonation (0.5 mgL^{-1}), GAC filtration follow by slow sand filtration and the other had conventional treatment followed by chlorination. The first plant was challenged with a microcystin concentration of approximately 8.0 MCY ugL^{-1} where as the conventional plant was challenged with a concentration of 0.2 MCY ugL^{-1}. Both treatment plants removed microcystin below the WHO guideline.

During the summer of 2005, Wood and coworkers (2005) studied cyanobacteria and algal distribution as well as microcystin concentrations in source and finished waters from five utilities in Oklahoma, Vermont, Texas, California and Florida. They found that several of the source waters had low levels of microcystin from 0.2 ugL^{-1} MCY to 0.5 ugL^{-1} MCY and none of the finished waters were above 0.05 ugL^{-1} MCY, as determined by ELISA. This study also showed that typical conventional treatment removed from 2 to 5 log of algal units mL^{-1} (Fig. 2) and finished water commonly contained less than 2 algal units mL^{-1}.

New Technologies

Electrochemical degradation was investigated as a new drinking water technology to degrade extracellular microcystin LR through a hydroxyl radical mechanism similar to other advanced oxidation processes (Feng 2005). At an applied current of 100mA, the degradation rate was 0.219 min^{-1} and the half–life was 2.5 min. This experiment suggested that the electrochemical oxidation may be a promising approach for microcystin degradation.

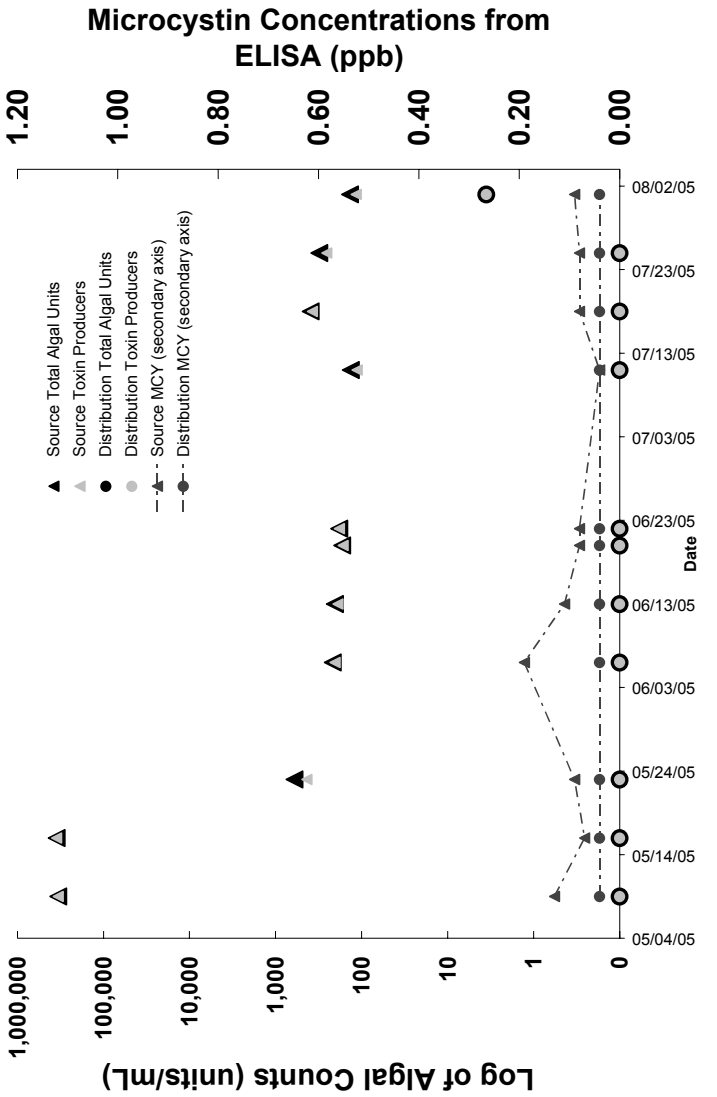


Fig. 2. This graph depicts the removal of intact algal cells and microcystin by conventional treatment.

Conclusion

Drinking water utility managers have several source water and treatment options for removing and inactivating both intracellular and extracellular cyanotoxins. The first consideration should be to optimize the effectiveness of existing processes. Intracellular cyanotoxins can be removed effectively by conventional treatment and membrane filtration. Chloramination and UV treatment are not effective for inactivating extracellular algal toxins. Although several oxidants inactivate some cyanotoxins, it is important to remember that not one oxidant inactivates all cyanotoxins. Chlorine effectively degrades extracellular cyanotoxins both microcystins and cylindrospermopsin between pH 6.0 and 8.0, and saxitoxins at pH values at 9 and higher. Utilities that want to determine the risk of cyanotoxins to their system may use a simple and rapid procedure published by the World Health Organization (Chorus 1999).

References

Acero JL, Rodriguez, E, Meriluoto, J (2005) Kinetics of reaction between chlorine and the cyanobacterial toxin microcystins. Water Research 39:1628–1638.

Bruchet A, Bernazeau F, Baudin I, Peironne P (1998) Algal toxins in surface water analysis and treatment. Water Supply 16 (1–2):619–623.

Carmicheal WW (2001) Assessment of Blue–Green Algal Toxins in Raw and Finished Drinking Water. AWWA Research Foundation

Carlile PR (1994) Further Studies to Investigate Microcystin–LR and Anatoxin–a Removal from Water. Report No. 0458, Foundation of Research, UK.

Chorus I and Bartram J (1999) Toxic Cyanbacteria in Water: A Guide to their Public Health Consequences, Monitoring and Management. London, UK: E & FN Spon.

Chow C, Drikas M, House J, Burch M, Velzeboer R, Gimbel R (1997) A study of membrane filtration for the removal of cyanobacterial cells AQUA 46(6):324–334.

Cook D and Newcombe G (2002) Removal of microcystin variants with powdered activated carbon. Water Science Techonology:Water Supply, 2 (5–6):201–207.

Cornish IT, Lawton LA, Robertson P (2000) Hydrogen peroxide enhanced photocatalytic oxidation of microcystin–LR using titanium dioxide. Applied Catalyst B: Environmental 25:481–485.

Drikas, M, Chow CWK, House J, Burch MD (2001) Using coagulation, flocculation, and settling to remove toxic cyanobacteria. Journal of American Water Works Association 93:2:100.

EPA Guidance Manual (May 2003) Appendix A. Glossary. LT1ESWTR Disinfection Profiling and Benchmarking. 103

Fawell J, Hart J, James H, Parr W (1993) Blue–green algae and their toxins: analysis, treatment, and environmental Control. Water Supply 11(3–4) 243–249.

Feng C, Sugiura N, Masaoka Y, Masaoka T (2005) Electrochemical Degradation of Microcystin–LR. Journal of Environmental Science and Health A40:453–465.

Fritz AJ, Waite TD, Jones GJ, Boyden BH, Orr PT (1999) Photocatalytic degradation of the blue green algal toxin microcystin–LR in a natural organic–aqueous matrix. 33(2):243–249.

Hart J, Fawell J, Croll B (1998) The fate of bath intra and extracellular toxins during drinking water treatment. Water Supply 16(1–2):611–616.

Himberg K, Keijola A, Hiisvirta L, Pyysalo H, Sivonen (1989) The effect of Water Treatment Processes on the removal of hepatotoxins from *Microcystis* and *Oscillatoria* Cyanobacteria: A Laboratory Study. Water Research 23 (8): 979–984

Ho L, Newcombe G, Rinck–Pfeiffer (2005) submitted to Water Research

Hoeger SJ, Hitzfield BC, and Dietrich DR (2005) Occurene and elimination of cyanobacterial toxins in drinking water treatment plants. Toxicology and Applied Pharmacology 203 231–242.

Keijola A, Himberg K, Esala A, Sivonen K, Kiisvirata L (1988) Removal of cyaonbacterail toxins in water treatment processes: laboratory and pilot plant experiments. Toxicological Assessment 3:643–656.

Kull T, Backlund PH, Karlsson KM, Meriluoto JA (2004) Oxidation of the Cyanobacterial Hepatotoxin Microcystin–LR by Chlorine Dioxide: Reaction Kinetics, Characterization, and Toxicity of Reaction Products. Environmental Science Technology 38:6025–6031.

Lawton LA, Robertson, PK (1999) Physio–chemical treatment methods for the removal of microcystins (cyanobacterial hepatotoxins) from potable waters. Chemical Society Review 28(4):217–224.

Muntisov M and Tromboli P (1996) Removal of algal toxins using membrane technology. Water Journal Australian Water Association 23(3):34.

Neumann, U and Wecknesser J (1998) Elimination of microcystin peptide toxins from water by reverse osmosis. Envirnomential Toxicology Water Quality 13(2):143–148.

Newcombe, G and Nicholson BC (2002a) Treatment options for the saxitoxins class of cynatoxins. Water Science and Technology:Water Supply 2(5–6):271–275.

Newcombe G (2002b) Removal of Algal Toxins from Drinking Water Using Ozone and GAC. Denver, Colorado: American Water Works Research Foundation.

Newcombe G and Nicholson B (2004) Water treatment options for dissolved cyanotoxins. Journal of Water Supply and Technology–AQUA 52(4):227–239.

Nicholson BC, Rositano J, Burch MD (1994) Destruction of cyanobacterial peptide heptatotoxins by chlorine and chloramines. Water Research 28:1297–1303.

Nicholson BC, Shaw GR, Morrell J, Senogles PJ, Woods TA, Papageorgiou C, Kapralos C, Wickramasinghe W, Davis BC, Eaglesham GK, Moore MR (2003) Chlorination for degrading saxitoxins (paralytic shellfish poison) in water. Environmental Technology 24:1341–1348.

Qiao RP, Li N, Qi XH, Wang QS, Zhuang YY (2005) Degradation of microcystin–RR by UV radiation in the presence of hydrogen peroxide. Toxin 45(6);745–752.

Rositano J, Nicholson B, and Pieronne P (1998) Destruction of cyanobacterial toxins by ozone. Ozone Science Engineering 20:223–238.

Rositano J, Newcombe G, Nicholson B, Sztajnbok P (2001) Ozonation of NOM and alglal toxins in four treated waters. Water Research 36(1):23–32.

Senogles P, Shaw G, Smith M., Norris R., Chiswell R., Mueller J, Sadler R. Eaglesham (2000) Degradation of the cyanobacterial toxin cylindrospermopsin from Cylindrospermopsis raciborskii, by chlorination. Toxin 38:1203–1213.

Senogles PJ, Smith M, Shaw G (2001) Photocatalytic degradation of the cyanotoxin cylindrospermopsin, using titanium dioxide and UV radiation. Water Research 35(5):1245–1255.

Senogles–Derham PJ, Seawright A, Shaw G, Wickramisingh W, Shahin M (2003) Toxicological aspects of treatment to remove cyanobacterial toxins from drinking water determined using the heterozygous P53 transgenic mouse model. Toxicon 41(8) 979–988.

Shaw GR, Shen X, Wickramasinghe W, Senogles P, Easglesham GK, Lam PKS, Moore MR (2001) Toxicological aspects of byproducts of chlorination of the cyanobacterial toxin, cylindrospermopsin. Toxicology 164(1–3):174.

Shephard MR, Stockenstrom S, de Villiers D, Engelbracht WJ, Wessel GFS (2002) Degradation of microcystin toxins in a falling fim photocatalytic reactor with immobilized titanium dioxide catalyst. Water Research 36(1):140–146.

Simpson MR and MacLeod BW (2002) An integrated approach of algal by–products including bench scale evaluation of nanofiltration for microcystin removal. Proceedings of the AWWA 2002 Water Quality Technology Conference Seattle Washington 10–14 November.

Smith DP, Falls V, Levine AD, McLeod BW, Simpson M, Champlin TL (2002) Nanofiltration to augement conventional treatment for removal of algal toxins, taste and odor compounds, and natural organic matter. Proceedings of the AWWA 2002 Water Quality Technology Conference Seattle Washington 10–14 November.

Svrcek, C. and Smith, D.W. 2004. An Overview of the Cyanobacteria Toxins and the Current State of Knowledge on Water Treatment Options. Journal of Environmental Engineering and Science 3: 155–185.

Tech Brief. (March 1999) National Drinking Water Clearinghouse http://www.nesc.wvu.edu/ndwc/pdf/OT/TB/TB10_membrane.pdf.

Tsuji K, Watanuki T, Kondo F, Watanabe MF, Suzuki SM, Nakazawa H, Suzuki M, Uchida H, Harada KI, (1995) Stability of microcystin from cyanobacterial –II. Effect of UV light on decomposition and isomerization. Toxin 33(12):1619–1631.

Tsuji K, Watanuki T, Kondo F., Watanabe,MF, Nakazawa H, Suzuki, M. Uchida H, Harada KI (1997) Stability of microcystins from cyanbacteria –IV. Effect of chlorination on decomposition. Toxicon 35(7):1033–1041.

Vuori E, Pelander, Himberg K, Waris M, Niinivaara K (1997) Removal of nodularin from brackish water with reverse osmosis or vacuum distillation. Water Research 31(11): 2922–2924.

Westrick JA (2003) Everything a Manager Should Know about Algal Toxins but was Afraid to Ask. Journal of American Water Works Association 95:26–34.

Wood S, Abbott S, Westrick JA (2005) Update on a national preliminary algal toxin occurrence study that monitored source and distribution drinking water (presentation) Kentucky/Tennessee America Water Works Association Conference Covington KY, September 12, 2005.

Yoo RS, Carmichael WW, Hoehn RC, and Hurdey SE (1995) Cyanobacterial (Blue–Green Algal) Toxins: A Resource Guide. Denver, Colorado: American Water Works Research Foundation.

Zhou H and Smith DW (2001) Advanced technologies in water and wastewater treatment Canadian Journal of Civil Engineering 28 (Supplement 1):49–66.