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10 Control and management of Harmful Algal Blooms

10.1 Introduction

Everyone would agree that the best cure is prevention. This is certainly applicable in the case of cyanobacterial blooms and their tremendous and widespread impact on human and environmental health, natural and man-made assets, as well as overall ecosystem services. There have even been suggestions and also some evidence that cyanobacteria blooms may have a negative impact on the housing markets in some parts of the world. Certainly, common sense would dictate that the level of appreciation and the livability of a neighborhood may be positively influenced by the presence of a nice lake (natural or artificial); however, the opposite is true when that lake is affected by water quality issues including odor and toxins from the development of algal blooms. Prevention of cyanobacterial blooms has been at the heart of the nutrient management strategies around the world and has resulted in many success stories. However, blooms still occur and they also occur in assets that the public are not necessarily exposed to, such as drinking water reservoirs, wastewater stabilization ponds and retention dams, in addition to natural systems such as lakes and rivers. When prevention fails or less than satisfactory results are achieved, we need to tackle the problem through direct mitigation technologies that aim at reducing or eliminating the risk generated by the presence of cyanobacteria and their toxins. In this chapter, we will discuss a broad range of mitigation approaches that have been applied with various degrees of success, and we also explore the opportunities for future development of innovative solutions for this important problem.

10.2 Global water crisis

Humans utilize water in agriculture, industry, the household and for recreation [1]. Water also provides incidental ecosystem services to humans and other organisms, such as habitats, climate control and media for nutrient cycling [2]. It is a natural resource essential to life on earth, and adequate management of water is required to maintain these vital services to the global population and the environment.

Water resources are generally not well managed, and as a result, water quality remains of significant concern. Waterways are continually polluted through the addition of nutrients and heavy metals, high-quality water is wasted on activities where it is not suited to the purpose, and water is not equally distributed between people of all nations and social status. These anthropogenic impacts on water quality and quantity have led to a crisis that is affecting the entire biosphere of the earth [1, 3, 4]. This crisis

is increasing with the concurrent rise in global population and climate change [4, 5]. In order to combat this, water resource management must be adaptive, flexible and engage with stakeholders at multiple levels, from the local to the global.

One of the most significant anthropogenic impacts on waterways is eutrophication. Eutrophication occurs when high nutrient loads enter a water body, often as a result of agricultural and industrial processes. This increase in nutrients, particularly nitrates and phosphates, can detrimentally affect ecosystems and reduce the quality of water for reuse purposes [6, 7]. Eutrophication is often a precursor to the occurrence of harmful algal blooms, which commonly contain toxin-producing cyanobacterial species and are a threat to human and environmental health [8].

10.3 Cyanobacteria and cyanotoxins

Cyanobacteria are prokaryotic phytoplanktons that occur in fresh, brackish and salt water systems throughout the world [9, 10]. Species of cyanobacteria differ in their morphology and may exist as single cells, colonies and filaments [11]. When cells aggregate, they form dense cyanobacterial blooms, a potential threat to human and environmental health.

Cyanobacteria generally dominate in reservoirs containing high nutrient loads and stagnant water, although cyanobacterial blooms do occasionally occur in oligotrophic systems and favor water temperatures between 15 and 30 °C [8]. Studies into the dynamics of cyanobacterial blooms predict that the expected increase in global temperature will result in increased surface water temperatures and thermal stratification, as well as changing meteorological patterns, possibly stimulating increased cyanobacterial growth rates [12–19]. It is likely that this will result in an increased frequency of cyanobacterial bloom events. Of particular concern to water utility managers are those cyanobacterial species that form blooms in freshwater reservoirs that are used for drinking, recreation and irrigation.

Cyanobacterial blooms have several detrimental environmental effects. Blooms often proliferate in the surface layer of stratified reservoirs, shading organisms below, which can result in the death of pelagic and benthic organisms [20–23]. When blooms collapse, the release of organic cell matter to the water column increases the system's oxygen demand. The concentration of dissolved oxygen is lowered due to its consumption in reactions to degrade organic and inorganic compounds; this results in mass deaths of fish and other aquatic organisms [10, 24]. Such deaths are often observed by the general public and receive considerable media attention.

Many species of cyanobacteria also produce toxins. Cyanobacterial toxins (cyanotoxins) vary in their toxicity to humans and animals, and include hepatotoxins, dermatoxins, cytotoxins, neurotoxins and lipopolysaccharides. Cyanotoxins can induce both acute and chronic effects, and can pose a risk to both humans and ecological systems [25–32].

The most common routes of human contact with cyanotoxins are through the contamination of drinking water, the recreational use of lakes and rivers containing cyanobacteria and via the ingestion of blue-green algal supplements [33–37]. Organisms within the environment are often harmed by direct exposure to cyanotoxins or through bioaccumulation [38–48]. Bioaccumulation can lead to the magnification of cyanotoxins throughout food webs, potentially altering growth patterns, grazing behavior and development, and leading to significant health risks for organisms, including humans, that predate species which have bio-accumulated cyanotoxins [29, 41, 49, 50].

The shading of underlying organisms, reduction of dissolved oxygen and bioaccumulation of cyanotoxins can lead to shifts in ecological assemblages and potentially ecosystem collapse, as well as significant threats to human health. As such, it is imperative that the risks of cyanobacterial blooms in various freshwater bodies are assessed and mitigated so that they can be appropriately managed to avoid detrimental effects.

10.4 Cyanobacterial prevention and mitigation

Many techniques for cyanobacterial bloom prevention and mitigation have been investigated (Tab. 10.1 and Tab. 10.2). Some have been applied directly in reservoir management, while others have been trialed only under laboratory conditions. The success of preventative and mitigation techniques depends upon the underlying conditions present, and the characteristics of individual water bodies must be considered when determining the most appropriate management strategies to apply.

Prevention of cyanobacterial blooms has been achieved with varying success through techniques including nutrient reduction, artificial destratification, macrophyte establishment, predation, the addition of allelopathic chemicals, ultraviolet radiation (UVR) and ultrasonication (Tab. 10.1). Nutrient reduction and destratification have shown reasonable success in large reservoirs, though most success has been where nutrient inputs can be significantly reduced and reservoirs are relatively deep.

Despite preventative attempts, often cyanobacterial blooms still occur. It is therefore imperative that mitigation measures for controlling blooms are investigated. Many such methods have been trialed in both the laboratory and field, with varying success (Tab. 10.2).

It is common practice to remove cyanobacteria using copper sulfate, chlorine or coagulants and flocculants [109], although the dynamics of the removal of cyanobacteria from wastewater by such methods has not been thoroughly investigated. These cyanobacterial removal techniques currently practiced on a large scale may be environmentally damaging and ineffective for the removal of cyanotoxins [93, 109–112]. Several of the removal methods used in drinking water treatment are highly successful where cyanobacterial and cyanotoxin concentrations are low and the water will not

be released to the environment, but are often prohibitively expensive for use in highly eutrophic systems and generally less effective in reservoirs containing high concentrations of organic matter [112, 113] (Tab. 10.2).

Tab. 10.1: Commonly used prevention strategies for cyanobacterial blooms.

	Specific comments for use	References
Allelopathic chemicals	Chemicals generally secreted by decaying organic matter. Addition of organic matter increases WSP sludge production. May be unanticipated effects on non-target organisms.	[51–64]
Destratification	Can promote growth of non-buoyant phytoplankton over cyanobacteria. Often ineffective in shallow and highly eutrophic water bodies. Generally requires electrical connection on-site.	[65–71]
Macrophytes	Interfere with WSP processes. Provide breeding grounds for mosquitos and other disease vectors.	[72–81]
Nutrient reduction	50 % of phosphorus in wastewater is from human waste and cannot be reduced. Likely that WSPs will be high in nutrients regardless of reduction measures. N:P ratio may be more important than actual phosphorus and nitrogen concentrations.	[8, 71, 82–85]
Predation	May alter WSP ecology, particularly if zooplankton are added and preferentially consume non-target phytoplankton. Consumption of cyanotoxins may result in the death of predators.	[86–91]
Ultrasonication	Only tested at reduced scales. May not be appropriate for full-scale WSPs.	[92–103]
Ultraviolet radiation	Cells in WSP are likely adapted to high UVR doses. May be practical in association with other treatment methods. Can only be used at pond inlets and outlets.	[104–108]

Tab. 10.2: Commonly used mitigation strategies for cyanobacterial blooms.

	Cyanobacterial removal	Toxin removal	Specific comments for use	References
Adsorption (naturally occurring particles)		√	Occurs naturally, but may be insufficient for complete cyanotoxin removal. Cyanotoxin variants adsorb differently. Adsorption decreases as pH increases. Often biodegradation is greater than adsorption when in contact with particles. If cyanotoxins are filtered through natural soil and adsorption is insufficient, this can endanger aquifers.	[114–123]
Adsorption (activated carbon)		√	Other organic compounds compete for adsorption-sites. May increase sludge loading.	[124–130]
Biodegradation		√	Detailed in Tab. 10.3.	Tab. 10.3
Chlorine and chlorinated compounds	√	√	Ineffective at removing microcystins at pH > 8. Phytoplankton cells, rather than cyanotoxins, may preferentially react with chlorinated compounds. Cyanotoxins may be released from cells more quickly than they can be degraded by chlorinated compounds in solution. Produces by-products dangerous to humans and the environment (e.g. trihalomethanes).	[93, 106, 131–142]
Coagulation and flocculation	√		Increases sludge loading. Generally does not affect membrane integrity, so cyanotoxins are not released. If flocs are not removed, cyanotoxins accumulate in sludge. Must consider flow environment of WSP. Increases concentration of aluminum in the environment.	[111, 143–150]

Tab. 10.2 (continued)

	Cyanobacterial removal	Toxin removal	Specific comments for use	References
Copper sulfate	√		Traditional method of cyanobacterial removal in WSPs. Releases cyanotoxins to the dissolved state, but does not subsequently degrade them. Increases copper concentration in the environment.	[110, 151–156]
Filtration and reverse osmosis	√	√	Impractical for water containing high suspended sediment loads. Most degradation in successful studies appears to be biological, except where nanofiltration is used.	[157–163]
Hydrogen peroxide	√	√	Effectiveness may be increased by presence of UVR and/or iron. Cyanotoxin degradation decreases with increasing pH. May release cyanotoxins to the dissolved state over hours/days. Often found ineffective on scales of minutes when not coupled with other methods. May affect microcystin synthesis within cells.	[129, 140, 141, 152, 164–179]
Ozone	√	√	Cost prohibitive – may preferentially react with other organic compounds. Cyanotoxin degradation decreases with increasing pH. Release of cellular organic compounds increases the ozone dose required. Requires electricity on-site for the production of ozone.	[93, 106, 111–113, 129, 132, 140, 180–182]

Tab. 10.2 (continued)

	Cyanobacterial removal	Toxin removal	Specific comments for use	References
Permanganate		√	Does not seem to be significantly affected by pH. Likely produces harmful by-products.	[93, 106, 132, 136, 140, 141, 183, 184]
Predation and biomanipulation	√	√	May alter WSP ecology, particularly if zooplankton are added and preferentially consume non-target phytoplankton. Addition of organic matter increases sludge loading.	[51–54, 56, 58–60, 86–90, 185]
Titanium dioxide			Large amounts of catalyst are required – up to mg per l – so impractical in wastewater.	[167, 186–188]
Ultrasonication	√	√	Only tested at reduced scales. May not be appropriate for full-scale WSPs. Requires electricity on-site.	[92–102]
Ultraviolet radiation	√	√	Occurs naturally, but can be enhanced by coupling with other removal methods. Can reduce cyanotoxins through photosensitized reaction with compounds including phycocyanin. Currently used to ensure the removal of coliforms at some WWTP outlets. Impractical alone where cell concentrations are high. Requires electricity on-site.	[93, 105, 106, 165, 170, 174, 181, 189–194]

Effective techniques for the mitigation of both cyanobacteria and cyanotoxins must be determined by considering the underlying properties of the water system in question, including depth, pH values, concentrations of suspended solids, and dissolved organic and inorganic compounds [195, 196]. To be successful, any mitigation approach must reduce both cyanobacteria and cyanotoxins and pose no or negligible threat to ecosystems.

10.5 Cyanobacterial management

The management of cyanobacteria in any freshwater resource must consider the entire cycle of water through catchments, water reservoirs, treatment plants and distribution systems [71]. Appropriate management of cyanobacterial blooms is imperative to reducing their negative impacts on human and ecological health, water treatment processes and income-generating activities, including tourism and property development. This is not simply about implementing prevention and mitigation strategies. It is also important to assess the potential risks associated with such blooms, so that they can be treated effectively and efficiently at the site of interest. Incorporating risk assessment into management will allow plans to be developed which minimize the costs of bloom mitigation and the potentially undesirable environmental effects of many cyanobacterial removal methods. Such plans should be developed for all freshwater resources that suffer from potentially toxic blooms. This will reduce or eliminate the undesirable consequences of cyanobacteria and cyanotoxins on both humans and ecosystems. A management plan should consist of the following actions required during four distinct time periods (Fig. 10.1):

1. Prior to bloom;
2. Hazardous bloom suspected;
3. Hazardous bloom identified;
4. Mitigation ineffective.

Barrington et al. [197] offered a detailed approach for the appropriate monitoring regime to minimize the risk of undetected or undetectable incidents. Currently the authorities rely on some form of either public reporting of an incident or some form of monitoring which is usually a response to a visual inspection of the water system (Fig. 10.2).

This form of risk assessment assumes that hazardous events will always be correctly identified. Although useful, such an approach may lead to overly cautious and cost-intensive behavior by following the precautionary principle, whereby potentially hazardous events are assumed to be dangerous regardless of their actual characteristics [198, 199]. If a monitoring methodology is overly precautionary, costs may be incurred by implementing unnecessary control measures to treat "false positive" results. This traditional method of assessment also fails to consider the associated risk should

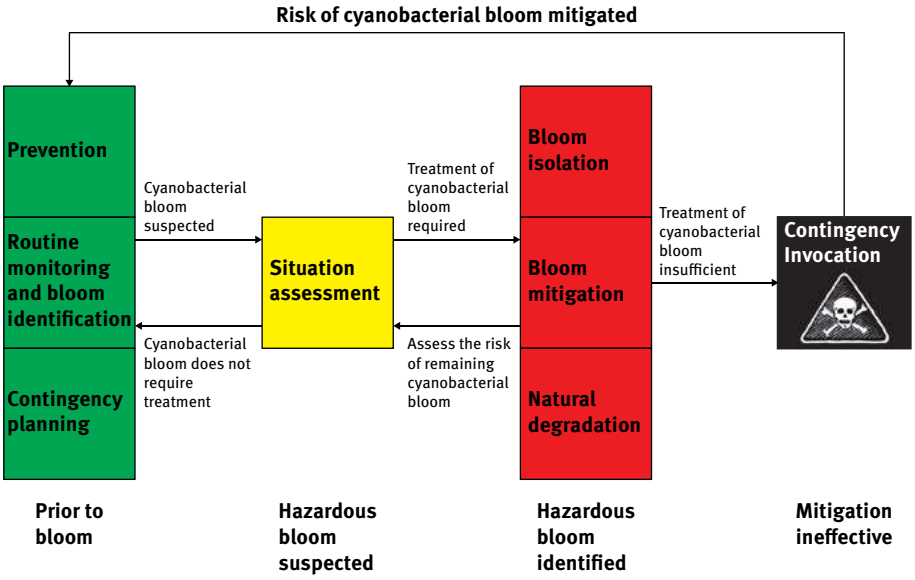

Fig. 10.1: Management framework for the removal of toxic cyanobacteria from water bodies.

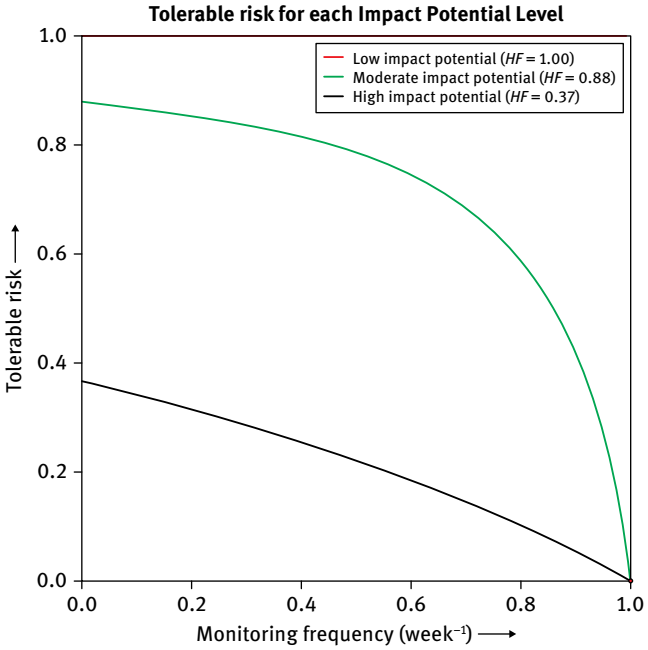

Fig. 10.2: Tolerated/tolerable risk according to monitoring frequency for each impact potential level, for Swan Coastal Plain Lakes. The tolerated risk indicates the probability that a cyanobacterial bloom at or above each impact potential level will not be detected given it is occurring. HF = Hazard Frequency. For further details on the methodology and definitions, please refer to [203].

a hazardous event not be identified by the monitoring regime (a "false negative" result). Since most monitoring methods are not of significant accuracy to identify every occurrence of a hazardous event, it is imperative that water managers are aware of the possibility that a dangerous event may occur which is not identified by the current monitoring methodology (Fig. 10.3). Water utilities and managers must optimize their monitoring regimes to reduce the occurrence of both "false positive" and "false negative" results, which will in turn reduce the risks and costs associated with hazardous events such as toxic cyanobacterial blooms.

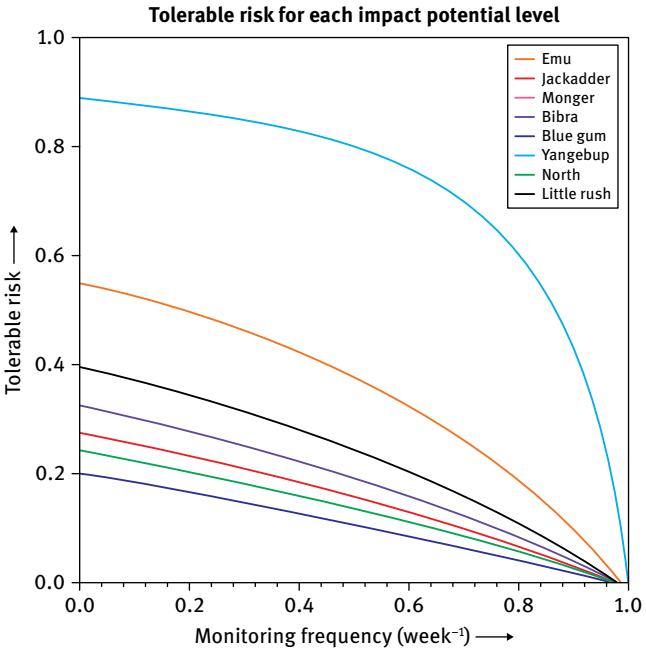

Fig. 10.3: Tolerated risk according to monitoring frequency for each impact potential level, at each of the investigated lakes of the Swan Coastal Plain. The tolerated risk indicated the probability that a cyanobacterial bloom at or above each impact potential level will not be detected given it is occurring. For further details on the methodology and definitions, please refer to [203].

A form of risk assessment that is used in medical diagnosis can be applied to environmental conditions and determines the probability that monitoring results will correctly identify hazardous situations (so long as the approximate frequency of the hazardous event is known). The development and application of this risk assessment methodology is outlined in Barrington et al. [197]. This assessment considers the relative probability of diagnostic tools returning "true positive", "true negative", "false positive" and "false negative" results [200]. This can assist in the development of monitoring programs and environmental decision-making [201, 202].

10.6 Case study: The management of cyanobacteria in waste stabilization ponds

Waste stabilization ponds (WSPs) are one form of freshwater body that have received minimal research with regards to the management of toxic cyanobacteria. Within such systems, the health and environmental risks associated with cyanobacterial blooms are coupled with the negative effects of cyanobacteria upon wastewater treatment processes, which may result in further indirect health, ecological and economic concerns. Cyanobacterial blooms are a serious problem in these systems, where they result in substantial increases to operational and maintenance costs of these assets. Hence the timely management of blooms in such reservoirs is essential.

Water containing human excreta has been treated since the link between sewage and human health was first recognized. There are many methods of wastewater treatment currently utilized, but the most commonly used process throughout both the developed and developing worlds consists of systems of WSPs [196, 204]. Such wastewater treatment plants (WWTPs) are generally utilized in rural and remote areas (Fig. 10.4), but plants servicing upwards of one million people have shown success where the land is available and reasonably priced [204].

Fig. 10.4: Examples of waste stabilization ponds (WSPs) in Australia.

WSPs are a simple, highly efficient, low-cost, low-maintenance and robust process for treating wastewater [204–206]. In WSPs, wastewater constituents are removed by sedimentation or transformed by biological and chemical processes, and a sludge layer forms due to the sedimentation of influent suspended solids, algae, and bacteria [205, 207]. In addition, WSPs are more efficient at removing pathogens than the electrochemical methods utilized in most urban WWTPs [195, 208].

After coarse screening to remove large objects, wastewater enters an initial deep WSP where sedimentation removes settlable particles including helminth eggs and protozoan cysts. These ponds are likely anaerobic due to the high biological oxygen demand (BOD) loading, and such oxygen-depleted conditions result in the significant reduction of BOD. Following sedimentation and anaerobic processing, wastewater enters facultative WSPs, which primarily remove pollutants through algal-bacterial mutualism. The photosynthetic algae present in these WSPs produce oxygen, which is then consumed by the heterotrophic bacteria that degrade any remaining organic and

inorganic compounds (Fig. 10.5) [195]. This further reduces the BOD of the water, such that it will not consume large amounts of dissolved oxygen when discharged. In the final WSP of the WWTP, referred to as the maturation or polishing pond, the majority of suspended sediments and pollutants have been reduced to acceptable levels for release. The primary function of the maturation pond is to kill dangerous wastewater organisms, including coliform bacteria and viruses, by the presence of natural radiation, high pH values and adsorption to settlable solids (Fig. 10.6) [195, 209]. In an ideal WWTP system, passage through these multiple WSPs will have decreased suspended sediment, organic and inorganic compounds, and dangerous wastewater organisms

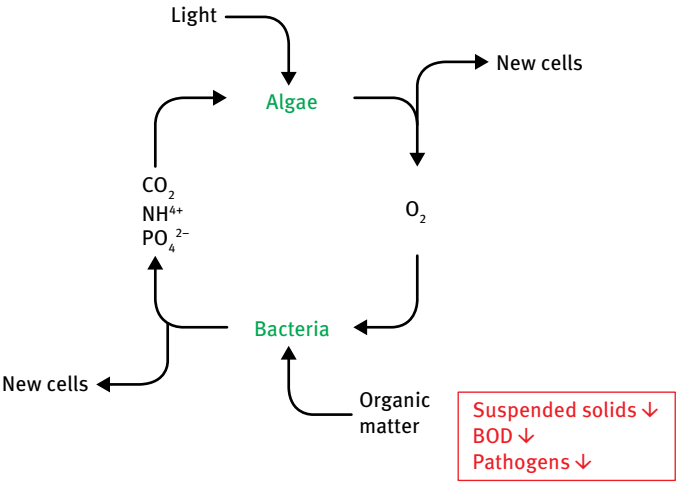

Fig. 10.5: Algal-bacterial mutualism in waste stabilization ponds (adapted from [195]).

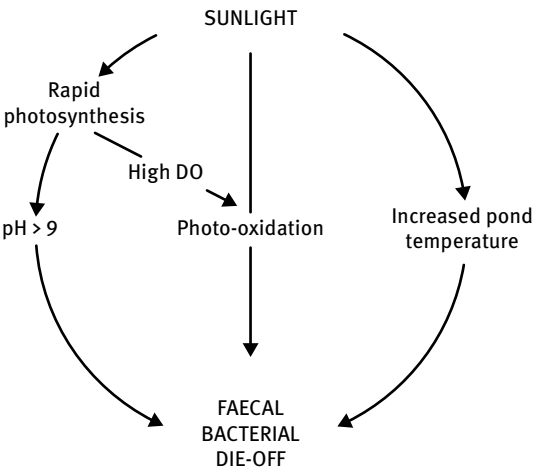

Fig. 10.6: Conceptual mechanisms for faecal-bacterial die-off in facultative and maturation waste stabilization ponds (reproduced from [195]).

to levels which will not harm humans or animals upon their release [195, 196]. Treated water is generally discharged to on-site evaporation, the environmental flow or human reuse [210].

Cyanobacteria have been recorded in WSPs throughout the world (e.g. [211–216], Fig. 10.7). Cyanobacterial blooms increase the sludge and suspended solids loadings of WSPs and change their ecology. By altering WSP ecology, cyanobacteria inhibit the natural processes of water purification anticipated by design engineers, particularly algal-bacterial mutualism, and this can result in the discharge of inadequately treated wastewater effluent.

Fig. 10.7: Cyanobacterial bloom at in WSP in central Western Australia (Photos: [217, 218]).

Cyanobacteria and cyanotoxins impact upon WSP ecology through physical, chemical and biological mechanisms [219]. The ability of cyanobacteria to regulate their buoyancy gives them a competitive advantage over other phytoplankton, forming dense blooms and surface scums that shade the organisms below [21–23]. This shading inhibits the growth of other autotrophic organisms required for wastewater treatment and the removal of coliform bacteria and viruses by natural radiation. Cyanobacterial blooms may also alter the BOD, either by inhibiting natural wastewater treatment or by increasing the BOD when cells decay. This in turn decreases dissolved oxygen concentrations, which can have dire effects on aquatic species when effluent is discharged to the natural environment [10, 24]. Cyanotoxins are harmful to the aquatic biota involved in WSP treatment, including other phytoplankton, zooplankton and protozoa (reviewed in [220]), which can negatively impact treatment processes.

These changes in pond ecology caused by cyanobacteria have the potential to cause a shift away from beneficial wastewater treatment organisms, thus inhibiting treatment. This decreases the removal of wastewater pollutants such as coliform bacteria, nutrients and BOD, increasing the risk that insufficiently treated wastewater effluent will be discharged to reuse or the environment.

In highly eutrophic, shallow WSPs, prevention of blooms through these measures may not be practical or possible. Other preventative measures may also be of limited use in the WSP environment. The establishment of macrophyte communities has been shown to lower cyanobacterial concentrations, but is impractical in WSPs as macrophyte communities may impact upon wastewater treatment processes and provide breeding habitats for insects that carry vector-borne disease. Predation by the addi-

tion of zooplankton and fish, and biomanipulation by the addition of decaying organic matter and allelopathic chemicals, have been successful in some reservoirs, but may alter WSP ecology or significantly increase sludge production, further decreasing WWTP efficiency. Ultrasonication to prevent the formation of large cyanobacterial blooms has shown some promise in reduced-scale experiments, but is yet to be trialed on scales large enough to infer its preventative efficiency at the full-scale. Ultraviolet radiation may prevent cyanobacteria in some situations, but where cyanobacteria are already exposed to UVR they have likely developed defense mechanisms, reducing the suitability of UVR as a preventative technique.

Waste stabilization ponds are eutrophic, shallow systems which experience high levels of natural irradiance, so it is unlikely that cyanobacterial blooms can always be prevented.

Consideration of the cyanobacterial and cyanotoxin removal methods studied in the literature (Tab. 10.3) suggests that hydrogen peroxide (H_2O_2) may be suitable for reducing cyanobacterial and cyanotoxin concentrations, and may be more successful when coupled with other mitigation techniques. Hydrogen peroxide degrades within hours of addition [221–224], is not considered to be carcinogenic [225] and is unlikely to impact significantly upon aquatic biota at the concentrations required for cyanobacterial removal [177, 226, 227]. Hydrogen peroxide should not pose a risk to ecosystems or humans if treated effluent is discharged to the environment or for reuse.

Hydrogen peroxide has been used occasionally for cyanobacterial management in WSPs, although there has been minimal scientific investigation into the removal dynamics of cyanobacteria and cyanotoxins by this method. The addition of H_2O_2 alone has often been considered inadequate for cyanotoxin treatment [140, 165, 170, 174, 228]. However, there are many physical, chemical and biological properties of WSPs that differ from laboratory studies, and the presence of such factors may improve the potential for cyanobacterial and cyanotoxin treatment by H_2O_2 under WSP conditions.

10.7 Treatment of cyanobacteria and cyanotoxins with hydrogen peroxide

Cyanobacterial and cyanotoxin removal by H_2O_2 proceeds via the generation of hydroxyl (•OH) and hydroperoxyl (•OOH) radicals and is illustrated in Fig. 10.8. Hydroxyl and hydroperoxyl radicals are produced naturally from H_2O_2 through interaction with chemical catalysts (e.g. iron) and UVR. These radicals damage cells via multiple pathways, including membrane disruption, mutagenesis, bleaching of pigments, oxidation of photosystem II, reduction of carbon dioxide fixation and the division of peptides [164, 166, 179, 229–233]. These mechanisms lead to oxidative stress within cells, similar to the effects caused by photo-inhibition under high natural radiation doses [234]. Most phytoplankton are able to repair systems damaged by photo-

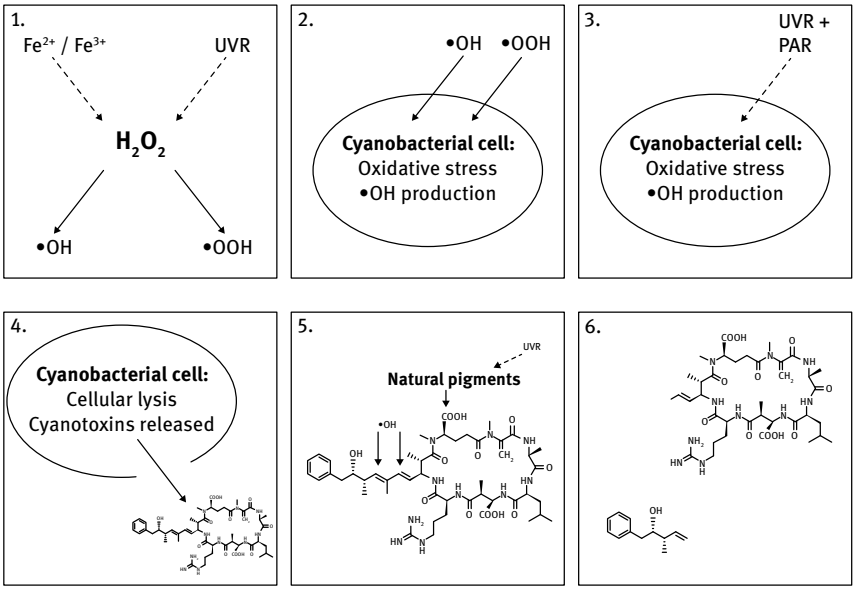

Fig. 10.8: Method of action of hydrogen peroxide (H_2O_2) for cyanobacterial and cyanotoxin removal.

1. H_2O_2 is added to the water column. Chemical catalysts (e.g. iron) and ultraviolet radiation (UVR) react with H_2O_2 to produce hydroxyl (•OH) and hydroperoxyl (•OOH) radicals.
2. •OH and •OOH radicals attack cyanobacterial cells, inducing oxidative stress and an increased cellular production of •OH.
3. Oxidative stress may lead to death if cellular processes are unable to repair the damage to core systems. Damaged cells become more susceptible to damage by UVR and photosynthetically active radiation (PAR).
4. Cell death and lysis occurs. This releases cyanotoxins (here microcystin-LR) into the water column.
5. •OH and •OOH radicals attack the conjugated bonds of cyanotoxins, which can lead to oxidation and ultimately cleavage. UVR interacts with natural pigments in the water column, which can lead to isomerization of the cyanotoxin molecule.
6. The toxicity of the cyanotoxin is destroyed.

inhibition and oxidative stress within hours [229, 232–235]. Where these mechanisms are insufficient, permanent damage occurs, and it is likely that core photosynthetic activities have been lost, resulting in cyanobacterial death. The ensuing cell lysis releases cyanotoxins to the water column. Hydroxyl and hydroperoxyl radicals destroy the toxicity of cyanotoxins by targeting their conjugated diene structure, forming dihydroxylated products and inducing cleavage of the molecule [236]. Other natural processes such as thermal decomposition, isomerization, adsorption and biodegradation also destroy the toxicity of dissolved cyanotoxins, and studies have determined that oxidation of cyanotoxins results in non-toxic by-products [180, 188, 237]. Hydrogen peroxide may thus be effective for treating both cyanobacteria and cyanotoxins in WSPs.

Most studies into the use of H_2O_2 have been conducted within the laboratory, often on batch cultures of single cyanobacterial species [e.g. 168, 140, 164, 171, 166, 237] or purified cyanotoxin compounds in distilled water [e.g. 172, 173, 165, 170, 227, 174, 175]. The results of such investigations are not necessarily indicative of the dynamics of cyanobacteria and cyanotoxins following H_2O_2 addition to WSPs.

The effects of H_2O_2 on mixed phytoplankton assemblages under environmental conditions may be altered by a number of physical, chemical and biological variables. Natural irradiance increases the concentration of •OH and •OOH in solution [168] and further damages cells already inhibited by oxidation [239]. Mixing and stratification alter the position of phytoplankton and algicides within the water column, directly affecting the treatment of cells [240, 241]. The presence of other biota and compounds that may react preferentially with •OH and •OOH may reduce the removal of cyanobacteria and cyanotoxins by H_2O_2, and the dynamics of H_2O_2 as an algicide may differ between prokaryotic cyanobacteria and eukaryotic phytoplankton species present in the assemblage [166, 168, 177, 242]. Although H_2O_2 does induce cell death in various phytoplankton, the decay of cyanobacterial cells appears to occur more rapidly than eukaryotic phytoplankton, suggesting that H_2O_2 may be a selective algicide when applied under environmental conditions [177]. Such environmental variables must be investigated to determine the applicability of H_2O_2 as an algicide.

Many studies into the effectiveness of H_2O_2 for removing cyanobacteria and cyanotoxins have been conducted on short timescales and at low temporal resolution (e.g. [140, 165, 167, 168, 170, 172–175, 228, 243]). In these investigations, particularly where H_2O_2 has not been coupled with other physical or chemical mitigation techniques, the removal of cyanotoxins by H_2O_2 has been considered negligible [140, 165, 170, 174, 228]. Short measurement periods have been considered sufficient given the rapid decay of H_2O_2 and the requirement for fast removal of cyanobacteria and cyanotoxins in rapid flow-through systems such as drinking water treatment. However, in systems such as WSPs, immediate cyanobacterial cell death and cyanotoxin removal is not required. Where H_2O_2 induces oxidative stress, cells may die within a timescale longer than that of H_2O_2 decay, releasing cyanotoxins to the dissolved state. Natural mechanisms may then degrade dissolved cyanotoxins given sufficient retention time, and it is thus important to monitor the effectiveness of H_2O_2 addition on timescales that allow for the induction of cellular stress followed by death, as well as natural degradation of cyanotoxins. In WSPs, water can be retained for several days following algicidal treatment, and the degradation of cyanobacteria and cyanotoxins over a longer timeframe than those traditionally investigated may be suitable.

Cyanobacterial treatment methods that result in the release of cyanotoxins from cells (e.g. [110, 244–246]) have generally been considered unfavorable for water management. Most past studies into the use of H_2O_2 have been conducted within the laboratory, so the degradation of cyanotoxins by natural mechanisms following H_2O_2 addition has not been thoroughly investigated. Cyanotoxins may be degraded more

rapidly from the dissolved state than whilst cell bound [247], suggesting that cyanobacterial mitigation techniques that induce cell lysis may be suitable where dissolved cyanotoxins can be degraded naturally. The methods of detoxication vary between water bodies, but studies indicate that thermal, photolytic, adsorptive and biodegradation of cyanotoxins occur within the environment.

Thermal decomposition of cyanotoxins is generally most effective at acidic pH values [248], which are rarely encountered in WSPs. Photolytic degradation can occur rapidly in systems containing high concentrations of pigments, but it is often not a significant removal mechanism in the short-term [191, 249, 250]. Adsorption onto natural matter may be unreliable for ensuring cyanotoxin degradation, particularly where cyanotoxins are suspended and not undergoing filtration [114–117, 119, 122].

Biodegradation has shown considerable success in degrading cyanotoxins (reviewed in [251]) (Tab. 10.3). Complete cyanotoxin removal has been observed to occur within two to three weeks under most environmental conditions. Cyanotoxins may act as substrates for certain bacteria, so the increase in bacterial populations may increase the rate of cyanotoxin removal. This suggests that higher concentrations of cyanotoxins may be biodegraded more rapidly than lower concentrations [252]. If natural biodegradation processes can be relied upon for the removal of cyanotoxins from WSPs, the expensive toxin removal techniques often utilized in drinking water treatment will be unnecessary.

The use of H_2O_2 for the removal of cyanobacteria and cyanotoxins from reservoirs introduces a larger scale than that investigated through laboratory experiments. Although controlled microcosm experiments are important when initially investigating the use of cyanobacterial and cyanotoxin removal methods, it is difficult to replicate environmental phenomena and heterogeneity at smaller scales. Such characteristics may significantly impact the use of H_2O_2 as a mitigation technique. The position of cyanobacterial cells and cyanotoxins within the water column is an example of a larger-scale phenomenon that may impact H_2O_2 use. Cells and cyanotoxins may be influenced by buoyancy regulation or stratification [253, 254], which themselves depend upon temperature, radiation and wind conditions [255], phenomena not often included in laboratory-scale studies. In order to test the true management potential of H_2O_2 as an algicide, scaled field trials are required to infer the differences in cyanobacteria and cyanotoxin dynamics following full-scale application [256–258].

Hydrogen peroxide has shown promise in reducing cyanobacteria and cyanotoxin concentrations in multiple studies. However, there has been limited investigation into the use of H_2O_2 for removing cyanobacteria and cyanotoxins from natural phytoplankton assemblages, particularly under field conditions and at the reservoir-scale, and no previous work has investigated the dynamics of cyanobacteria and cyanotoxin removal using H_2O_2 in WSPs. Should H_2O_2 be determined to be a suitable method for the removal of cyanobacteria and cyanotoxins from WSPs, a framework for the management of cyanobacterial blooms within WWTPs using H_2O_2 may be developed for use by water utilities.

Tab. 10.3: Studies into the biodegradation of cyanotoxins.

Study	Bacterial source	Toxin source	Experimental environment	Light regime	Initial cyanotoxin concentration	Temp. (°C)	Results
[259]	River	Extract from natural bloom	river water with biofilm (L)	AD (0/12 µE m^{-2} s^{-1})	160 µg MC-LR/-YR l^{-1}	22	Half-life of 20–23 hours
[259]	River	Extract from natural bloom	tap water with biofilm (L)	AD (0/12 µE m^{-2} s^{-1})	160 µg MC-LR/-YR l^{-1}	22	Half-life of 17–84 hours
[260]	River	Extract from natural bloom	river water (L)	—	50 µg MC-LR l^{-1}	—	90 % reduction in 2 days, 100 % reduction in 12 days
[261]	Lake	Extract from natural bloom	culture medium (L)	—	0.7 µg MC-LR l^{-1} / 1.7 µg MC-RR l^{-1}	30	100 % reduction in 24 hours at pH 7, less degradation at other pH values
[262]	Lake	Lysed natural algal material	lake water (L)	D	10–136 µg MC-LR eq. l^{-1}	20	Decreased to < 1 µg/l after 7 days, 100 % reduction in 21 days
[262]	Lake	Lysed natural algal material	Lake water (F)	ND	2–54 µg MC-LR eq. l^{-1}	15.5–21.5	Decreased to < 1 µg/l after 1–4 days, reduced to detection levels after 8 days
[263]	Reservoir water and bed sediment	Stock solution	Reservoir water and bed sediment (L)	AD	10 µg MC-LR l^{-1}	17–21	100 % reduction in 6–7 days
[264]	River and loch	Extract from culture	River and loch water (L)	—	1000–5000 µg MC-LR/-RR/-LW/ -LF/NOD l^{-1}	29	100 % reduction in 7–19 days

Tab. 10.3 (continued)

Study	Bacterial source	Toxin source	Experimental environment	Light regime	Initial cyanotoxin concentration	Temp. (°C)	Results
[265]	Lake (during cyanobacterial bloom)	—	Lake water (L)	AD	20 µg NOD l^{-1}	8–25	100 % reduction in 2–15 days
[265]	Lake (no cyanobacterial bloom present)	—	Lake water (L)	AD	20 µg NOD l^{-1}	20–25	100 % reduction in 7 days
[163]	Biological sand filter	Extract from natural bloom	Reservoir water (L)	—	3–25 µg MC-LR/-LA l^{-1}	22–30	100 % reduction in 2–15 days
[252]	Tertiary treated effluent	Stock solution	Tertiary treated effluent (L)	—	6–20 µg MC-LR l^{-1}	22	100 % reduction in 3–4 days
[252]	Activated sludge treated effluent	Stock solution	Tertiary treated effluent (L)	—	20 µg MC-LR l^{-1}	10–22	100 % reduction in 7–22 days
[266]	Lake sludge	Extract from culture	Culture medium (L)	—	≈ 2–4 µg MC-LR l^{-1} / ≈ 4 µg MC-RR l^{-1}	30	100 % reduction in 4–17 hours
[267]	Lake	Extract from natural bloom	Culture medium (L)	D	6000 µg MC-RR/NOD l^{-1}	30	100 % reduction in 4 days
[268]	Lake	Lake (naturally occurring)	Mesocosms within lake (F)	ND	0.14–8.93 µg MC-LR/-RR l^{-1}	Variable	Toxin removal varied over several months
[244]	Lake bloom treated with copper sulfate	Lake bloom treated with copper sulfate	Lake bloom treated with copper sulfate (F)	ND	1300–1800 µg MC-LR eq. l^{-1}	Variable	9 day lag phase before degradation began, 94 % degradation after 12 days

Tab. 10.3 (continued)

Study	Bacterial source	Toxin source	Experimental environment	Light regime	Initial cyanotoxin concentration	Temp. (°C)	Results
[269]	Culture	Extract from natural bloom	Drain and dam water (L)	—	1000 µg MC-LR l^{-1}	20	3–6 day lag phase before degradation began, >95 % reduction in 10 days
[269]	Culture	Extract from natural bloom	Lake water (L)	—	1000 µg MC-LR l^{-1}	20	No significant degradation in 12 days
[269]	Culture	Extract from natural bloom	River water (L)	—	1000–16 000 µg MC-LR l^{-1}	20	Degradation rates increased with initial MC concentration > 95 % reduction in 23 days
[110]	Lake	Lake	Lake water treated with copper sulfate (L)	AD	≈ 1 µg MC-LR l^{-1}	20	MC released to dissolved phase over 4 days Half-life of 3 days following maximum release
[270]	Lake	Lake	Mesocosms within lake (F)	D/ND	0.06–3.2 µg MC-LR l^{-1}	Variable	90 % reduction in 15–30 days
[271]	Final effluent from activated sludge WWTP	Extract from culture	Sewage effluent (L)	AD	210–1620 µg MC-LR l^{-1}	25	Undetected by day 13–27
[272]	Lake	Stock solution	Filtered and sterilised lake water (L)	D	10 000 µg MC-LR/-LF/-LW/-LY/-RR/NOD l^{-1}	25	Half-life of 5–> 10 days
[273]	Estuarine water	Stock solution or lysed culture material	Estuarine water (L)	—	≈ 1000 µg MC-LR/[D-Leu^1]MC-LR l^{-1}	14–15	100 % reduction in 10–20 days

Tab. 10.3 (continued)

Study	Bacterial source	Toxin source	Experimental environment	Light regime	Initial cyanotoxin concentration	Temp. (°C)	Results
[274]	Culture	Extract from culture	Culture medium (L)	—	1/10/100 µg MC-LR l^{-1}	4/22/37	Varied significantly between bacterial strains. Greatest reduction 60.1 % in 24 hours
[275]	Culture	Extract from culture	Culture medium (L)	—	Various concentrations of CYN and MC-LR/-RR/-LW/-LY/-LF	37	Varied. Greatest reductions 60.3 % MC-LR, 62.8 % MC-RR, 77.4 % MC-LF, 31.6 % CYN
[276]	Culture	Extract from culture	Culture medium (L)	—	100–4000 µg MC-LR/-RR l^{-1}	—	99–100 % reduction in 5–9 days
[277]	Lake	Extract from natural bloom	Culture medium (L)	—	20 µg MC-LR/-RR/-YR l^{-1}	27	100 % reduction in 6 days
[277]	Lake	Extract from natural bloom	Culture medium (L)	D	3–37 µg MC-LR/-RR/-YR l^{-1}	5–30	10–30 °C, 100 % reduction in 6 days 5 °C, 60 % reduction in 7 days
[278]	Lake	—	Culture medium (L)	D	1000 µg MC-LR/-RR/-YR l^{-1}	30	95–98 % reduction in 2.5 hours
[279]	Cultures	—	Culture medium (L)	—	100 µg MC-LR l^{-1}	22/37	Varied between bacteria, greatest reduction 80 % in 25 hours
[280]	Lake	Stock solution	Culture medium (L)	—	50 000 µg MC-LR l^{-1}	25	95.5 % reduction in 21 days

Tab. 10.3 (continued)

Study	Bacterial source	Toxin source	Experimental environment	Light regime	Initial cyanotoxin concentration	Temp. (°C)	Results
[281]	Lake water and sediment	Stock solution and extract from natural bloom	Culture medium (L)	D	200 µg MC-LR/-RR l^{-1}	27	< 1 day for total degradation
[282]	Reservoir	—	Culture medium (L)	—	200 µg MC-LR/-RR l^{-1}	—	100 % degradation in 36 hours
[283]	Lake sediment	Stock solution	Culture medium (L)	—	42 300 µg MC-RR l^{-1}	30	100 % degradation in 10–36 hours
[284]	Culture	Culture	Culture medium (L)	—	114 µg MC-LR l^{-1}	—	Up to 82.7 % removal in 40 hours

Experimental environment:
L: laboratory, F: field.

Light regime:
D: dark, AD: artificial diurnal, ND: natural diurnal, AC: artificial constant.

Concentration:
MC-LR: microcystin-LR, MC-YR: microcystin YR, MC-RR: microcystin-RR, MC-LW: microcystin-LW, MC-LF: microcystin-LF, MC-LA: microcystin-LA, [D-Leu^1]MC-LR: [D-Leu^1] microcystin-LR, NOD: nodularin, CYN: cylindrospermopsin, MC-LR eq.: microcystin-LR equivalents.

—: indicates that details of the property were not stated.

10.8 New techniques for the control and characterization of cyanobacterial blooms

10.8.1 Allelopathic control of cyanobacteria

As discussed previously, there are many control methods for blooms; however, in some situations only a few of them are applicable due to other factors, including secondary pollution, high cost or no-target ecosystem effects. Consequently, there is a need for anti-algal agents that are more specific, environmentally-friendly and cost-effective.

Allelopathy is the direct or indirect effect of plants (including microorganisms) on others through the production of chemicals. This technique could be utilized for the development of an anti-algal agent for the control of harmful algal blooms; the allelopathic activity of barley straw to many kinds of algae including *Microcystis* has already been documented in the field and laboratory [58, 285–287].

Barley, *Hordeum vulgare L.*, is one of the earliest cultivated crops in the world and can be divided into two distinct groups: occidental and oriental. Most studies on cyanobacteria control using barley were undertaken in Europe and America with the cultivated occidental barley as an anti-algal agent. Oriental barley, i.e. Tibetan hulless barley (*Hordeum vulgare L. var. nudum*) originated from the Qinghai–Tibetan Plateau and is regarded as the progenitor of cultivated barley [288].

One of the key interests in allelopathy in China is to assess the acute, mid and long-term effects of Tibetan hulless barley straw extract on the growth, physiology and morphology of *Microcystis aeruginosa* at a single cell level [54, 63]. Recent data shows that a dosage of 2.0 g (dry weight) l^{-1} of Tibetan hulless barley straw reduces the *in vivo* chlorophyll-*a* (chl-*a*) fluorescence of *M. aeruginosa* cells, resulting in a significant decline of the cell density (Fig. 10.9). These studies show promise for future use of Tibetan hulless barley straw algicidic agent for *M. aeruginosa* [54, 63, 64].

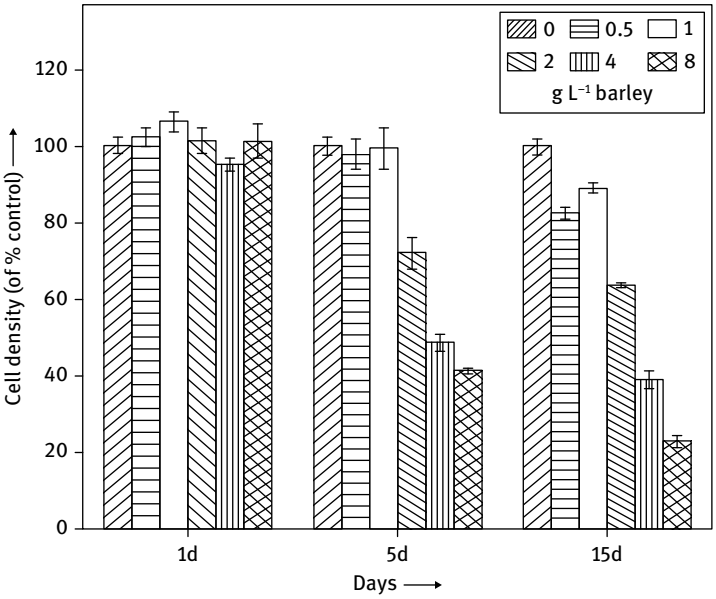

Fig. 10.9: Inhibition effect on growth of *Microcystis aeruginosa* cultures after a 1-, 5-, and 15-d Tibetan hulless barley straw exposure. All error bars correspond to the standard deviation. Reproduced from [54].

10.8.2 Optimization of the FDA-PI method using flow cytometry to measure metabolic activity of cyanobacteria

The control of *M. aeruginosa* blooms has been the focus of many studies because of the widespread occurrence and the spectacular nature of the bloom events (Fig. 10.10), where this particular species can completely dominate the phytoplankton community [289, 290].

Fig. 10.10: Spectacular *M. aeruginosa* algal bloom in Lake Taihu, China. (Photos: Hohai University, Nanjing, China).

Advanced methods for the detection and assessment of the physiological status of this particular species have been developed to ensure a reliable diagnosis of the algal blooms. One such method is the exploration of the inhibition of enzyme activity, which is now a widely accepted method to determine acute and sub-lethal endpoints for bioassays, and to assess the integrity of the cell membrane during the blooms [291]. The release of large amounts of endotoxins can constitute a major environmental and human health hazard. Therefore, it is important to develop reliable diagnostic tools to monitor the blooms events, for a better risk assessment [292, 293].

Many kinds of fluoresceins had been used to detect the enzyme activities and cell membrane integrity. Among them, fluorescein diacetate (FDA) and propidium iodide (PI) were most frequently used. FDA is a non-polar, hydrophobic, non-fluorescent esterified compound; it readily permeates the cell membrane, and is hydrolyzed by non-specific esterases producing a fluorescein [294]. The mean fluorescence intensity per cell (MFI) of FDA-converted fluorescein was used to estimate the the enzyme activity (i.e. hydrolysis rate of esterase) in algae. PI is a fully cell-membrane impermeable fluorescent dye which has been used to indicate dead cells for a wide range of microorganisms [295, 296]; it can only combine with DNA in dead cells or cells with damaged membranes [295, 297]. In contrast to PI, the use of FDA was first reported for detecting the viability of marine phytoplankton after the exposure of environmental contaminants using a fluorescence microscope [298].

Cells with an intact cell membrane are stained bright green by FDA; in contrast, cells with a broken cell membrane are stained bright orange with PI [299, 300]. Furthermore, FDA also indicates the presence of active esterase [294, 300]. Cells with an intact cell membrane and inactive esterase do not stain with FDA or PI. Afterwards, the efficiency of PDA/PI detection was greatly improved by the detection of the fluorescence of individual cells using flow cytometry [301]. More recently, Franklin et al. [291] developed a rapid enzyme inhibition bioassay based on PDA/PI for marine and freshwater microalgae with the use of flow cytometry, but no evidence of *M. aeruginosa* was detected in this study. Moreover, FDA has been used to evaluate the esterase activity of *M. aeruginosa*, but dosages used ranged from 1.6 to 16 mg l^{-1} and the incubation time differed from 8 min to 2 h [302, 303].

Recently, a new procedure based on an optimized FDA/PI condition has been developed for short-term bioassays [54, 304]. This new procedure takes working conditions such as pH and impure cultures into consideration, could avoid algal cell damages in sample preparation and separate algal cells from non-algal particles by fluorescence triggering. This procedure has been used to assess the toxicity of copper on *M. aeruginosa* in a short-term exposure (36 h). As copper concentrations increased, the esterase activity was found to decrease in a concentration-dependent manner and the membrane fragments increased (Fig. 10.11). Moreover, esterase activity was a good indicator of copper toxicity in *M. aeruginosa*. The EC_{50} value based on MFI was 101.5–146.2 µg/l (95 % confidence intervals) [304]. Therefore, this new procedure has the potential to be sub-lethal endpoint detection, and could be used for the selec-

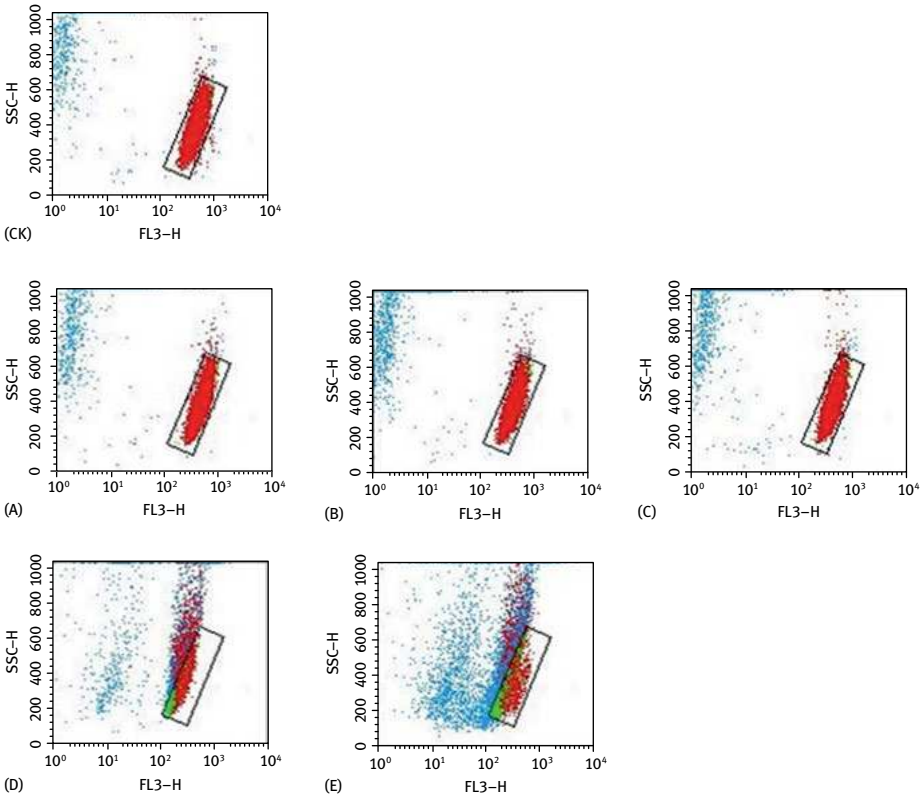

Fig. 10.11: Flow cytometry images of *M. aeruginosa* cells after 36 h copper exposure by Side Scatter (SSC) and FL3 detectors. (CK: blank control; A: 25 µg (Cu)/l; B: 40 µg (Cu)/l; C: 63 µg (Cu)/l; D: 100 µg (Cu)/l; E: 158 µg (Cu)/l).

tion of *M. aeruginosa* control methods or investigation of the *M. aeruginosa* activity inhibition mechanism as a rapid and cost-effective bioassay.

10.9 New perspectives and future directions

In recent years, the study of waste stabilization ponds (WSPs) as ecological systems has revealed new considerations that are likely to influence how we see a wide range of aquatic systems with cyanobacteria including a lakes, rivers and reservoirs. In particular, it was determined that the interplay between the hydrodynamics and ecology within these systems can explain the occurrence, magnitude and frequency of cyanobacterial blooms (Fig. 10.12).

Sludge accumulation can impact performance by reducing pond effective volume and changing the shape of the bottom surface, thus altering pond hydraulics [205,

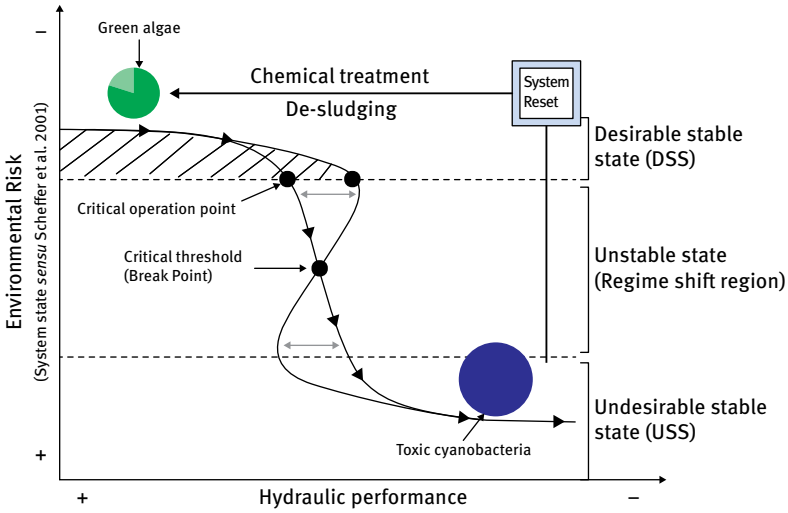

Fig. 10.12: Conceptual framework inspired by Scheffer's theory of catastrophic shift in ecosystems applied to a situation of intense eutrophication of WSP systems. The shaded area (lines) represents the desirable operational state of the system. Below the critical operation point small changes in hydraulic performance can lead to catastrophic shifts, driving the system into an undesirable stable state. A similar situation could be expected in hypereutrophic systems where intense cyanobacterial blooms occur. Reproduced from: Ghadouani and Coggins [218].

305, 306]. While periodic sludge removal is required, it is rarely considered integral to pond design [205], and the long-term sustainability of WSP systems is dependent on the safe management of sludge [206]. Previous studies have shown that distribution of sludge in ponds can be very uneven [205, 206, 217] (Fig. 10.13) and that different climatic regions have an effect on sludge accumulation rates [206]. Despite the number of WSPs worldwide (e.g. in regional Western Australia there are 84 wastewater treatment plants using 302 WSPs for treatment), there is still little information available on sludge distribution, sludge characteristics, accumulation rates, and their effect on wastewater treatment efficiency.

Besides sludge accumulation, factors that influence pond hydraulic performance are mainly related to shape, flow, inlet/outlet configuration, wind and temperature [307–310]. Optimal flow within treatment systems is described as flow with a uniform velocity profile, and it is recommended that ponds be designed to adhere to plug flow [307]; this flow regime provides mean maximum residence time. However, in reality, water in ponds does not move homogeneously, but with eddies and recirculation [307], and the actual mean residence time is always less than the nominal residence time [308]

Microbial and phytoplankton communities are essential for the functionality of WSPs, and community health is important for overall treatment efficiency. Microbial processes in ponds, such as algal growth, aerobic and anaerobic heterotrophic

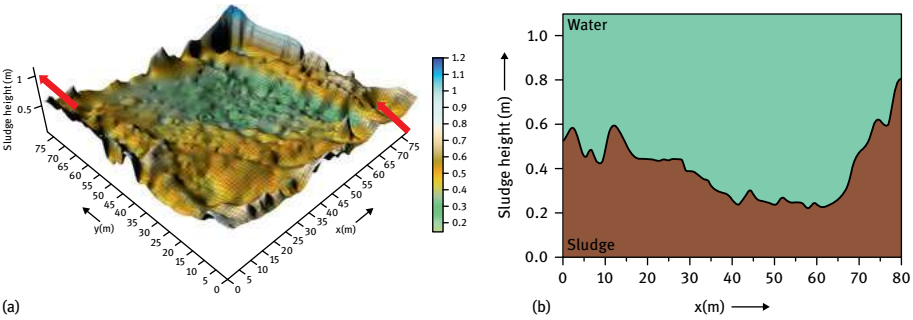

Fig. 10.13: (a) Sludge profile of a Western Australian WSP. *z*-axis shows sludge height from pond bottom in meters. Inlet and outlet configuration shown (red arrows). (b) Cross section of pond at $y = 40$ m. Note the formation of a channel between the inlet at outlet (a), and the variable horizontal distribution (b). Depth of pond: 1.1 m.

metabolism, nitrification and denitrification, work in conjunction with physical processes and exposure to sunlight to remove pathogens, nitrogen and organic contaminants [207]. The highly complex microbial communities present in wastewater treatment systems are not well understood, despite their importance in the treatment process [311]. Recent studies have indicated that there is a link between microbial diversity, community structure and treatment efficiency [312, 313], and that total bacterial cell counts [314] and phytoplankton presence [315] can be used as descriptive parameters for treatment processes and performance.

References

[1] Oelkers EH, Hering JG, Zhu C.. Water: Is there a global crisis? Elements 2011;7:157–162.
[2] Baron JS, Poff NL, Angermeier PL, Dahm CN, Gleick PH, Hairston NG, Jackson RB, Johnston CA, Richter BD, Steinman AD. Meeting ecological and societal needs for freshwater. Ecological Applications 2002;12:1247–60.
[3] Duda AM, El-Ashray MT. Addressing the global water and environmental crises through integrated approaches to the management of land, water and ecological resources. Water International 2000;25:115–26.
[4] Hanjra MA, Qureshi ME. Global water crisis and future food security in an era of climate change. Food Policy 2010;35:366–77.
[5] Kanae S. Global warming and the water crisis. Journal of Health Science 2009;55:860–4.
[6] Khan FA, Ansari AA. Eutrophication: An ecological vision. Botanical Review 2005;71:449–82.
[7] Smith VH, Schindler DW. Eutrophication science: where do we go from here? Trends in Ecology & Evolution 2009;24:201–207.
[8] Chorus I, Bartram J. [eds.]. Toxic Cyanobacteria in Water: A guide to their public health consequences, monitoring and management. London: St Edmundsbury Press, Bury St. Edmonds, Suffolk, World Health Organization:1999.

[9] Codd GA, Lindsay J, Young FM, Morrison LF, Metcalf JS. Harmful cyanobacteria: From mass mortalities to management measures. In: Huisman J, Matthijs HCP, Visser PM. [eds.] Harmful Cyanobacteria. Dordrecht, The Netherlands; Springer: 2005.
[10] Sivonen K, Jones G. Cyanobacterial Toxins. In: Chorus I, Bartram J. [eds.] Toxic Cyanobacteria in Water: A guide to their public health consequences, monitoring and management. London: St Edmundsbury Press, Bury St Edmunds, Suffolk, World Health Organization: 1999.
[11] Mur LR, Skulberg OM, Utkilen H. Cyanobacteria in the environment. In: Chorus I, Bartram J. [eds.] Toxic Cyanobacteria in Water: A guide to their public health consequences, monitoring and management. London; St Edmundsbury Press, Bury St Edmunds, Suffolk, World Health Organization: 1999.
[12] El-Shehawy R, Gorokhova E, Piñas FF, Campo FF. Global warming and hepatotoxin production by cyanobacteria: What can we learn from experiments? Water Research; 2011. doi: 10.1016/j.watres.2011.11.021.
[13] Paerl HW, Paul V. Climate change: Links to global expansion of harmful cyanobacteria. Water Research; 2011. doi:10.1016/j.watres.2011.08.002.
[14] Zhang M, Duan H, Shi X, Yu Y, Kong F. Contributions of meteorology to the phenology of cyanobacterial blooms: implications for future climate change. Water Research; 2011. doi: 10.1016/j.watres.2011.11.013.
[15] Davis TW, Berry DL, Boyer GL, Gobler CJ. The effects of temperature and nutrients on the growth and dynamics of toxic and non-toxic strains of Microcystis during cyanobacteria blooms. Harmful Algae 2009;8:715–25.
[16] Wagner C, Adrian R. Cyanobacteria dominance: Quantifying the effects of climate change. Limnology and Oceanography 2009;54:2460–8.
[17] Reichwaldt ES, Ghadouani A. Effects of rainfall patterns on toxic cyanobacterial blooms in a changing climate: between simplistic scenarios and complex dynamics. Water Research 2012;46:1372–93.
[18] Paerl HW, Hall NS, Calandrino ES. Controlling harmful cyanobacterial blooms in a world experiencing anthropogenic and climatic-induced change. Science of the Total Environment 2011; 409:1739–1745.
[19] Jöhnk KD, Huisman J, Sharples J, Sommeijer B, Visser PM, Stroom JM. Summer heatwaves promote blooms of harmful cyanobacteria. Global Change Biology 2008;14:495–512.
[20] Brookes JD, Ganf GG. Variations in the buoyancy response of Microcystis aeruginosa to nitrogen, phosphorus and light. Journal of Plankton Research 2001;23:1399–411.
[21] Scheffer M, Rinaldi S, Gragnani A, Mur LR, Van Nes EH. On the dominance of filamentous cyanobacteria in shallow, turbid lakes. Ecology 1997;78:272–82.
[22] Casanova MT, Burch MD, Brock MA, Bond PM. Does toxic Microcystis aeruginosa affect aquatic plant establishment? Environmental Toxicology 1999;14:97–109.
[23] Paerl HW. Nuisance phytoplankton blooms in coastal, estuarine, and inland waters. Limnology and Oceanography 1988;33:823–47.
[24] Havens KE. Cyanobacteria blooms: effects on aquatic ecosystems. In: Hudnell HK. [ed.] Cyanobacterial Harmful Algal Blooms: State of the Science and Research Needs. Berlin; Springer-Verlag Berlin: 2008.
[25] Funari E, Testai E. Human health risk assessment related to cyanotoxins exposure. Critical Reviews in Toxicology 2008;38:97–125.
[26] Wiegand C, Pflugmacher S. Ecotoxicological effects of selected cyanobacterial secondary metabolites a short review. Toxicology and Applied Pharmacology 2005;203:201–18.
[27] Carmichael WW. Health effects of toxin-producing cyanobacteria: "The CyanoHABs". Human and Ecological Risk Assessment 2001;7:1393–407.

[28] Babica P, Hilscherova K, Bartova K, Blaha L, Maralek B. Effects of dissolved microcystins on growth of planktonic photoautotrophs. Phycologia 2007;46:137–42.
[29] Christoffersen K. Ecological implications of cyanobacterial toxins in aquatic food webs. Phycologia 1996;35:42–50.
[30] Dawson RM. The toxicology of microcystins. Toxicon 1998;36:953–62.
[31] De Figueiredo DR, Azeiteiro UM, Esteves SM, Goncalves FJM, Pereira MJ. Microcystin producing blooms – a serious global public health issue. Ecotoxicology and Environmental Safety 2004;59:151–63.
[32] Landsberg JH. The effects of harmful algal blooms on aquatic organisms. Reviews in Fisheries Science 2002;10:113–390.
[33] Codd GA, Bell SG, Kaya K, Ward CJ, Beattie KA, Metcalf JS. Cyanobacterial toxins, exposure routes and human health. European Journal of Phycology 1999;34;405–15.
[34] Falconer IR. An overview of problems caused by toxic blue-green algae (cyanobacteria) in drinking and recreational water. Environmental Toxicology 1999;14:5–12.
[35] Gilroy DJ, Kauffman KW, Hall RA, Huang X, Chu FS. Assessing potential health risks from microcystin toxins in blue-green algae dietary supplements. Environmental Health Perspectives 2000;108:435–9.
[36] Pilotto LS, Douglas RM, Burch MD, Cameron S, Beers M, Rouch GJ, Robinson P, Kirk M, Cowie CT, Hardiman S, Moore C, Attewell RG. Health effects of exposure to cyanobacteria (blue-green algae) during recreational water-related activities. Australian and New Zealand Journal of Public Health 1997;21:562–6.
[37] Ueno Y, Nagata S, Tsutsumi T, Hasegawa A, Watanabe MF, Park HD, Chen GC, Chen G, Yu SZ. Detection of microcystins, a blue-green algal hepatotoxin, in drinking water sampled in Haimen and Fusui, endemic areas of primary liver cancer in China, by highly sensitive immunoassay. Carcinogenesis 1996;17:1317–21.
[38] Carbis CR, Rawlin GT, Grant P, Mitchell GF, Anderson JW, McCauley I. A study of feral carp, Cyprinus carpio L, exposed to Microcystis aeruginosa at Lake Mokoan, Australia, and possible implications for fish health. Journal of Fish Diseases 1997;20:81–91.
[39] Vasconcelos VM. Uptake and depuration of the heptapeptide toxin microcystin-LR in Mytilus galloprovincialis. Aquatic Toxicology 1995;32:227–37.
[40] Eriksson JE, Meriluoto J, Lindholm T. Accumulation of a peptide toxin from the cyanobacterium Oscillatoria agardhii in the freshwater mussel Anadonta cygnea. Hydrobiologia 1989; 183:211–6.
[41] Cox PA, Banack SA, Murch SJ. Biomagnification of cyanobacterial neurotoxins and neurodegenerative disease among the Chamorro people of Guam. Proceedings of the National Academy of Sciences of the United States of America 2003;100:13380–3.
[42] Peng LA, Liu YM, Chen W, Liu LM, Kent M, Song LR. Health risks associated with consumption of microcystin-contaminated fish and shellfish in three Chinese lakes: Significance for freshwater aquacultures. Ecotoxicology and Environmental Safety 2010;73:1804–11.
[43] Chen J, Xie P, Guo LG, Zheng L, Ni LY. Tissue distributions and seasonal dynamics of the hepatotoxic microcystins-LR and -RR in a freshwater snail (Bellamya aeruginosa) from a large shallow, eutrophic lake of the subtropical China. Environmental Pollution 2005;134:423–30.
[44] Chen J, Xie P. Tissue distributions and seasonal dynamics of the hepatotoxic microcystins-LR and -RR in two freshwater shrimps, Palaemon modestus and Macrobrachium nipponensis, from a large shallow, eutrophic lake of the subtropical China. Toxicon 2005;45:615–25.
[45] Chen J, Xie P, Zhang DW, Ke ZX, Yang H. In situ studies on the bioaccumulation of microcystins in the phytoplanktivorous silver carp (Hypophthalmichthys molitrix) stocked in Lake Taihu with dense toxic Microcystis blooms. Aquaculture 2006a;261:1026–38.

[46] De Magalhaes VF, Soares RM, Azevedo S. Microcystin contamination in fish from the Jacarepagua Lagoon (Rio de Janeiro, Brazil): ecological implication and human health risk. Toxicon 2001;39:1077–85.
[47] Soares RM, Yuan M, Servaites JC, Delgado A, Maglhaes VF, Hilborn ED, Carmichael WW, Azevedol S. Sublethal exposure from microcystins to renal insufficiency patients in Rio de Janeiro, Brazil. Environmental Toxicology 2006;21:95–103.
[48] Papadimitriou T, Kagalou I, Bacopoulos V, Leonardos ID. Accumulation of microcystins in water and fish tissues: An estimation of risks associated with microcystins in most of the Greek lakes. Environmental Toxicology 2010;25:418–27.
[49] Lehtiniemi M, Engstrom-Ost J, Karjalainen M, Kozlowsky-Suzuki B, Viitasalo M. Fate of cyanobacterial toxins in the pelagic food web: transfer to copepods or to faecal pellets? Marine Ecology Progress Series 2002;241:13–21.
[50] Kotak BG, Zurawell RW, Prepas EE, Holmes CFB. Microcystin-LR concentration in aquatic food web compartments from lakes of varying trophic status. Canadian Journal of Fisheries and Aquatic Sciences 1996;53:1974–85.
[51] Park MH, Chung IM, Ahmad A, Kim BH, Hwang SJ. Growth inhibition of unicellular and colonial Microcystis strains (cyanophyceae) by compounds isolated from rice (Oryza sativa) hulls. Aquatic Botany 2009a;90:309–14.
[52] Park MH, Kim BH, Chung IM, Hwang SJ. Selective bactericidal potential of rice (Oryza sativa L. var. japonica) hull extract on Microcystis strains in comparison with green algae and zooplankton. Bulletin of Environmental Contamination and Toxicology 2009b;83:97–101.
[53] Park MH, Han MS, Ahn CY, Kim HS, Yoon BD, Oh HM. Growth inhibition of bloom-forming cyanobacterium Microcystis aeruginosa by rice straw extract. Letters in Applied Microbiology 2006;43:307–12.
[54] Xiao X, Chen YX, Liang XQ, Lou LP, Tang XJ. Effects of Tibetan hulless barley on bloom-forming cyanobacterium (Microcystis aeruginosa) measured by different physiological and morphologic parameters. Chemosphere 2010;81:1118–23.
[55] Nakai S, Zhou S, Hosomi M, Tominaga M. Allelopathic growth inhibition of cyanobacteria by reed. Allelopathy Journal 2006;18:277–85.
[56] Zhou LR, Hou LG, Hu YY, Song JG, Chen WQ. Effects of wattle extract on Microcystic aeruginosa growth and the simulated mini fresh water ecosystem. Journal of Environmental Biology 2010;31:1023–30.
[57] Lurling M, Beekman W. Anti-cyanobacterial activity of Moringa oleifera seeds. Journal of Applied Phycology 2010;22:503–10.
[58] Ball AS, Williams M, Vincent D, Robinson J. Algal growth control by a barley straw extract. Bioresource Technology 2001;77:177–81.
[59] Barrett PRF, Littlejohn JW, Curnow J. Long-term algal control in a reservoir using barley straw. Hydrobiologia 1999;415:309–13.
[60] Everall NC, Lees DR. The use of barley-straw to control general and blue-green algal growth in a Derbyshire reservoir. Water Research 1996;30:269–76.
[61] Hilt S, Ghobrial MGN, Gross EM. In situ allelopathic potential of Myriophyllum verticillatum (Haloragaceae) against selected phytoplankton species. Journal of Phycology 2006;42: 1189–98.
[62] Fistarol GO, Legrand C, Graneli E. Allelopathic effect of Prymnesium parvum on a natural plankton community. Marine Ecology-Progress Series 2003;255:115–25.
[63] Xiao X, Huang H, Ge Z, Rounge TB, Shi J, Xu X, Li R, Chen Y. A pair of chiral flavonolignans as novel anti-cyanobacterial allelochemicals derived from barley straw (Hordeum vulgare): characterization and comparison of their anti-cyanobacterial activities. Environmental Microbiology 2014;16:1238–51.

[64] Huang H, Xiao X, Ghadouani A, Wu J, Nie Z, Peng C, Xu X, Shi J. Effects of Natural Flavonoids on Photosynthetic Activity and Cell Integrity in Microcystis aeruginosa. Toxins 2015;7:66–80.
[65] Visser PM, Ibelings BW, Vanderveer B, Koedood J, Mur LR. Artificial mixing prevents nuisance blooms of the cyanobacterium Microcystis in Lake Nieuwe Meer, the Netherlands. Freshwater Biology 1996a;36:435–50.
[66] Becker A, Herschel A, Wilhelm C. Biological effects of incomplete destratification of hypertrophic freshwater reservoir. Hydrobiologia 2006;559:85–100.
[67] Chorus I, Mur LR. Preventative measures. In: CHORUS, I, BARTRAM, J. [eds.] Toxic Cyanobacteria in Water: A guide to their public health consequences, monitoring and management. London; St Edmundsbury Press, Bury St Edmunds, Suffolk, World Health Organization: 1999.
[68] Vanderveer B, Koedood J, Visser PM. Artificial mixing- a therapy measure combating cyanobacteria in Lake Nieuwe-Meer. Water Science and Technology 1995;31:245–8.
[69] Heo WM, Kim B. The effect of artificial destratification on phytoplankton in a reservoir. Hydrobiologia 2004;524:229–39.
[70] Brookes JD, Baker P, Burch M. Ecology and management of cyanobacteria in rivers and reservoirs. Blue-Green Algae: Their significance and management within water supplies. Salisbury, Australia; CRC for Water Quality and Treatment: 2002.
[71] Steffensen D, Burch M, Nicholson B, Drikas M, Baker P. Management of toxic blue-green algae (cyanobacteria) in Australia. Environmental Toxicology 1999;14:183–95.
[72] Nakai S, Inoue Y, Hosomi M, Murakami A. Myriophyllum spicatum-released allelopathic polyphenols inhibiting growth of blue-green algae Microcystis aeruginosa. Water Research 2000;34:3026–32.
[73] Nakai S, Zou G, Okudo T, Tsai, TY, Song X, Nishijima W, Okada M. Anti-cyanobacterial allelopathic effects of plants used for artificial floating islands. Allelopathy Journal 2010;26: 113–21.
[74] Mohamed ZA, Al Shehri AM. Differential responses of epiphytic and planktonic toxic cyanobacteria to allelopathic substances of the submerged macrophyte Stratiotes aloides. International Review of Hydrobiology 2010;95:224–34.
[75] Zhu JY, Liu BY, Wang J, Gao YN, Wu ZB. Study on the mechanism of allelopathic influence on cyanobacteria and chlorophytes by submerged macrophyte (Myriophyllum spicatum) and its secretion. Aquatic Toxicology 2010;98:196–203.
[76] Zhang TT, He M, Wu AP, Nie LW. Allelopathic effects of submerged macrophyte Chara vulgaris on toxic Microcystis aeruginosa. Allelopathy Journal 2009;23:391–401.
[77] Wu ZB, Zhang SH, Wu XH, Cheng SP, He F. Allelopathic interactions between Potamogelon maackianus and Microcystis aeruginosa. Allelopathy Journal 2007;20:327–38.
[78] Nakai S, Yamada S, Hosomi M. Anti-cyanobacterial fatty acids released from Myriophyllum spicatum. Hydrobiologia 2005;543:71–8.
[79] Erhard D, Gross EM. Allelopathic activity of Elodea canadensis and Elodea nuttallii against epiphytes and phytoplankton. Aquatic Botany 2006;85:203–11.
[80] Korner S, Nicklisch A. Allelopathic growth inhibition of selected phytoplankton species by submerged macrophytes. Journal of Phycology 2002;38:862–71.
[81] Jasser I. The influence of macrophytes on a phytoplankton community in experimental conditions. Hydrobiologia 1995;306:21–32.
[82] Falconer IR. Prevention, Mitigation, and Remediation of Cyanobacterial Blooms in Reservoirs. Cyanobacterial Toxins of Drinking Water Supplies. Boca Raton; CRC Press: 2005b.
[83] Falconer IR. Cyanobacterial Toxins of Drinking Water Supplies. Boca Raton; CRC Press: 2005a.
[84] Jugnia L-B, Debroas D, Romagoux JC, Dévaux J. Initial results of remediation activities to restore hypereutrophic Villerest Reservoir (Roanne, France). Lakes and Reservoirs: Research & Management 2004;9:109–17.

[85] Conley DJ, Paerl HW, Howarth RW, Boesch DF, Seitzinger SP, Havens KE, Lancelot C, Likens GE. Controlling eutrophication: nitrogen and phosphorus. Science 2009;323:1014–5.
[86] Matveev V, Matveeva L, Jones GJ. Study of the ability of Daphnia-carinata king to control phytoplankton and resist cyanobacterial toxicity – Implications for biomanipulation in Australia. Australian Journal of Marine and Freshwater Research 1994;45:889–904.
[87] Christoffersen K, Riemann B, Klysner A, Sondergaard, M.Potential role of fish predation and natural populations of zooplankton in structuring a plankton community in eutrophic lake water. Limnology and Oceanography 1993;38:561–73.
[88] Davis TW, Gobler CJ. Grazing by mesozooplankton and microzooplankton on toxic and non-toxic strains of Microcystis in the Transquaking River, a tributary of Chesapeake Bay. Journal of Plankton Research 2011;33:415–30.
[89] Demott WR, Moxter F. Foraging on cyanobacteria by copepods- responses to chemical defenses and resource abundance. Ecology 1991;72:1820–34.
[90] Boon PI, Bunn SE, Green JD, Shiel RJ. Consumption of cyanobacteria by fresh-water zooplankton- implications for the success of top-down control of cyanobacterial blooms in Australia. Australian Journal of Marine and Freshwater Research 1994;45:875–87.
[91] Sigee DC, Glenn R, Andrews MJ, Bellinger EG, Butler RD, Epton HAS, Hendry RD. Biological control of cyanobacteria: principles and possibilities. Hydrobiologia 1999;395:161–72.
[92] Lee TJ, Nakano K, Matsumura M. A novel strategy for cyanobacterial bloom control by ultrasonic irradiation. Water Science and Technology 2002;46:207–15.
[93] Ding J, Shi HL, Timmons T, Adams C. Release and removal of microcystins from Microcystis during oxidative-, physical-, and UV-based disinfection. Journal of Environmental Engineering 2010;136:2–11.
[94] Joyce EM, Wu XG, Mason TJ. Effect of ultrasonic frequency and power on algae suspensions. Journal of Environmental Science and Health Part a-Toxic/Hazardous Substances & Environmental Engineering 2010;45:863–6.
[95] Zhang GM, Zhang PY, Wang B, Liu H. Ultrasonic frequency effects on the removal of Microcystis aeruginosa. Ultrasonics Sonochemistry 2006;13:446–50.
[96] Ahn CY, Park MH, Joung SH, Kim HS, Jang KY, Oh HM. Growth inhibition of cyanobacteria by ultrasonic radiation: Laboratory and enclosure studies. Environmental Science & Technology 2003;37:3031–7.
[97] Srisuksomwong P, Whangchai N, Yagita Y, Okada K, Peerapornpisal Y, Nomura N. Effects of ultrasonic irradiation on degradation of microcystin in fish ponds. International Journal of Agriculture and Biology 2011;13:67–70.
[98] Hao HW, Wu MS, Chen WF, Tang JW, Wu QY. Cyanobacterial bloom control by ultrasonic irradiation at 20 kHz and 1.7 MHz. Journal of Environmental Science and Health Part a-Toxic/Hazardous Substances & Environmental Engineering 2004a;39:1435–46.
[99] Hao HW, Wu MS, Chen YF, Tang JW, Wu QY. Cavitation mechanism in cyanobacterial growth inhibition by ultrasonic irradiation. Colloids and Surfaces B-Biointerfaces 2004b;33:151–6.
[100] Lee TJ, Nakano K, Matsumara M. Ultrasonic irradiation for blue-green algae bloom control. Environmental Technology 2001;22:383–90.
[101] Nakano K, Lee TF, Matsumura M. In situ algal bloom control by the integration of ultrasonic radiation and jet circulation to flushing. Environmental Science & Technology 2001;35: 4941–6.
[102] Tang JW, Wu QY, Hao HW, Chen YF, Wu MS. Effect of 1.7 MHz ultrasound on a gas-vacuolate cyanobacterium and a gas-vacuole negative cyanobacterium. Colloids and Surfaces B-Biointerfaces 2004;36:115–21.
[103] Rajasekhar P, Fan L, Nguyen T, Roddick FA. A review of the use of sonication to control cyanobacterial blooms. Water Research 2012;46:4319–29.

[104] Ehling-Schulz M, Scherer S. UV protection in cyanobacteria. European Journal of Phycology 1999;34:329–38.
[105] Bin Alam MDZ, Otaki M, Furumai H, Ohgaki S. Direct and indirect inactivation of Microcystis aeruginosa by UV-radiation. Water Research 2001;35:1008–14.
[106] Cheng XL, Shi HL, Adams CD, Timmons T, Ma YF. Effects of oxidative and physical treatments on inactivation of Cylindrospermopsis raciborskii and removal of cylindrospermopsin. Water Science and Technology 2009;60:689–97.
[107] Singh SP, Hader DP, Sinha RP. Cyanobacteria and ultraviolet radiation (UVR) stress: Mitigation strategies. Ageing Research Reviews 2010;9:79–90.
[108] Sommaruga R, Chen YW, Liu ZW. Multiple strategies of bloom-forming Microcystis to minimize damage by solar ultraviolet radiation in surface waters. Microbial Ecology 2009;57:667–74.
[109] Hrudey SE, Burch MD, Drikas M, Gregory R. Remedial measures. In: CHORUS, I, BARTRAM, J. [eds.] Toxic cyanobacteria in water: a guide to their public health consequences, monitoring, and management. New York; St Edmundsbury Press, Bury St Edmunds, Suffolk, World Health Organization: 1999.
[110] Kenefick SL, Hrudey SE, Peterson HG, Prepas EE. Toxin release from Microcystis aeruginosa after chemical treatment. Water Science and Technology 1993;27:433–40.
[111] Himberg K, Keijola AM, Hiisvirta L, Pyysalo H, Sivonen K. The effect of water-treatment processes on the removal of hepatotoxins from Microcystis and Oscillatoria cyanobacteria – a laboratory study. Water Research 1989;23:979–84.
[112] Hitzfeld BC, Höger SJ, Dietrich DR. Cyanobacterial toxins: Removal during drinking water treatment, and human risk assessment. Environmental Health Perspectives 2000;108:113–22.
[113] Miao HF, Tao WY. The mechanisms of ozonation on cyanobacteria and its toxins removal. Separation and Purification Technology 2009;66:187–93.
[114] Liu GL, Qian Y, Dai SG, Feng N. Adsorption of microcystin LR and LW on suspended particulate matter (SPM) at different pH. Water Air and Soil Pollution 2008;192:67–76.
[115] Miller MJ, Critchley MM, Hutson J, Fallowfield HJ. The adsorption of cyanobacterial hepatotoxins from water onto soil during batch experiments. Water Research 2001;35:1461–8.
[116] Morris RJ, Williams DE, Luu HA, Holmes CFB, Andersen RJ, Calvert SE. The adsorption of microcystin-LR by natural clay particles. Toxicon 2000;38:303–8.
[117] Rapala J, Lahti K, Sivonen K, Niemela SI. Biodegradability and adsorption on lake-sediments of cyanobacterial hepatotoxins and anatoxin-a. Letters in Applied Microbiology 1994;19:423–8.
[118] Klitzke S, Beusch C, Fastner J. Sorption of the cyanobacterial toxins cylindrospermopsin and anatoxin-a to sediments. Water Research 2011;45:1338–46.
[119] Eynard F, Mez K, Walther JL. Risk of cyanobacterial toxins in Riga waters (Latvia). Water Research 2000;34:2979–88.
[120] Pan G, Zou H, Chen H, Yuan XZ. Removal of harmful cyanobacterial blooms in Taihu Lake using local soils. III. Factors affecting the removal efficiency and an in situ field experiment using chitosan-modified local soils. Environmental Pollution 2006;141:206–12.
[121] Klitzke S, Apelt S, Weiler C, Fastner J, Chorus I. Retention and degradation of the cyanobacterial toxin cylindrospermopsin in sediments – The role of sediment preconditioning and DOM composition. Toxicon 2010;55:999–1007.
[122] Chen W, Song LR, Gan NQ, Li L. Sorption, degradation and mobility of microcystins in Chinese agriculture soils: Risk assessment for groundwater protection. Environmental Pollution 2006b;144:752–8.

[123] Mohamed ZA, El-Sharouny HM, Ali WS. Microcystin concentrations in the Nile River sediments and removal of Microcystin-LR by sediments during batch experiments. Archives of Environmental Contamination and Toxicology 2007;52:489–95.
[124] Lambert TW, Holmes CFB, Hrudey SE. Adsorption of microcystin-LR by activated carbon and removal in full scale water treatment. Water Research 1996;30:1411–22.
[125] Mohamed ZA, Carmichael WW, An J, El-Sharouny HM. Activated carbon removal efficiency of microcystins in an aqueous cell extract of Microcystis aeruginosa and Oscillatoria tenuis strains isolated from Egyptian freshwaters. Environmental Toxicology 1999;14:197–201.
[126] Donati C, Drikas M, Hayes R, Newcombe G. Microcystin-LR adsorption by powdered activated carbon. Water Research 1994;28:1735–42.
[127] Warhurst AM, Raggett SL, McConnachie GL, Pollard SJT, Chipofya V, Codd GA. Adsorption of the cyanobacterial hepatotoxin microcystin-LR by a low-cost activated carbon from the seed husks of the pan-tropical tree, Moringa oleifera. Science of the Total Environment 1997; 207:207–11.
[128] Falconer IR, Runnegar MTC, Buckley T, Huyn VL, Bradshaw P. Using activated carbon to remove toxicity from drinking-water containing cyanobacterial blooms. Journal American Water Works Association 1989;81:102–5.
[129] Orr PT, Jones GJ, Hamilton GR. Removal of saxitoxins from drinking water by granular activated carbon, ozone and hydrogen peroxide – implications for compliance with the Australian Drinking Water Guidelines. Water Research 2004;38:4455–61.
[130] Wang HX, Ho L, Lewis DM, Brookes JD, Newcombe G. Discriminating and assessing adsorption and biodegradation removal mechanisms during granular activated carbon filtration of microcystin toxins. Water Research 2007;41:4262–70.
[131] Xagoraraki I, Harrington GW, Zulliger K, Zeier B, Krick W, Karner DA, Standridge JH, Westrick J. Inactivation kinetics of the cyanobacterial toxin microcystin-LR by free chlorine. Journal of Environmental Engineering 2006;132:818–23.
[132] Rodriguez E, Onstad GD, Kull TPJ, Metcalf JS, Acero JL, Von Gunten U. Oxidative elimination of cyanotoxins: Comparison of ozone, chlorine, chlorine dioxide and permanganate. Water Research 2007b;41:3381–93.
[133] Acero JL, Rodriguez E, Meriluoto J. Kinetics of reactions between chlorine and the cyanobacterial toxins microcystins. Water Research 2005;39:1628–38.
[134] Nicholson BC, Rositano J, Burch MD. Destruction of cyanobacterial peptide hepatotoxins by chlorine and chloramine. Water Research 1994;28:1297–303.
[135] Daly RI, Ho L, Brookes JD. Effect of chlorination on Microcystis aeruginosa cell integrity and subsequent microcystin release and degradation. Environmental Science & Technology 2007;41:4447–53.
[136] Rodriguez EM, Acerp JL, Spoof L, Meriluto J. Oxidation of MC-LR and -RR with chlorine and potassium permanganate: Toxicity of the reaction products. Water Research 2008;42: 1744–52.
[137] Kull TPJ, Sjovall, OT, Tammenkoski MK, Backlund PH, Meriluoto JAO. Oxidation of the cyanobacterial hepatotoxin microcystin-LR by chlorine dioxide: Influence of natural organic matter. Environmental Science & Technology 2006;40:1504–10.
[138] Ho L, Kayal N, Trolio R, Newcombe G. Determining the fate of Microcystis aeruginosa cells and microcystin toxins following chloramination. Water Science and Technology 2010b; 62:442–50.
[139] Senogles PJ, Seawright AA, Shaw GR, Moore MR. Evaluation of toxic byproducts of chlorination of cyanobacterial toxins. Toxicology 2001;164:174.
[140] Rositano J, Nicholson BC, Pieronne P. Destruction of cyanobacterial toxins by ozone. Ozone-Science & Engineering 1998;20:223–38.

[141] Pyo D. Degradation of cyanobacterial toxin, microcystin LR, using chemical oxidants. Journal of Immunoassay & Immunochemistry 2008;29:211–9.
[142] Tsuji K, Watanuki T, Kondo F, Watanabe MF, Nakazawa H, Suzuki M, Uchida H, Harada KI. Stability of microcystins from cyanobacteria 4. Effect of chlorination on decomposition. Toxicon 1997;35:1033–41.
[143] Beaulieu SE, Sengco MR, Anderson DM. Using clay to control harmful algal blooms: deposition and resuspension of clay/algal flocs. Harmful Algae 2005;4:123–38.
[144] Chow CWK, House J, Velzeboer RMA, Drikas M, Burch MD, Steffensen DA. The effect of ferric chloride flocculation on cyanobacterial cells. Water Research 1998;32:808–14.
[145] Chow CWK, Drikas M, House J, Burch MD, Velzeboer RMA. The impact of conventional water treatment processes on cells of the cyanobacterium Microcystis aeruginosa. Water Research 1999;33:3253–62.
[146] Drikas M, Chow CWK, House J, Burch MD. Using coagulation, flocculation, and settling to remove toxic cyanobacteria. Journal American Water Works Association 2001;93:100–11.
[147] Guida M, Mattei M, Melluso G, Pagano G, Meric S. Daphnia magna and Selenastrum capricornutum – In evaluating the toxicity of alum and polymer used in coagulation-flocculation. Fresenius Environmental Bulletin 2004;13:1244–7.
[148] Mohamed ZA. Alum and lime-alum removal of toxic and nontoxic phytoplankton from the Nile River water: Laboratory study. Water Resources Management 2001;15:213–21.
[149] Lam AKY, Prepas EE, Spink D, Hrudey SE. Chemical control of hepatotoxic phytoplankton blooms – Implications for human health. Water Research 1995b;29:1845–54.
[150] Van Hullebusch E, Deluchat V, Chazal PM, Baudu M. Environmental impact of two successive chemical treatments in a small shallow eutrophied lake: Part I. Case of aluminium sulphate. Environmental Pollution 2002a;120:617–26.
[151] Van Hullebusch E, Chatenet P, Deluchat V, Chazal PM, Froissard D, Botineau M, Ghestem A, Baudu M. Copper accumulation in a reservoir ecosystem following copper sulfate treatment (St. Germain Les Belles, France). Water Air and Soil Pollution 2003;150:3–22.
[152] Qian HF, Yu SQ, Sun ZQ, Xie XC, Liu WP, Fu ZW. Effects of copper sulfate, hydrogen peroxide and N-phenyl-2-naphthylamine on oxidative stress and the expression of genes involved photosynthesis and microcystin disposition in Microcystis aeruginosa. Aquatic Toxicology 2010;99:405–12.
[153] Murray-Gulde CL, Heatley JE, Schwartzman AL, Rodgers JH. Algicidal effectiveness of clearigate, cutrine-plus, and copper sulfate and margins of safety associated with their use. Archives of Environmental Contamination and Toxicology 2002;43:19–27.
[154] Hanson MJ, Stefan HG. Side effects of 58 years of cupper–sulfate treatment of the Fairmont Lakes, Minnesota. Water Resources Bulletin 1984;20:889–900.
[155] Van Hullebusch E, Deluchat V, Chazal PM, Baudu M. Environmental impact of two successive chemical treatments in a small shallow eutrophied lake: Part II. Case of copper sulfate. Environmental Pollution 2002b;120:627–34.
[156] Zhou XX, He ZL, Liang ZB, Stoffella PJ, Fan JH, Yang YG, Powell CA. Long-term use of copper-containing fungicide affects microbial properties of citrus grove soils. Soil Science Society of America Journal 2011;75:898–906.
[157] Grutzmacher G, Bottcher G, Chorus I, Bartel H. Removal of microcystins by slow sand filtration. Environmental Toxicology 2002;17:386–94.
[158] Lawton LA, Cornish B, Macdonald AWR. Removal of cyanobacterial toxins (microcystins) and cyanobacterial cells from drinking water using domestic water filters. Water Research 1998; 32:633–8.
[159] Neumann U, Weckesser J. Elimination of microcystin peptide toxins from water by reverse osmosis. Environmental Toxicology and Water Quality 1998;13:143–8.

[160] Teixeira MR, Rosa MJ. Integration of dissolved gas flotation and nanofiltration for M. aeruginosa and associated microcystins removal. Water Research 2006a;40:3612–20.
[161] Teixeira MR, Rosa MJ. Neurotoxic and hepatotoxic cyanotoxins removal by nanofiltration. Water Research 2006b;40:2837–46.
[162] Ho L, Meyn T, Keegan A, Hoefel D, Saint CP, Newcombe G. Bacterial degradation of microcystin toxins within a biologically active sand filter. Water Research 2006;40:768–74.
[163] Ho L, Hoefel D, Saint CP, Newcombe G. Isolation and identification of a novel microcystin-degrading bacterium from a biological sand filter. Water Research 2007;41:4685–95.
[164] Samuilov VD, Bezryadnov DB, Gusev MV, Kitashov AV, Fedorenko TA. Hydrogen peroxide inhibits photosynthetic electron transport in cells of cyanobacteria. Biochemistry (Moscow) 200;66:640–5.
[165] Li L, Gao NY, Deng Y, Yao JJ, Zhang KJ, Li HJ, Yin DD, Ou HS, Guo JW. Experimental and model comparisons of H_2O_2 assisted UV photodegradation of Microcystin-LR in simulated drinking water. Journal of Zhejiang University-Science A 2009;10:1660–9.
[166] Barroin G, Feuillade M. Hydrogen peroxide as a potential algicide for Oscillatoria-Rubescens DC. Water Research 1986;20:619–23.
[167] Cornish BJPA, Lawton, LA, Robertson PKJ. Hydrogen peroxide enhanced photocatalytic oxidation of microcystin-LR using titanium dioxide. Applied Catalysis, B: Environmental 2000; 25:59–67.
[168] Drábková M, Admiraal W, Marsálek B. Combined exposure to hydrogen peroxide and light – Selective effects on cyanobacteria, green algae, and diatoms. Environmental Science & Technology 2007a;41:309–14.
[169] Ouzts JC, Ouzts JD, Quimby PC. Hydrogen peroxide as an algicide in sewage lagoons. Journal of the Mississippi Academy of Science 1989;34:39–43.
[170] Qiao RP, Li N, Qi XH, Wang QS, Zhuang YY. Degradation of microcystin-RR by UV radiation in the presence of hydrogen peroxide. Toxicon 2005a;45:745–52.
[171] Samuilov VD, Bezrydnov DV, Gusev MV, Kitashov AV, Fedorenko TA. Hydrogen peroxide inhibits the growth of cyanobacteria. Biochemistry (Moscow) 1999;64:47–53.
[172] Bandala ER, Martinez D, Martinez E, Dionysiou DD. Degradation of microcystin-LR toxin by Fenton and photo-Fenton processes. Toxicon 2004;43:829–32.
[173] Gajdek P, Lechowski Z, Bochnia T, Kepczynski M. Decomposition of microcystin-LR by Fenton oxidation. Toxicon 2001;39:1575–8.
[174] Ren J, Ma QW, Huang HH, Wang XR, Wang SB, Fan ZQ. Oxidative degradation of microcystin-LR by combination of UV/H2O2. Fresenius Environmental Bulletin 2010;19:3037–44.
[175] Zhong Y, Jin XC, Qiao RP, Qi XH, Zhuang YY. Destruction of microcystin-RR by Fenton oxidation. Journal of Hazardous Materials 2009;167:1114–8.
[176] Dránková M, Matthijs HCP, Admiraal W, Marsálek B. Selective effects of H_2O_2 on cyanobacterial photosynthesis. Photosynthetica 2007b;45:363–9.
[177] Matthijs HCP, Visser PM, Reeze B, Meeuse J, Slot PC, Wijn G, Talens R, Huisman J. Selective suppression of harmful cyanobacteria in an entire lake with hydrogen peroxide. Water Research 2012;46:1460–72.
[178] Dziallas C, Grossart HP. Increasing oxygen radicals and water temperature select for toxic Microcystis sp. Plos One 2011;6.
[179] Qian HF, Hu BL, Yu SQ, Pan XJ, Wu T, Fu ZW. The effects of hydrogen peroxide on the circadian rhythms of Microcystis aeruginosa. Plos One 2012;7.
[180] Brooke S, Newcombe G, Nicholson B, Klass G. Decrease in toxicity of microcystins LA and LR in drinking water by ozonation. Toxicon 2006;48:1054–9.

[181] Liu XW, Chen ZL, Zhou N, Shen JM, Ye MM. Degradation and detoxification of microcystin-LR in drinking water by sequential use of UV and ozone. Journal of Environmental Sciences – China 2010;22:1897–902.
[182] Onstad GD, Strauch S, Meriluoto J, Codd GA, Von Gunten U. Selective oxidation of key functional groups in cyanotoxins during drinking water ozonation. Environmental Science & Technology 2007;41:4397–404.
[183] Rodriguez E, Majado ME, Meriluoto J, Acero JL. Oxidation of microcystins by permanganate: Reaction kinetics and implications for water treatment. Water Research 2007a;41:102–10.
[184] Ou H, Gao NY, Wei CH, Deng Y, Qiao JL. Immediate and long-term impacts of potassium permanganate on photosynthetic activity, survival and microcystin-LR release risk of Microcystis aeruginosa. Journal of Hazardous Materials 2012;219:267–75.
[185] Islami HR, Filizadeh Y. Use of barley straw to control nuisance freshwater algae. Journal American Water Works Association 2011;103:111–8.
[186] Shephard GS, Stockenstrom S, De Villiers D, Engelbrecht WJ, Sydenham EW, Wessels GFS. Photocatalytic degradation of cyanobacterial microcystin toxins in water. Toxicon 1998; 36:1895–901.
[187] Antoniou MG, Shoemaker JA, De la Cruz AA, Dionysiou DD. Unveiling new degradation intermediates/pathways from the photocatalytic degradation of microcystin-LR. Environmental Science & Technology 2008;42:8877–83.
[188] Liu I, Lawton LA, Cornish B, Robertson PKJ. Mechanistic and toxicity studies of the photocatalytic oxidation of microcystin-LR. Journal of Photochemistry and Photobiology a-Chemistry 2002;148:349–54.
[189] Mazur-Marzec H, Meriluoto J, Plinski M. The degradation of the cyanobacterial hepatotoxin nodularin (NOD) by UV radiation. Chemosphere 2006;65:1388–95.
[190] Kaya K, Sano T. A photodetoxification mechanism of the cyanobacterial hepatotoxin microcystin–LR by ultraviolet irradiation. Chemical Research in Toxicology 1998;11:159–63.
[191] Robertson PKJ, Lawton LA, Cornish B. The involvement of phycocyanin pigment in the photodecomposition of the cyanobacterial toxin, microcystin-LR. Journal of Porphyrins and Phthalocyanines 1999;3:544–51.
[192] Song WH, Bardowell S, O'shea KE. Mechanistic study and the influence of oxygen on the photosensitized transformations of microcystins (cyanotoxins). Environmental Science & Technology 2007;41:5336–41.
[193] Welker M, Steinberg C. Rates of humic substance photosensitized degradation of microcystin-LR in natural waters. Environmental Science & Technology 2000;34:3415–19.
[194] Welker M, Steinberg C. Indirect photolysis of cyanotoxins: One possible mechanism for their low persistence. Water Research 1999;33:1159–64.
[195] Mara D. Low-cost treatment systems. In: Mara D, Horan N. [eds.] The Handbook of Water and Wastewater Microbiology. London; Elsevier: 2003.
[196] Gray NF. Biology of Wastewater Treatment. London; Imperial College Press: 2004.
[197] Barrington DJ, Ghadouani A, Sinang SC, Ivey GN. Development of a new risk-based framework to guide investment in water quality monitoring. Environmental Monitoring and Assessment 2014;186:2455–64.
[198] Burger J. Differing perspectives on the use of scientific evidence and the precautionary principle. Pure and Applied Chemistry 2003;75:2543–5.
[199] Gollier C, Treich N. Decision-making under scientific uncertainty: The economics of the precautionary principle. Journal of Risk and Uncertainty 2003;27:77–103.
[200] Hrudey SE, Leiss W. Risk management and precaution: Insights on the cautious use of evidence. Environmental Health Perspectives 2003;111:1577–81.

[201] M'Gonigle RM, Jamieson TL, McAllister MK, Perterman RM. Taking uncertainty seriously: from permissive regulation to preventative design in environmental decision making. Osgoode Hall Law Journal 1994;32:99–169.
[202] Pollard SJ, Yearsley R, Reynard N, Meadowcroft IC, Duarte-Davidson R, Duerden SL. Current directions in the practice of environmental risk assessment in the United Kingdom. Environmental Science & Technology 2002;36:530–8.
[203] Barrington DJ. Towards a comprehensive framework for the management, risk assessment, and mitigation of toxic cyanobacteria. Doctor of Philosophy. The University of Western Australia, Crawley, Australia 2012.
[204] Mara D. Domestic wastewater treatment in developing countries. London; Earthscan: 2004.
[205] Nelson KL, Cisneros BJ, Tchobanoglous G, Darby JL. Sludge accumulation, characteristics, and pathogen inactivation in four primary waste stabilization ponds in central Mexico. Water Research 2004;38:111–27.
[206] Picot B, Sambuco JP, Brouillet JL, Riviere Y. Wastewater stabilisation ponds: sludge accumulation, technical and financial study on desludging and sludge disposal case studies in France. Water Science and Technology 2005;51:227–34.
[207] Grant SB, Saphores J-D, Feldman DL, Hamilton AJ, Fletcher TD, Cook PLM, Stewardson M, Sanders BF, Levin LA, Ambrose RF, Delectic A, Brown R, Jiang SC, Rosso D, Cooper WJ, Marusic I. Taking the "Waste" Out of "Wastewater" for Human Water Security and Ecosystem Sustainability. Science 2012;337:681–6.
[208] Hosetti B, Frost S. A review of the control of biological waste treatment in stabilization ponds. Critical Reviews in Environmental Science and Technology 1998;28:193–218.
[209] Maynard HE, Ouki SK, Williams SC. Tertiary lagoons: A review of removal mechanisms and performance. Water Research 1999;33:1–13.
[210] Toze S. Reuse of effluent water – benefits and risks. Agricultural Water Management 2006; 80:147–59.
[211] Furtado ALFF, Calijuri MD, Lorenzi AS, Honda RY, Genuario DB, Fiore MF. Morphological and molecular characterization of cyanobacteria from a Brazilian facultative wastewater stabilization pond and evaluation of microcystin production. Hydrobiologia 2009;627:195–209.
[212] Kotut K, Ballot A, Wiegand C, Krienitz L. Toxic cyanobacteria at Nakuru sewage oxidation ponds – A potential threat to wildlife. Limnologica 2010;40:47–53.
[213] Martins J, Peixe L, Vasconcelos V. Cyanobacteria and bacteria co-occurrence in a wastewater treatment plant: absence of allelopathic effects. Water Science and Technology 2010; 62:1954–62.
[214] Oufdou K, Mezrioui N, Oudra B, Barakate M, Loudiki M, Alla AA. Relationships between bacteria and cyanobacteria in the Marrakech waste stabilisation ponds. Water Science and Technology 2000;42:171–8.
[215] Vasconcelos VM, Pereira E. Cyanobacteria diversity and toxicity in a wastewater treatment plant (Portugal). Water Research 2001;35:1354–7.
[216] Oudra B, Loudiki M, Vasconcelos V, Sabour B, Sbiyyaa B, Oufdou K, Mezrioui N. Detection and quantification of microcystins from cyanobacteria strains isolated from reservoirs and ponds in Morocco. Environmental Toxicology 2002;17:32–9.
[217] Coggins LX. Impact of sludge accumulation on the hydraulic efficiency of waste stabilisation ponds: Towards sustainable management of wastewater sludge in Australia. Bachelor of Engineering (Environmental) Honours, The University of Western Australia; 2011.
[218] Ghadouani A, Coggins LX. Science, technology and policy for water pollution control at the watershed scale: Current issues and future challenges. Physics and Chemistry of the Earth 2011;36:335–41.

[219] Martins J, Peixe L, Vasconcelos VM. Unraveling cyanobacteria ecology in wastewater treatment plants (WWTP). Microbial Ecology 2011;62:241–56.
[220] Zurawell RW, Chen HR, Burke JM, Prepas EE. Hepatotoxic cyanobacteria: A review of the biological importance of microcystins in freshwater environments. Journal of Toxicology and Environmental Health – Part B: Critical Reviews 2005;8:1–37.
[221] Cooper WJ, Shao CW, Lean DRS, Gordon AS, Scully FE. Factors affecting the distribution of H_2O_2 in surface waters. In: BAKER, L. A. [ed.] Environmental Chemistry of Lakes and Reservoirs. Washington, D.C.: American Chemical Society; 1994.
[222] Moffett JW, Zafiriou OC. An investigation of hydrogen peroxide chemistry in surface waters of Vineyard South with (H-2)-O-18(2) and O-18(2). Limnology and Oceanography 1990;35: 1221–9.
[223] Richard LE, Peake BM, Rusak SA, Cooper WJ, Burritt DJ. Production and decomposition dynamics of hydrogen peroxide in freshwater. Environmental Chemistry 2007;4:49–54.
[224] Cooper WJ, Zepp RG. Hydrogen-peroxide decay in waters with suspended soils- evidence for biologically mediated processes. Canadian Journal of Fisheries and Aquatic Sciences 1990; 47:888–93.
[225] Desessp JM, Lavin AL, Hsia SM, Mavis RD. Assessment of the carcinogenicity associated with oral exposures to hydrogen peroxide. Food and Chemical Toxicology 2000;38:1021–41.
[226] EKA Chemicals Inc. Freedom of information summary: Original new animal drug application: NADA 141–255, 35% Perox-aid hydrogen peroxide liquid solution. Eka Chemicals, Inc.; 2007
[227] ECETOC. Joint Assessment of Commodity Chemical No. 22, Hydrogen Peroxide, CAS No. 7722-84-1. Brussels: European Centre for Ecotoxicology and Toxicology of Chemicals (ECETOC); 1992.
[228] Qiao RP, Ma YM, Qi XH, Li N, Jin XC, Wang QS, Zhuang YY. Degradation of microcystin-RR by combination of UV/H_2O_2 technique. Chinese Chemical Letters 2005b;16:1271–4.
[229] Ohnishi N, Allakhverdiev SI, Takahashi S, Higashi S, Watanabe M, Nishiyama Y, Murata N. Two-step mechanism of photodamage to photosystem II: Step 1 occurs at the oxygen-evolving complex and step 2 occurs at the photochemical reaction center. Biochemistry 2005;44:8494–9.
[230] Wong GTF, Dunstan WM, Kim DB. The decomposition of hydrogen peroxide by marine phytoplankton. Oceanologica Acta 2003;26:191–8.
[231] Lupinkova L, Komenda J. Oxidative modifications of the Photosystem II D1 protein by reactive oxygen species: From isolated protein to cyanobacterial cells. Photochemistry and Photobiology 2004;79:152–62.
[232] Nishiyama Y, Allakhverdiev SI, Murata N. A new paradigm for the action of reactive oxygen species in the photoinhibition of photosystem II. Biochimica Et Biophysica Acta-Bioenergetics 2006;1757:742–9.
[233] Nishiyama Y, Yamamoto H, Allakhverdiev SI, Inaba M, Yokota A, Murata N. Oxidative stress inhibits the repair of photodamage to the photosynthetic machinery. Embo Journal 2001; 20:5587–94.
[234] Bouchard JN, Roy S, Campbell DA. UVB effects on the photosystem II-D1 protein of phytoplankton and natural phytoplankton communities. Photochemistry and Photobiology 2006; 82:936–51.
[235] Marwood CA, Smith REH, Furgal JA, Charlton MN, Solomon KR, Greenberg BM. Photoinhibition of natural phytoplankton assemblages in Lake Erie exposed to solar ultraviolet radiation. Canadian Journal of Fisheries and Aquatic Sciences 2000;57:371–9.
[236] Perez S, Aga DS. Recent advances in the sample preparation, liquid chromatography tandem mass spectrometric analysis and environmental fate of microcystins in water. Trends in Analytical Chemistry 2005;24:658–70.

[237] Höger SJ, Hitzfeld BC, Dietrich DR. Efficacy of different methods in the removal of cyanobacterial toxins in drinking water treatment and toxicity of by-products after ozonation of microcystin-LR. Toxicology 2001;164:183–183.
[238] Kay SH, Quimby PC, Ouzts JD. Photo-enhancement of hydrogen-peroxide toxicity to submersed vascular plants and algae. Journal of Aquatic Plant Management 1984;22:25–34.
[239] He YY, Hader DP. Reactive oxygen species and UV-B: effect on cyanobacteria. Photochemical and Photobiological Sciences 2002;1:729–36.
[240] Zhang M, Kong FX, Wu XD, Xing P. Different photochemical responses of phytoplankters from the large shallow Taihu Lake of subtropical China in relation to light and mixing. Hydrobiologia 2008;603:267–78.
[241] Gu RC, Stefan HG. Stratification dynamics in wastewater stabilization ponds. Water Research 1995;29:1909–23.
[242] Yousef N, Pistorius EK, Michel KP. Comparative analysis of idiA and isiA transcription under iron starvation and oxidative stress in Synechococcus elongatus PCC 7942 wild-type and selected mutants. Archives of Microbiology 2003;180:471–83.
[243] Samuilov VD, Timofeev KN, Sinitsyn SV, Bezryadnov DV. H_2O_2-induced inhibition of photosynthetic O_2 evolution by Anabaena variabilis cells. Biochemistry (Moscow) 2004;69: 926–33.
[244] Jones GJ, Orr PT. Release and degradation of microcystin following algicide treatment of a Microcystis aeruginosa bloom in a recreational lake, as determined by HPLC and protein phosphatase inhibition assay. Water Research 1994;28:871–6.
[245] Berg K, Skulberg OM, Skulberg R. Effects of decaying toxic blue-green-algae on water-quality – A laboratory study. Archiv Fur Hydrobiologie 1987;108:549–63.
[246] Griggiths DJ, Saker ML. The palm island mystery disease 20 years on: A review of research on the cyanotoxin cylindrospermopsin. Environmental Toxicology 2003;18:78–93.
[247] World Health Organization 2003. Algae and Cyanobacteria in Fresh Water. Guidelines for Safe Recreational Water Environments: Volume 1: Coastal and freshwaters. Geneva: World Health Organization; 2003.
[248] Harada K, Tsimeli K, Watanabe M, Kondo F. Stability of microcystins from cyanobacteria-III. Effect of pH and temperature. Phycologia 1996;35:83–8.
[249] Tsuji K, Naito S, Kondo F, Ishikawa N, Watanabe MF, Suzuki M, Harada K-I. Stability of Microcystins from cyanobacteria: Effect of light on decomposition and isomerisation. Environmental Science and Technology 1994;28:173–7.
[250] Harada K, Tsuji K. Persistence and decomposition of hepatotoxic microcystins produced by cyanobacteria in natural environment. Journal of Toxicology – Toxin Reviews 1998;17: 385–403.
[251] Ho L, Sawade E, Newcombe G. Biological treatment options for cyanobacteria metabolite removal – A review. Water Research 2011; doi: 10.1016/j.watres.2011.11.018.
[252] Ho L, Hoefel D, Palazot S, Sawade E, Newcombe G, Saint CP, Brookes JD. Investigations into the biodegradation of microcystin-LR in wastewaters. Journal of Hazardous Materials 2010a; 180:628–33.
[253] Ibelings BW, Mur LR, Kinsman R, Walsby AE. Microcystis changes its buoyancy in response to the average irradiance in the surface mixed layer. Archiv Fur Hydrobiologie 1991;120: 385–401.
[254] Visser PM, Ketelaars HAM, Vanbreemen L, Mur LR. Diurnal buoyancy changes of Microcystis in an artificially mixed storage reservoir. Hydrobiologia 1996b;331:131–41.
[255] Oliver RL, Whittington J, Lorenz Z, Webster IT. The influence of vertical mixing on the photoinhibition of variable chlorophyll a fluorescence and its inclusion in a model of phytoplankton photosynthesis. Journal of Plankton Research 2003;25:1107–1129.

[256] Thrush SF, Schneider DC, Legendre P, Whitlatch RB, Dayton PK, Hewitt JE, Hines AH, Cummings VJ, Lawrie SM, Grant J, Pridmore RD, Turner SJ, Mcardle BH. Scaling-up from experiments to complex ecological systems: Where to next? Journal of Experimental Marine Biology and Ecology 1997;216:243–54.
[257] Levin SA. The problem of pattern and scale in ecology. Ecology 1992;73:1943–67.
[258] Benndorf J. Conditions for effective biomanipulation – conclusions derived from whole-lake experiments in Europe. Hydrobiologia 1990;200:187–203.
[259] Babica P, Blaha L, Marsalek B. Removal of microcystins by phototrophic biofilms – A microcosm study. Environmental Science and Pollution Research 2005;12:369–74.
[260] Bourne DG, Blakeley RL, Riddles P, Jones GJ. Biodegradation of the cyanobacterial toxin microcystin LR in natural water and biologically active slow sand filters. Water Research 2006;40:1294–302.
[261] Chen J, Hu LB, Zhou W, Yan SH, Yang JD, Xue YF, Shi ZQ. Degradation of microcystin-LR and RR by a Stenotrophomonas sp strain EMS isolated from Lake Taihu, China. International Journal of Molecular Sciences 2010;11:896–911.
[262] Christoffersen K, Lyck S, Winding A. Microbial activity and bacterial community structure during degradation of microcystins. Aquatic Microbial Ecology 2002;27:125–36.
[263] Cousins IT, Bealing DJ, James HA, Sutton A. Biodegradation of microcystin-LR by indigenous mixed bacterial populations. Water Research 1996;30:481–5.
[264] Edwards C, Graham D, Fowler N, Lawton LA. Biodegradation of microcystins and nodularin in freshwaters. Chemosphere 2008;73:1315–21.
[265] Heresztyn T, Nicholson BC. Nodularin concentrations in Lakes Alexandrina and Albert, South Australia, during a bloom of the cyanobacterium (blue-green alga) Nodularia spumigena and degradation of the toxin. Environmental Toxicology and Water Quality 1997;12:273–82.
[266] Hu LB, Yang JD, Zhou W, Yin YF, Chen J, Shi ZQ. Isolation of a Methylobacillus sp that degrades microcystin toxins associated with cyanobacteria. New Biotechnology 2009;26: 205–11.
[267] Ishii H, Nishijima M, Abe T. Characterization of degradation process of cyanobacterial hepatotoxins by a gram-negative aerobic bacterium. Water Research 2004;38:2667–76.
[268] Ji RP, Lu XW, Li XN, Pu YP. Biological degradation of algae and microcystins by microbial enrichment on artificial media. Ecological Engineering 2009;35:1584–8.
[269] Jones G, Bourne DG, Blakeley RL, Doelle H. Degradation of the cyanobacterial hepatotoxin microcystin by aquatic bacteria. Natural Toxins 1994;2:228–35.
[270] Lahti K, Rapala J, Fardig M, Niemela M, Sivonen K. Persistence of cyanobacterial hepatotoxin, microcystin-LR in particulate material and dissolved in lake water. Water Research 1997;31: 1005–12.
[271] Lam AKY, Fedorak PM, Prepas EE. Biotransfemation of the cyanobacterial hepatotoxin microcystin-U1, as determined by HPLC and protein phosphatase bioassay. Environmental Science & Technology 1995a;29:242–6.
[272] Lawton LA, Welgamage A, Manage PM, Edwards C. Novel bacterial strains for the removal of microcystins from drinking water. Water Science and Technology 2011;63:1137–42.
[273] Lemes GAF, Kersanach R, Pinto LD, Dellagostin OA, Yunes JS, Matthiensen A. Biodegradation of microcystins by aquatic Burkholderia sp. from a South Brazilian coastal lagoon. Ecotoxicology and Environmental Safety 2008;69:358–65.
[274] Nybom SMK, Salmninen SJ, Meriluoto JAO. Removal of microcystin-LR by strains of metabolically active probiotic bacteria. FEMS Microbiology Letters 2007;270:27–33.
[275] Nybom SMK, Salminen SJ, Meriluoto JAO. Specific strains of probiotic bacteria are efficient in removal of several different cyanobacterial toxins from solution. Toxicon 2008;52:214–20.

[276] Ou DY, Song LR, Gan NQ, Chen W. Effects of microcystins on and toxin degradation by Poterioochromonas sp. Environmental Toxicology 2005;20:373–80.
[277] Park HD, Sasaki Y, Maruyama T, Yanagisawa E, Hiraishi A, Kato K. Degradation of the cyanobacterial hepatotoxin microcystin by a new bacterium isolated from a hypertrophic lake. Environmental Toxicology 2001;16:337–43.
[278] Saitour T, Sugiura N, Itayama T, Inamori Y, Matsumura M. Degradation characteristics of microcystins by isolated bacteria from Lake Kasumigaura. Journal of Water Supply Research and Technology-Aqua 2003;52:13–8.
[279] Surono IS, Collado MC, Salminen S, Meriluoto J. Effect of glucose and incubation temperature on metabolically active Lactobacillus plantarum from dadih in removing microcystin-LR. Food and Chemical Toxicology 2008;46:502–7.
[280] Takenaka S, Watanabe MF. Microcystin LR degradation by Pseudomonas aeruginosa alkaline protease. Chemosphere 1997;34:749–57.
[281] Tsuji K, Asakawa M, Anzai Y, Sumino T, Harada K. Degradation of microcystins using immobilized microorganism isolated in an eutrophic lake. Chemosphere 2006;65:117–24.
[282] Valeria AM, Ricardo EJ, Stephan P, Alberto WD. Degradation of Microcystin-RR by Sphingomonas sp CBA4 isolated from San Roque reservoir (Cordoba – Argentina). Biodegradation 2006;17:447–55.
[283] Wang JF, Wu PF, Chen J, Yan H. Biodegradation of microcystin-RR by a new isolated Sphingopyxis sp USTB-05. Chinese Journal of Chemical Engineering 2010;18:108–12.
[284] Zhang WH, Zhang XH, Zhang GM, Xu XQ. Variation of microcystins in a lake for water supply. Journal of Environmental Science and Health Part a-Toxic/Hazardous Substances & Environmental Engineering 2003;38:2857–65.
[285] Everall NC, Lees DR. The identification and significance of chemicals released from decomposing barley straw during reservoir algal control. Water Research 1997;31:614–20.
[286] Ferrier MD, Butler BR, Terlizzi DE, Lacouture R. The effects of barley straw (Hordeum vulgare) on the growth of freshwater algae. Bioresource Technology 2005;96:1788–95.
[287] Waybright TJ, Terlizzi DE, Ferrier MD. Chemical characterization of the aqueous algistatic fraction of barley straw (Hordeum vulgare) inhibiting Microcystis aeruginosa. Journal of Applied Phycology 2009;21:333–40.
[288] Zeng X, Long H, Wang Z, Zhao S, Tang Y, Huang Z, Wang Y, Xu Q, Mao L, Deng G, Yao X, Li X, Bai L, Yuan H, Pan Z, Liu R, Chen X, Wangmu Q, Chen M, Yu L, Liang J, Dunzhu D, Zheng Y, Yu S, Luobu Z, Guang X, Li J, Deng C, Hu W, Chen C, Taba X, Gao L, Lv X, Abu YB, Fang X, Nevo E, Yu M, Wang J, Tashi N. The draft genome of Tibetan hulless barley reveals adaptive patterns to the high stressful Tibetan Plateau. Proceedings of the National Academy of Sciences 2015;112:1095–100.
[289] Paerl HW, Huisman J. Climate – Blooms like it hot. Science 2008;320:57–58.
[290] Wilhelm SW, Farnsley SE, Lecleir GR, Layton AC, Satchwell MF, Debruyn JM, Boyer GL, Zhu G, Paerl HW. The relationships between nutrients, cyanobacterial toxins and the microbial community in Taihu (Lake Tai), China. Harmful Algae 2011;10:207–15.
[291] Franklin NM, Adams MS, Stauber JL, Lim RP. Development of an improved rapid enzyme inhibition bioassay with marine and freshwater microalgae using flow cytometry. Archives of Environmental Contamination and Toxicology 2001;40:469–80.
[292] Liu YM, Chen W, Li DH, Huang ZB, Shen YW, Liu YD. Cyanobacteria-/cyanotoxin-contaminations and eutrophication status before Wuxi Drinking Water Crisis in Lake Taihu, China. Journal of Environmental Sciences-China 2011;23:575–81.
[293] Ye WJ, Liu XL, Tan J, Li DT, Yang H. Diversity and dynamics of microcystin-Producing cyanobacteria in China's third largest lake, Lake Taihu. Harmful Algae 2009;8:637–44.

[294] Dorsey J, Yentsch CM, Mayo S, McKenna C. Rapid analytical technique for the assessment of cell metabolic-activity in marine microalgae. Cytometry 1989;10:622–8.
[295] Bunthof CJ, Van den Braak S, Breeuwer P, Rombouts FM, Abee T. Rapid fluorescence assessment of the viability of stressed Lactococcus lactis. Applied and Environmental Microbiology 1999;65:3681–9.
[296] Cid A, Fidalgo P, Herrero C, Abalde J. Toxic action of copper on the membrane system of a marine diatom measured by flow cytometry. Cytometry 1996;25:32–6.
[297] Williams SC, Hong Y, Danavall DCA, Howard-Jones MH, Gibson D, Frischer ME, Verity PG. Distinguishing between living and nonliving bacteria: Evaluation of the vital stain propidium iodide and its combined use with molecular probes in aquatic samples. Journal of Microbiological Methods 1998;32:225–36.
[298] Bentleymowat JA. Application of fluorescence microscopy to pollution studies on marine-phytoplankton. Botanica Marina 1982;25:203–4.
[299] Jones KH, Senft JA. An improved method to determine cell viability by simultaneous staining with fluorescein diacetate propidium iodide. Journal of Histochemistry & Cytochemistry 1985; 33:77–9.
[300] Rotman B, Papermaster BW. Membrane properties of living mammalian cells as studied by enzymatic hydrolysis of fluorogenic esters. Proceedings of the National Academy of Sciences of the United States of America 1966;55:134–141.
[301] Humphreys MJ, Allman R, Lloyd D. Determination of the viability of Trichomonas vaginalis using flow-cytometry. Cytometry 1994;15:343–8.
[302] Brookes JD, Geary SM, Ganf GG, Burch MD. Use of FDA and flow cytometry to assess metabolic activity as an indicator of nutrient status in phytoplankton. Marine and Freshwater Research 2000;51:817–23.
[303] Yu Y, Kong F, Wang M, Qian L, Shi X. Determination of short-term copper toxicity in a multi-species microalgal population using flow cytometry. Ecotoxicology and Environmental Safety 2007;66:49–56.
[304] Xiao X, Han Z-Y, Chen Y-X, Liang X-Q, Li H, Qian Y-C. Optimization of FDA-PI method using flow cytometry to measure metabolic activity of the cyanobacteria, Microcystis aeruginosa. Physics and Chemistry of the Earth 2011;36:424–9.
[305] Peña MR, Mara DD, Sanchez A. Dispersion studies in anaerobic ponds: implications for design and operation. Water Science and Technology 2000;42:273–82.
[306] Sah L, Rousseau DPL, Hooijmans CM. Numerical Modelling of Waste Stabilization Ponds: Where Do We Stand? Water Air and Soil Pollution 2012;223:3155–71.
[307] Persson J. The hydraulic performance of ponds of various layouts. Urban Water 2000;2: 243–50.
[308] Persson J, Wittgren HB. How hydrological and hydraulic conditions affect performance of ponds. Ecological Engineering 2003;21:259–69.
[309] Torres JJ, Soler A, Saez J, Ortuno JF. Hydraulic performance of a deep wastewater stabilization pond. Water Research 1997;31:679–88.
[310] Papadopoulos A, Parisopoulos G, Papadopoulos F, Karteris A. Sludge accumulation pattern in an anaerobic pond under Mediterranean climatic conditions. Water Research 2003;37: 634–44.
[311] Daims H, Taylor MW, Wagner M. Wastewater treatment: a model system for microbial ecology. Trends in Biotechnology 2006;24:483–9.
[312] Li P, Wang YX, Wang YH, Liu K, Tong L. Bacterial community structure and diversity during establishment of an anaerobic bioreactor to treat swine wastewater. Water Science and Technology 2010;61:243–52.

[313] Wagner M, Loy A, Nogueira R, Purkhold U, Lee N, Daims H. Microbial community composition and function in wastewater treatment plants. Antonie Van Leeuwenhoek International Journal of General and Molecular Microbiology 2002;81:665–80.
[314] Hammes F, Berney M, Wang YY, Vital M, Koster O, Egli T. Flow-cytometric total bacterial cell counts as a descriptive microbiological parameter for drinking water treatment processes. Water Research 2008;42:269–77.
[315] Amengual-Morro C, Niell GM, Martinez-Taberner A. Phytoplankton as bioindicator for waste stabilization ponds. Journal of Environmental Management 2012;95:S71–6.
[316] Hunter PD, Hanley N, Czajkowski M, Mearns K, Tyler AN, Carvalho L, Codd GA. The effect of risk perception on public preferences and willingness to pay for reductions in the health risks posed by toxic cyanobacterial blooms. Science of the Total Environment 2012;426:32–44.
[317] Manganelli M, Scardala S, Stefanelli M, Vichi S, Mattei D, Bogialli S, Ceccarelli P, Corradetti E, Petrucci I, Gemma S, Testai E, Funari E. Health risk evaluation associated to Planktothrix rubescens: An integrated approach to design tailored monitoring programs for human exposure to cyanotoxins. Water Research 2010;44:1297–306.
[318] Stapelberg RF. Theoretical Overview of Safety and Risk in Engineering Design. Handbook of Reliability, Availability, Maintainability and Safety in Engineering Design. London; Springer-Verlag London Limited: 2009.