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## **REVIEW ARTICLE**

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## Review of current state of knowledge of microcystin and its impacts on fish in Lake Victoria

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## Abstract

Microcystins are part of algal toxins produced intracellularly within algal cells, being in the family of hepatotoxic cyclic peptides from various species of blue-green algae. Blue-green algae are widely abundant in many equatorial eutrophic lakes, including Lake Victoria, with microcystin mainly from cyanobacterial blooms released into the water column, with different effects along the aquatic ecosystem trophic levels. Depending on the length of exposure and exposure route, microcystin effects on fish can include embryonic hatching perturbations, reduced survival and growth rates, changes in behavior, osmoregulation, increased liver activities and heart rates, as well as histopathological effects. While bioaccumulation is confirmed among fish, biomagnification along food webs is debatable. Lake Victoria the second largest freshwater lake in the world, and the source of livelihoods to millions reported near the gulf and shore MCs of  $190 \pm 51$  to  $543 \pm 26$  ng MC/g DW, respectively. Little is known, however, on the effects of microcystin on the Lake Victoria fishery and, ultimately, on the human population against the WHO recommended human microcystin intake levels of 0.04  $\mu$ g/kg, thereby being the basis for this review.

KEYWORDS

fish, impact, Lake Victoria, microcystin, review

## **1** | INTRODUCTION

Cyanobacteria are gram-negative photosynthetic prokaryotes comprising more than 1,000 species of unicellular and multicellular microorganisms belonging to the class Cyanophyceae under the orders Gloeobacterales, Synechococcales, Oscillatoriales and Nostocales (Komarek, Kastovsky, Mares, & Johansen, 2014). It's dominance in eutrophic aquatic ecosystems is associated with a variety of factors particular to cyanobacteria, including possession of phycobilins (Sobiechowska-Sasim, Ston-Egiert, & Kosakowska, 2014), production of gas vesicles (Walsby, 1994), ability of certain species to fix nitrogen (Fay, 1992), and algae growth inhibition attributable to the ability of cyanobacteria to produce allelopathic chemicals (Gantar

et al., 2008; Pflugmacher, 2002). Recent global warming, coupled with nutrient loading in waterbodies, has catalysed the buildup of cyanobacteria blooms resulting in others producing harmful cyanotoxins, such as the genus Microcystis (Amorim, de Moura-Falcao, Valenca, de Souza, & do Nascimento Moura, 2019; Sivonen & Jones, 1999). Toxins produced by cyanobacteria are both secondary metabolites and diverse, including microcystin (MC) and nodularins cyclic peptides that inhibit protein phosphatases.

MC, the most frequently occurring and widespread cyanotoxins, are formed within algal cells, mainly from cyanobacterial blooms which are released into the water column (Merel et al., 2013; Sivonen & Jones, 1999). During a cyanobacteria bloom event, MC remain within cells, only being emitted when cells are lysed as a result



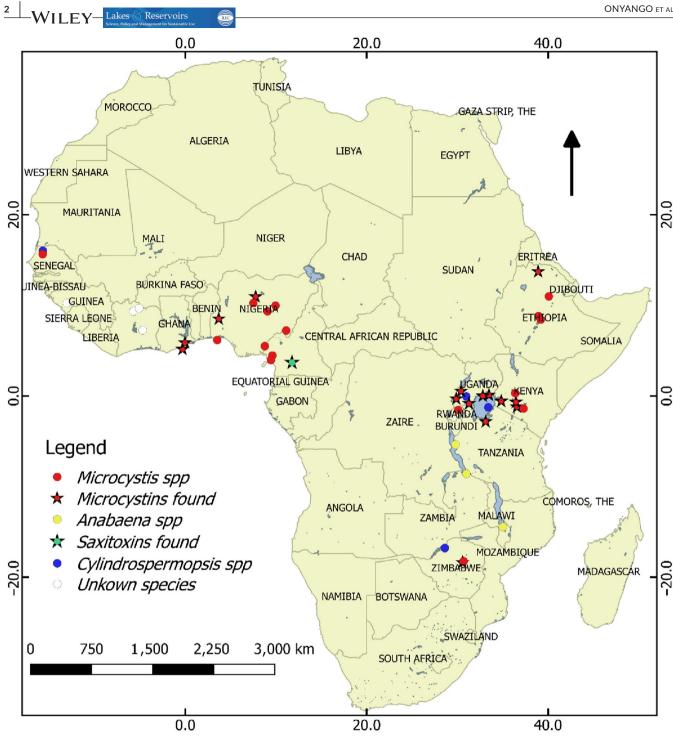


FIGURE 1 Map of tropical Africa showing locations of cyanobacterial blooms (adapted from Mowe et al. 2015)

of natural cell death and senescence, with neurotoxic, hepatotoxic or dermatotoxic effects. The emission of toxins also may be propagated by evolutionary-derived or environmentally mediated circumstances, including allelopathy or relatively sudden nutrient limitation (Merel et al., 2013).

Cyanobacteria is wildly seen in many equatorial eutrophic lakes, including Lake Victoria, because of elevated temperatures and eutrophication which accelerates algal growth and breakdown, culminating in the release of toxins. The released cyanotoxins, with MC being the most frequently detected, constitute part of

the biological water contaminant pathways for fish, livestock and human beings through water, aquatic vegetation and fish intake (Chorus & Bartram, 1999; Sivonen & Jones, 1999; Zhang et al. 2009; Badar et al., 2017). On a global scale, MC exhibits more than 90 variants of toxicity identified to date, with MC-LR being the most toxic and frequent (Pearson, Mihali, Moffitt, Kellmann, & Neilan, 2010). Thus, they have a potential negative impact on the food web with these effects increasing via bioaccumulation in the food web (Chen, Xie, Guo, Zheng, & Ni, 2005). Microcystins-LR, which is a water-soluble MC variant, remains relatively chemically stable in

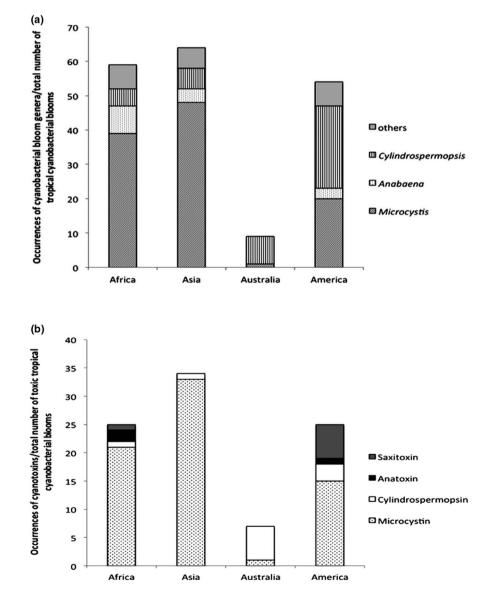
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the water column over time after their release. This can result in increased bioavailable extracellular cyanotoxins that can accumulate along the ecological food chain, resulting in animal poisoning and deaths worldwide (Lambert, Holmes, & Hrudey, 1994; Codd et al. 2005; Nizan, Dimentman, & Shilo, 1986; Fogg, Stewart, Fay, & Walsb, 1973). Depending on the length and route of exposure, MCs effects on fish can include damage or perturbation on the testis and ovaries, the hypothalamic-pituitary-gonadal (HPG) axis, embryonic hatching, survival and growth rate declines, changes in behavior, impaired osmoregulation, increased liver activities and heart rate, and histopathological effects (Lone, Koiri, & Bhide, 2015; Malbrouck & Kestemont, 2006; Svircev et al., 2010). Upon its ingestion, liver, muscle and viscera are the main MC accumulation organs. This can ultimately result in liver failure by inhibiting the protein phosphatases 1 and 2 (PP1 and PP2), leading to cellular toxicity, apoptosis, cancer and structural damage among others (Vaterio, Vasconcelos, & Campos, 2016). Lone et al. (2015) have shown that MCs exhibit genotoxicity attributable to their ability to damage DNA and promote tumor, while Annadotter et al. (1999) and Zhou, Hai, and Kun

(2002) reported on the correlation between MC levels in drinking water and incidence of primary liver cancer (PLC).

Microcystin can affect various trophic levels in the aquatic ecosystem, with concentrations increasing with exposure period and environmental concentration (Schmidt et al. 2013). An increased periodicity and intensity of harmful algal blooms occurring in many aquatic ecosystems (Figure 1) has attracted global interest regarding the presence of MC (Mowe, Mitrovic, Lim, Furey, & Yeo, 2015). Other studies have further highlighted tropical cyanobacterial genera and cyanobacterial proportionality (Figure 2).

Microcystin persistence and detoxification in aquatic organisms is a critical focus for fishery economics and public health, with several emerging ecological and public health issues focusing on plant and animal communities. This situation is attributed to the anthropogenic effects, mainly nutrient loads to freshwater systems and the related total biomass and composition alteration of algal communities (Beasley, Cook, Dahlem, Lovell, & Valentine, 1989; Galey et al., 1987; Hilborn & Beasley, 2015). Thus, the study of MC in Lake Victoria is critical to its aquatic toxicology and fauna effects in this



**FIGURE 2** Proportion of tropical cyanobacterial genera out of total tropical cyanobacteria blooms (source: Mowe et al. 2015)

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4 WILEY-Lakes Reservoirs

lake. Accordingly, the present study (a) reviews the current state of research regarding microcystis in Nyanza Gulf of Lake Victoria, (b) identifies knowledge gaps and (c) proposes future research directions.

#### 2 MICROCYSTIN IN NYANZA GULF OF LAKE VICTORIA

Over the last three decades, Lake Victoria, the world's second largest freshwater lake, has experienced major water guality deterioration over time attributable mainly to pollution from increased eutrophication, a trend associated with urbanization, agricultural activities and deforestation (Hecky, Muggide, Ramlal, Talbot, & Kling, 2010; Kolding, Medard, Mkumbo, & van Zwieten, 2014). Nyanza Gulf is the most eutrophic bay of Lake Victoria (Simiyu, Oduor, Rohrlack, Sitoki, & Kurmayer, 2018) because of nutrient enrichment originating from subsistence agricultural activities (Gikuma-Njuru, Hecky, Guildford, & MacIntyre, 2013). The high eutrophication levels have resulted in regular cyanobacterial blooms, some with the potential of producing cyanotoxins toxic to fish (Malbrouck & Kestemont, 2006). The most frequently occurring cyanotoxins are the hepatotoxic microcystins (MC) emitted by cyanobacteria (Table 1), including Microcystis, Dolichospermum (Anabaena), Planktothrix (Oscillatoria) and Nostoc (Meriluoto, Spoof, & Codd, 2017).

MC toxicity is based on the strong inhibition of the protein phosphatases (PP1 and PP2a), disturbing cytoskeleton formation in eukaryotic cells. Microcystin is associated with high eutrophication levels associated with increased nutrient concentrations and phytoplankton growth (Markensten, Moore, & Persson, 2010), resulting in hypoxic conditions in the lake. Several researchers, including Sitoki, Kurmayer and Rott (2012), reported positive results regarding the presence of microcystin in Lake Victoria, linking it with seasonality (Figure 3). Miles et al. (2013) reported the presence of putative microcystin analogues in his study of Mwanza Gulf in Lake Victoria. Recent studies by Simiyu et al. (2018) found the presence of higher microcystin levels (62 ± 7 ng/g fish dry weight) in Nyanza Gulf (Kisumu bay) of Lake Victoria, compared to the open waters of the Rusinga Channel (14  $\pm$  0.8 ng MC/g). These studies confirm higher cyanobacterial blooms within Winam Gulf, Mwanza Gulf and Murchison Bay (Ochumba and Kibbara, 1989; Sekedende et al. 2005; Haande et al., 2007). Semyalo, Rohrlack, Naggawa, and Nyakairu (2009) studied the presence of microcystin in Nile tilapia within Murchison Bay, finding a positive correlation of microcystin in fish gut with that in the water. He also noted an increase in microcystin-emitting cyanobacteria in Lake Victoria may have resulted in increased intake of microcystin in filter-feeding Nile tilapia (Table 2).

It is also worth noting, however, that fish such as tilapia are likely to have a detoxification system able to metabolize ingested toxins to less toxic states (Wiegand et al., 1999). Despite the likelihood of detoxification, other studies also have confirmed toxin accumulation in tissues, especially among planktivorous organisms, through ingestion of toxic microcystis in their diet and via food web transfer (Li, Chung, Kim, & Lee, 2004; Xie et al., 2004). The increased toxins and their effects to human and the aquatic environment are of great concern within freshwater systems. Severe MC exposure to fish has proved to result in high incidences of liver necrosis, growth retardation, impaired reproductive ability and mass mortalities (Malbrouck & Kestemont, 2006; Ochumba, 1990). These effects result from several factors, including reduced oxygen levels in the lake with a negative effect to the overall productivity of the lake. Fish and contaminated water are among the key MC exposure routes to humans, especially when the WHO recommended daily intake limit of 0.04  $\mu$ g/kg for fish and 1  $\mu$ g/L for water intake are exceeded (Jia, Luo, Lu, & Giesy, 2014; Okello, Portmann, Erhard, Gademann, & Kurmayer, 2010; WHO, 2003). Microcystin may not immediately pose a health threat to human beings upon consumption, but has potential long-term negative implications.

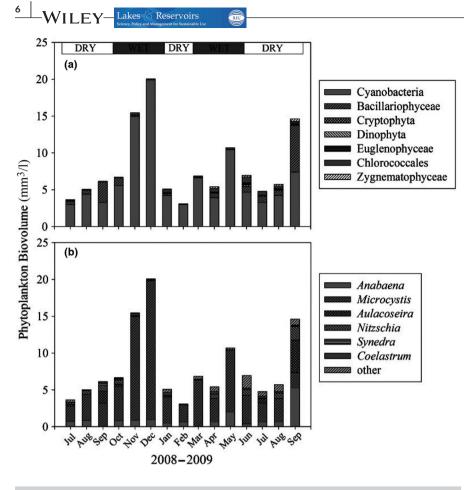
Despite the likelihood of continued ingestion of cvanobacteria by fish, even in the presence of microcystin which ultimately accumulates in fish tissue (Zhao, Zhu, Yang, Gan, & Song, 2006), research has confirmed that fish have the ability to physiologically deal with microcystin through biliary excretion (Sahin, Tencalla, Dietrich, & Naegeli, 1996), and behaviorally through lowering their feeding rate in the existence of toxic algae (Keshavanath et al., 1994). On the other hand, fish may change their behaviour to consume large amounts of cyanobacteria as a means of controlling the blooms in the lake (Datta & Jana, 1998). Substantial ingested quantities, however, may accumulate in fish livers and muscles, subsequently being transferred along the food web (Lance et al., 2014). Semyalo et al. (2009) recommended examination of microcystin accumulation at different levels in the fish food web in order to better understand the effects. Nile tilapia, being a generalist feeder, usually accumulates substantial quantities of microcystin in its diet. Previous research on tilapia diets in Lake Victoria have demonstrated cyanobacteria (especially microcystis sp.) can constitute about 30% of the phytoplankton community (Haande et al., 2007; Semyalo et al., 2009). Simiyu et al. (2018) noted the level of microcystin in Lake Victoria depends on the location, with open waters containing lower concentrations (56  $\pm$  56 ng MC/g DW), compared to gulfs and shore areas (190  $\pm$  51 to 543 ± 26 ng MC/g DW). Substantial quantities of microcystin absorbed by fish accumulates in the liver and muscles, subsequently being transferred up the food chain.

## 3 | BIOACCUMULATION AND BIOMAGNIFICATION

Bioaccumulation refers to the buildup of substances such as toxins or other chemicals in the body of an organism. It occurs when an organism ingests a substance at a rate faster than which it is eliminated through catabolism and excretion. This means the longer the biological half-life of a toxic substance, the higher the risk of chronic poisoning, even in spite low levels of the toxin in the environment. Studies have confirmed the intensity of toxin accumulation is not only based on a species feeding guild but is also dependent on

IABLE I SUM	Summary or cyanobacterial blooms, prevalence and toxins reported in tropical Arrica (Mowe et al., 2012)	valence and toxins reported in th	орісаї Агліса (імоме ет аг., 2015)		
Country	Potential toxic species found	Toxin test	Toxins found	Quantity of toxins	References
Cameroon	Planktothrix mougeotii, Oscillatoria putrida & Microcystis spp. (M. aeruginosa, M. wesenbergii)	Not tested	Not tested	Not tested	Kemka <i>et al.</i> (2013), Green (2010)
Cote d'Ivoire	Unknown bloom-forming species	Not tested	Not tested	Not tested	Bouvy et al. (1998), Arfi et al. (2001)
Ethiopia (2011)	Microcystis sp.*	ELISA	No MC found	No MC found	Gremberghe et al. (2011)
Ghana	Microcystis aeruginosa* & Anabaena flos-aquae	HPLC	MC-RR	0.03-3.21 μg/L	Addico, Hardege, Komarek, Babica, & de Graft-Johnson, (2006)
Kenya	Microcystis aeruginosa*, Arthrospira fusiformis* & Anabaenopsis abijatae*	ELISA/HPLC-MALDI-TOF	MS MC-LR & Anatoxin-a	MC: 1.6-39.0 μg/L Anatoxin:0.5-2.0 μg/L	Ballot, Krienitz, Kotut, Wiegand and Pflugmacher (2005), Haande et al.(2007), Kotut, Ballot, Wiegand and Krienitz (2010)
Malawi	Anabaena sp.	Not tested	Not tested	Not tested	Gondwe, Guildford and Heck (2007)
Nigeria	Cylindrospemopsis sp.*, Anabaena sp.*, Microcystis sp.*(Microcystis aeruginosa, M. flos-aquae, M. wesenbergii), Lyngbya sp.*, Aphanizomenon flos-aquae, Oscillatoria limnetica & Anabaena spiroides	ELISA/HPLC-MALDI-TOF	MC, CYN, Anatoxin-a, Anatoxin-a (S),STX	МС:1.4- 3.8 µg/L	Anadu, Obiaha and Ejike (1990), Kemdirim (2000), Ezra and Nwankwo (2001), Akin-Oriola (2003), Akin-Oriola, Anetekhai and Oriola (2006), Odokuma and Isirima 2007), Chia, Abolude, Ladan and Kalaboms (2009), Okechukwu and Ugwumba (2009), Onyema (2010), Ajuzie (2012)
Senegal	Microcystis aeruginosa &(2006);Cylindrospermopsis raciborskii	Mouse bioassay	No CYN/STX found	No CYN/STX found	Berger <i>et al.</i> (2006), Bouvy <i>et al.</i> (2006), Dufour, Sarazin, Quiblier, Sane and Leboulanger (2006)
Tanzania	Anabaena sp.,Microcystis sp.*	HPLC-DAD/-MALDI-TOF	MC-RR	0-1.0 µg/L	Sekadende, Lyimo and Kurmayer (2005)
Uganda	Microcystis aeruginosa, M. flos-aquae, Anabaenopsis spp., Aphanizomenon sp., Anabaena sp. & Cylindrospermopsis raciborskii	ELISA,HPLC/ MALDI-TOF&LC-MS/MS	MC-RR,(Asp <sup>3</sup> )MC-RR, MC- YR,(ASP <sup>3</sup> ) MC-YR, MC-LR, MC-RY,(Asp <sup>3</sup> ) MC-RY	0.2-61.2 µg/L	Ganf (1974), Komarek and Kling (1991), Oliver and Ganf (2000); Haande et al. (2007); Haande <i>et al.</i> (2007), Green (2010), Okello et al.(2010), Okello and Kurmayer (2011), Poste, Hecky and Guildford (2013)
Zimbabwe	Microcystis aeruginosa*, M. wesenbergii, M. novacekii, C. raciborskii, Lyngbya sp., Anabaena sp., Aphanizomenon sp. & Oscillatoria sp.	ELISA	MC-LR	0.2-22.48 µg/L	Ramberg (1987), Magadza (2006), Mhlanga, Day, Chimbari, Siziba and Cronberg (2006a), Mhlanga <i>et al.</i> (2006b), Magadza (2008-2009), Kunz (2011), Tendaupenyu (2012)

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**FIGURE 3** Proportion of different cyanobacterial toxins out of total number of tropical toxic cyanobacterial blooms (source: Mowe et al. 2015)

	Selectivity index (log Q)							
	Lake Mburo			Murchison				
Genus	Minimum	Mean	Maximum	Minimum	Mean	Maximum		
Microcystis	3.56	3.92	3.99	2.99	3.87	3.99		
Anabaena	- 1.26	0.93	3.06	-1.22	1.75	3.64		
Aphanocapsa	2.06	2.14	2.21	N/A	N/A	N/A		
Other cyanobacteria	- 0.39	0.89	3.48	-0.22	-0.11	0		

**TABLE 2** Selectivity index log Q of major cyanobacteria groups found in the diets of Nile tilapia fish in Lake Mburo (n = 22) and Murchison Bay, Lake Victoria (n = 44; N/A = not available; Source:Semyalo et al., 2011)

the environmental toxin concentration and rate of accumulation in a fish relative to depuration (Ibelings & Chorus, 2007; Kozlowsky-Suzuki, Wilson, & da Ferrão-Filho, 2012). MC bioaccumulation has been confirmed to exhibit different pathological effects on different fish species, with phytoplanktivorous species being more tolerant to MC effects (Xie et al., 2004).

In contrast, biomagnification refers to the increasing concentration of a substance within the tissues of organisms existing at successively higher levels in a food chain. This occurs as a result of persistence of the substance, food chain energetics and/or or low or non-existent rates of internal degradation or excretion. The toxin levels usually build up along the food chain. Nevertheless, there is inadequate knowledge on toxin biomagnification (Flores, Miller, & Stockwell, 2018; Kozlowsky-Suzuki et al., 2012). Although recent studies have proved fish and human accumulation of toxins is associated with the environmental concentration of particular toxins, coupled with the exposure period (Gurbuz, Uzunmehmetoglu, Diler, Metcalf, & Codd, 2016; Jia et al., 2014), the MCs concentration up the trophic level is limited because of toxin depuration (Ferrao-Filho and Kozlowsky-Suzuki, 2011; Zhang et al. 2009). It is important to note, however, that different studies have resulted in conflicting outcomes in relation to microcystin accumulation up the trophic levels because of their spatial temporal trends (Snyder, 2015; Xie et al., 2005; Zhang et al.2009).

## 4 | MICROCYSTIN IMPACTS IN FISH

Enhanced production of cyanotoxin-producing cyanobacteria is associated with nutrient loading into waterbodies primarily resulting from human activities. Anthropogenic nutrient inputs to freshwater systems has continued to shift the total biomass and composition of algal communities and subsequently fish, particularly for eutrophic freshwater lakes. Cyanobacteria blooms

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in highly eutrophic lakes can provide a rich and abundant source of nutrients for fish, including Roach (*Rutilus rutilus*), Silver carp (*Hypophthalmichthys molitrix*) and Nile tilapia (*Oreochromis niloticus*) (Bwanika, Chapman, Kizito, & Balirwa, 2006; Chen, Xie, Zhang, Ke, & Yang, 2006; Kamujunke, Schmidt, Pflugmacher, & Mehner, 2002). This is not always the case (Bednarska, 2006), however, since blooms are often dominated by colonial and filamentous forms that can cause mechanical interference for zooplankton grazers and clogging of gills in fish (Landsberg, 2002), leading to a decreased food web and subsequent fish population. The presence of aqueous and cell-bound cyanotoxins in a fish diet is not good for their morphology, physiology, behaviour and ultimately their survival (Malbrouck & Kestemont, 2006). The presence of cyanotoxins such as microcystins can induce stress in fish (Baganz, Staaks, Pflugmacher, & Steinberg, 2004) that can in turn lead to low foraging rates (Beveridge et al., 1993). Different effects

TABLE 3	Microcystins detected by LC-MS <sup>2</sup>	$^{2}$ (Method A) with and without thiol derivatization in standards and in extracts fro	m
Microcystis	bloom in Lake Victoria (BSA6) <sup>a</sup>		

Underivatized sample			Mercaptoethanol derivative		MEMHEG derivative		Abundance in sample		
Rt (min)	[MH] <sup>+</sup> m/z	Microcystin	Status	Rt (min)	[MH] <sup>+</sup> m/z	Rt (min)	[MH] <sup>+</sup> m/z	Standard	BSA6
1.8	1,038.5	MC-RR ( <b>3</b> )	Confirmed	1.81	1,116.5	2.59	1,394.5	Major	Major
3.3	1,031.5	[Asp <sup>3</sup> ]MC-YR ( <b>20</b> )	Tentative	3.35	1,109.5	4.1	1,387.5	ND	Minor
3.46	1,063.5	[Mser <sup>7</sup> ]MC-YR ( <b>14</b> )	Tentative	NR	-	NR	-	ND	Major
3.51	1,013.5	[Mser <sup>7</sup> ]MC-LR ( <b>15</b> )	Tentative	NR	-	NR	-	ND	Major
3.59	1,031.5	[Dha <sup>7</sup> ]MC-YR ( <b>21</b> )	Tentative	3.49	1,109.5	-	1,387.5	Minor	Minor
3.61	1,045.5	MC-YR (2)	Confirmed	3.63	1,123.5	4.33	1,401.5	Major	Major
3.68	981.5	[Asp <sup>3</sup> ]MC-LR ( <b>17</b> )	Tentative	3.69	1,059.5	4.26	1,337.5	Minor	Minor
3.77	995.5	MC-LR (1)	Confirmed	3.71	1,073.5	4.47	1,351.5	Major	Major
3.79	981.5	[Dha <sup>7</sup> ]MC-LR ( <b>8</b> )	Confirmed	3.84	1,059.5	4.5	1,337.5	Minor	Minor
4.02	1,009.5	MC-HilR ( <b>11</b> )	Tentative	3.93	1,087.5	4.68	1,365.5	ND	Minor
4.05	1,029.5	MC-FR ( <b>12</b> )	Tentative	3.97	1,107.5	ND	1,385.5	ND	Minor
4.9	953.5	MC-RA ( <b>10</b> )	Tentative	4.78	1,031.5	5.44	1,309.5	ND	Major
5.01	1,031.5	[Asp <sup>3</sup> ]MC-RY ( <b>16</b> )	Tentative	4.86	1,109.5	5.41	1,387.5	ND	Minor
5.07	1,063.5	[Mser <sup>7</sup> ]MC-RY ( <b>22</b> )	Tentative	NR	-	NR	-	ND	Minor
5.12	1,031.5	[Dha <sup>7</sup> ]MC-RY ( <b>23</b> )	Tentative	4.94	1,109.5	5.56	1,387.5	ND	Minor
5.29	1,045.5	MC-RY ( <b>9</b> )	Confirmed	5.22	1,123.5	5.79	1,401.5	ND	Major
5.37	1,075.5	MC-RY(OMe) ( <b>24</b> )	Tentative	5.3	1,153.5	5.88	1,431.5	ND	ND
5.44	967.5	MC-RAba ( <b>25</b> )	Tentative	5.29	1,045.5	5.92	1,323.5	ND	Minor
5.93	981.5	MC-RApa(1) ( <b>26</b> )	Tentative	5.76	1,059.5	6.34	1,337.5	ND	Minor
6.09	981.5	MC-RApa(2) ( <b>27</b> )	Tentative	5.89	1,059.5	6.48	1,337.5	ND	Minor
6.34	995.5	MC-RL (28)	Tentative	6.15	1,073.5	6.71	1,351.5	ND	Minor
6.37	1,029.5	MC-RF (13)	Tentative	6.22	1,107.5	6.74	1,385.5	ND	Major
8.09	960.5	MC-YA ( <b>29</b> )	Tentative	7.83	1,038.5	8.52	1,316.5	ND	Minor
8.36	910.5	MC-LA (4)	Confirmed	7.83	988.5	8.58	1,266.5	Major	Minor
8.55	1,002.5	MC-LY ( <b>6</b> )	Confirmed	8.1	1,080.5	8.72	1,358.5	Major	ND
8.7	1,032.5	MC-LY(OMe) (18)	Tentative	8.23	1,110.5	8.84	1,388.5	Minor	ND
8.91	974.5	MC-YAba ( <b>30</b> )	Tentative	8.58	1,052.5	9.23	1,330.5	ND	Minor
9.15	924.5	MC-LAba ( <b>31</b> )	Tentative	8.5	1,002.5	9.18	1,280.5	ND	Minor
9.8	1,025.5	MC-LW (7)	Confirmed	9.28	1,103.5	9.84	1,381.5	Major	ND
10.3	986.5	MC-LF (5)	Confirmed	9.59	1,064.5	10.13	1,342.5	Major	ND
10.45	952.5	MC-LL ( <b>19</b> )	Tentative	9.8	1,030.5	10.21	1,308.5	Minor	ND

<sup>a</sup>NR, no reaction; ND, not detected; identities considered confirmed only when peaks possessed identical retention time and mass spectral fragmentation to authentic standards and were considered tentative when these properties were consistent with proposed structure in absence of standard; retention times for thiol derivatives are for most abundanst diastereoisomer; compounds were denoted as "Major" when they constituted more than ca 10% of most abundant microcystin in sample; Source: Miles et al. (2013).

7

-WII FV-Lakes & Reservoirs

of microcystin and the potential consequences to fish have been reported for aquatic food webs. Malbrouck and Kestemont (2006), for example, reported that fish exposure to microcystin in a dosedependent manner in early life stages can results in perturbations to embryonic hatching, decreased survival and growth rates, and histopathological effects, including enlarged and opaque volk sac, small heads, curved body and tail. They also reported that ingested microcystin accumulates in the liver, muscle and viscera of juveniles and adults and can affect fish behavior, growth rate and osmoregulation. Depending on the microcystin exposure route, the effects of microcystin concentration in the water and the food chain can be many and varied. Sonia and Ramanibai (2015) found a decrease in the body weight and death after eight days in Poecilia sphenops after exposing them to the cyanobacteria Microcystis *aeruginosa* in the diet at a concentration of  $6 \times 10^4$  cells/ml to each individual fish. Exposure to microcystin can produce architectural changes in the liver, disturb parenchyma formation of the liver and enlarged hepatocytes with granular cytoplasm. Fish exposure to this microcystis concentration resulted in hepatocyte necrosis, further displaying ruptured follicle walls in the oocyte, while the compactness of the tissue also was disturbed. There was less yolk in the developing oocyte, deformed follicle cells and incidences of ovary necrosis. Weight loss and fish mortality was likely a result of a combination of stress and organ damage.

Microcystins, particularly MC-LR, a monocyclic peptide hepatotoxin, was found to accumulate mainly in the liver of fish, as well as in the kidneys, gills and intestines (Mohammed & Hussein, 2006; Radbergh, Bylund, & Erikkson, 1991; Zurawell, Chen, Burke, & Prepas, 2005). Ayles et al. (1986) noted the impacts of cyanobacteria on fish populations is not due only to the toxins they release, but also to reduced oxygen levels in the waterbody resulting from as well as the collapse of the algal bloom. Shebulsky (1951), however, proved that fish death occurred even in the presence of an adequate oxygen supply, indicating toxins contributed partly to the death of fish. With the current human health movement toward consuming leaner meat, many people in Kenya have embraced fish eating, particularly the communities living along Lake Victoria, which are known to depend highly on fish as one of their staple meals, which could result in human exceedance of the average daily tolerance intake limit provided by World Health Organization of 0.04 µg/kg body mass per day for MC-LR (WHO, 1998; Chorus & Bartram, 1999). Thus, it is critical to assess microcystin exposure risks to human via fish consumption, especially among the traditional fish-eating communities.

MCs detected by LC-MS<sup>2</sup>, with and without thiol derivatization in standards, have confirmed the presence of MC-LR, a lethal microcystin, in Lake Victoria (Table 3). Among other organs, the effects of MC-LR on fish livers has also been noted to extend its human health effects, with almost similar organs affected. As fish consumption grows in the country against a declining fisheries capture, the risk of exposure to MCs through fish intake are real (Poste, Hecky, & Guildford, 2011). Assessment of human health impacts associated with limited water supply and sanitation within a given area requires epidemiological studies to establish dose-response relationships (DRRs). Although often expensive and resource demanding, this will link environmental variables with observable health impacts (World Bank, 1998). In cases where drinking water is contaminated by cyanotoxins, however, bottled water or different sources of water are likely alternatives, even though they may require more time, energy and possibly funds to obtain such alternatives. Three categories of impacts are used to estimate the monetary consequences of illness to society attributable to water pollution, including (a) income losses due to illness, (b) health care expenditures to society and/or affected individuals, and (c) pain, suffering and/or inconvenience experienced by affected individuals. The costs associated with the two first categories can be determining by directly observing these factors (Ready et al., 2004). Establishing quantitative epidemiological relationships is the main data-demanding task in comparing the benefits and costs of measures to reduce algal toxins. Concluding whether or not liver damages within a given population is a result of cyanobacterial intoxication requires determining which drinking water sources is contaminated, who drinks it, and the particular population risk levels of suffering liver disease, compared to those drinking water from different water sources (Kuiper-Goodman, Falconer & Fitzgerald, 1999). Losses from fish sales also have direct effects on both national and individual revenues, as well as household nutritional requirements particularly around the affected areas. Monitoring of toxigenic and non-toxigenic algae are very costly to any nation's GDP (Reichwaldt and Ghadouani, 2012; Falconer & Humpage, 2005). However, the implications of unmonitored cyanotoxin emissions, particularly MC in eutrophic freshwater bodies that include Lake Victoria, can have far-reaching effects on aquatic ecosystem balances and human health.

## 5 | CONCLUSIONS AND RECOMMENDATIONS

Lake Victoria, like other freshwater eutrophic lakes, is experiencing cyanobacterial build-up resulting from increased nutrient loads associated with poor agricultural practices and domestic and industrial wastes. Being the second largest freshwater lake in the world, and the largest in Africa, the lake contributes significantly to food security and fish species richness. There is great risk of losing the diversity of fish species in the lake to the effects of cyanotoxins and further impact human health through fish consumption and contaminated water use. The commercially important fish species of the lake, with the exception of Lates niloticus, are herbivorous, thereby being predisposed to high levels of cyanobacterial microcystis, despite biodilution capability along food web. There is a paucity of information on microcystin biomagnification in fish. There is evidence of microcystin bioaccumulation in the muscle, liver and viscera of fish, therefore posing a health risk not only to fish performance and species diversity through disrupted food webs, but also to human health. With people embracing a healthier diet, they tend to consume more fish, a lean and healthy protein source. Thus, depending on the intake level, they may end up exceeding the average daily

tolerance limit of 0.04  $\mu$ g/kg body weight recommended by the World Health Organization. Thus, it is critical to assess the seasonal concentration levels of various microcystin species in Lake Victoria and their bioaccumulation pathways, especially for commercially-important fish species, in order to inform lake management and the fish food industry.

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10

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