

Toxic cyanobacteria and their toxins in standing waters of Kenya: implications for water resource use

Kiplagat Kotut, Andreas Ballot and Lothar Krienitz

ABSTRACT

Phytoplankton biodiversity studies in Kenya's standing waters were carried out between 2001 and 2003. Toxin producing cyanobacteria were recorded in twelve water bodies. *Microcystis* and *Anabaena* were the most common species in freshwaters while *Anabaena* and *Anabaenopsis* were common in alkaline saline lakes. Seven lakes with cyanobacteria blooms and a hot spring had detectable levels of microcystins and anatoxin-a. Cell bound microcystins (LR equivalents) concentration ranged from 1.6–19800 $\mu\text{g g}^{-1}$ Dry Weight (DW) while anatoxin-a varied from below the limit of detection to 1260 $\mu\text{g g}^{-1}$ DW. In alkaline-saline lakes, microcystins and anatoxin-a were also present in stomach contents and liver samples of dead flamingos. Monoculture strains of *A. fusiformis* from Lakes Sonachi and Bogoria had detectable levels of microcystins while anatoxin-a was present in strains isolated from Lakes Sonachi, Bogoria and Nakuru. Two freshwater sites, Nyanza Gulf (L. Victoria) and Lake Baringo recorded cyanotoxin concentration exceeding WHO's upper limit of 1.0 $\mu\text{g l}^{-1}$ for drinking water. The results confirm that cyanotoxins could have played a role in the mortality of flamingos in Lakes Bogoria and Nakuru. The implications of these findings on water resource use, measures to be taken to reduce the risk of exposure and eutrophication control steps to reduce cyanobacteria bloom formation are considered in this paper.

Key words | algal blooms, cyanotoxins, eutrophication, flamingos, Kenya, phytoplankton

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INTRODUCTION

Toxin production among microalgae and associated problems

Among the many algal secondary metabolites that have been identified, a number are potent toxins that have been associated with human illnesses, marine mammal and bird morbidity and mortality, and extensive fish kills (Van Dolah 2000). These toxins are produced by a few dozen of the thousands of species of algae that have been described. Toxic algal poisoning in most cases does not occur unless there is a heavy bloom in the water. However, many blooms are not hazardous even when dominated by toxin producing species. Hence toxic compounds are frequently, but not universally, associated with algal blooms (Plumley 1997; Van Ginkel

2003). The term 'harmful algal bloom' or simply HAB is presently used to describe the bloom phenomenon that contains harmful toxins or causes other negative impacts.

Toxins of human health significance originate primarily from three classes of unicellular algae; dinoflagellates, diatoms, and cyanobacteria. Among the cyanobacteria, over forty species mostly found in freshwater have been implicated in the generation of toxic blooms (Carmichael 1997). However, even within known toxin producing species (toxigenic), isolated strains may be non-toxic (atoxigenic). Both atoxigenic and toxigenic strains can occur in the same bloom (Dow & Swoboda 2000). It is not yet clear as to which factors regulate toxigenic and atoxigenic characteristics. Toxicity can vary between clones from the same

isolate (Carmichael 1992). Similarly, some strains produce three or more toxins with the relative proportion being influenced by the environment (Carmichael 1992).

Comparatively, most of the widely reported incidences associated with toxic algae are the poisoning of both domestic animals and wildlife, notably in the developed nations of Europe, North America, and Australia. This is perhaps because unlike humans who easily get repulsed by the sight of the bloom, animals are generally oblivious to the danger posed. In North America, Europe and Australia, mass poisonings of wild and domestic birds, associated with cyanobacterial blooms and scum, have been recorded for several years (Yoo *et al.* 1995). In England, 20 sheep and 15 dogs died after ingesting a microcystin scum in 1989 (Codd & Beattie 1991; Lawton & Codd 1991). In 1990, several dogs died after ingesting neurotoxic *Oscillatoria* scum in Loch Insh, Scotland (Codd & Beattie 1991; Gunn *et al.* 1992). Other examples include the net pen liver disease of pen-reared salmon in Canada (Stephen *et al.* 1993), the death of alligators in Lake Griffin, USA (Pollack 1999) and the mass death of the wild duck in Japan (Matsunaga *et al.* 1999).

In Africa, problems associated with algal toxins have been documented in a few countries with the majority of the cases being reported from South Africa (Harding 1997; Harding *et al.* 1995; Scott 1991; Van Ginkel 2003; Van Halderen *et al.* 1995; Wicks & Thiel 1990) where the toxic effects on animals have been known for over 50 years. Recently, the presence of cyanotoxins in ponds and reservoirs has been reported in Morocco (Nasri *et al.* 2004; Oudra *et al.* 2002).

Human health problems associated with cyanobacterial toxins have been on record for a long time. However, it is only recently that confirmed human death resulting from cyanotoxins poisoning has been reported. A widely reported incident is the serious outbreak of acute liver failure at the dialysis center of a clinic in Brazil in 1996, after the water supply to its haemodialysis units was contaminated with cyanobacteria. 76 of the 100 patients suffering from liver failure died (Pouria *et al.* 1998; Rinehart *et al.* 2001). Such a serious incident has not been reported for the African continent. However, this does not necessarily mean that the continent has been free of toxic algae problems. According to Zilberg (1966), gastroenteritis among school children in Harare, Zimbabwe in the 1960s could have been caused by

exposure to cyanobacterial toxins in the municipal drinking water supply. Hence the problem could be more widespread but going by the name of mystery diseases as was the case in Palm Island, Australia in the 1970s (Byth 1980). Presently, incidents associated with toxic alga and cyanobacteria appear to be on the rise globally (Luckas 2000).

Toxic microalgae problems in Kenya

The first officially confirmed case of toxic algal poisoning in Kenya is the widely reported death of thousands of marine wildlife along the Kenya Coast at the beginning of 2002 when huge numbers of fish, including manta rays, sharks and tuna were washed ashore (Tomlinson 2002). Several green and hawksbill turtles were also found dead. It is believed that their death was caused by a bloom of naturally occurring toxic algae triggered by an upwelling of nutrient-rich oxygen-poor deep ocean water (Agence France-Presse 2002; Wild Net Africa 2002).

Episodes of wildlife dying under mysterious circumstances have previously been reported for the inland waters of Kenya. The most widely reported case is the successive episodes of massive flamingo deaths recorded at lakes Nakuru and Bogoria in 1993, 1995, 1997 and 2000 (Okoko 2000). These deaths have raised a lot of concern among national and international conservationists. According to the World Wildlife Fund (WWF) experts, who have been studying the phenomenon, the deaths amount to a population decline of 20% in every two decades suggesting that unless the decline is reversed, the flamingo population would be extinct within one century (Wanjiru 2001). Initial investigations in Lake Nakuru indicated that high metal pollution was the primary cause of the flamingo deaths (Kairu 1996; Nelson *et al.* 1998). However, this explanation could not account for the flamingo die offs in Lake Bogoria, which is relatively free of heavy metal pollution (Wanjiru 2001). Later investigations also pointed at mycobacteria (Kock *et al.* 1999). The possibility of algal toxins being responsible for the die offs was acknowledged during these investigations, although no confirmatory studies were carried out. Domestic animal deaths suspected to have resulted from drinking contaminated water of impoundments have also been reported (Mwaura *et al.* 2004).

Study aims

The overall aim of the study was to document the biodiversity of phytoplankton communities in a selection of standing waters in Kenya and how their community structure and composition is related to some of the water quality problems experienced in the country. One of the specific aims of the study that forms the focus of this report was the identification of water bodies with known toxin producing algae. Additionally, toxin analyses were planned for water bodies with blooms of toxin producing cyanobacteria.

STUDY METHODS

Physico-chemical conditions

The analytical procedures for physical and chemical properties are outlined in Ballot *et al.* (2003), Ballot *et al.* (2004 a,b) and Krienitz *et al.* (2002, 2003). Briefly, temperature, conductivity, pH and salinity were measured directly in the field using a WTW Multiline P4 meter (Weilheim, Germany). Nutrient analyses (total nitrogen and total phosphorus) were carried out using a field nanocolor test kit and a field photometer (Nanocolor 300 D, Macherey, Germany). Total alkalinity was carried out titrimetrically using mixed bromocresol green – methyl red indicator and standard hydrochloric acid.

Phytoplankton analyses

Samples for determination of phytoplankton biomass and composition were collected on diverse dates between 2001 and 2003. Microscopic examination of phytoplankton samples from all the water bodies investigated was carried out using fresh and preserved samples. Enumeration of phytoplankton taxa in lake water samples was carried out on sedimentation chambers (Hydros-Bios Apparatebau GmbH Kiel, Germany) using a compound microscope (Eclipse TS 100 Nikon, Japan). Phytoplankton biomass was estimated by geometric approximations using a computer based count program (Opticount, Hepperle 2000) assuming a specific density of 1.

During the preliminary stage of the study, more than 46 water bodies (Figure 1) were investigated for physico-chemical

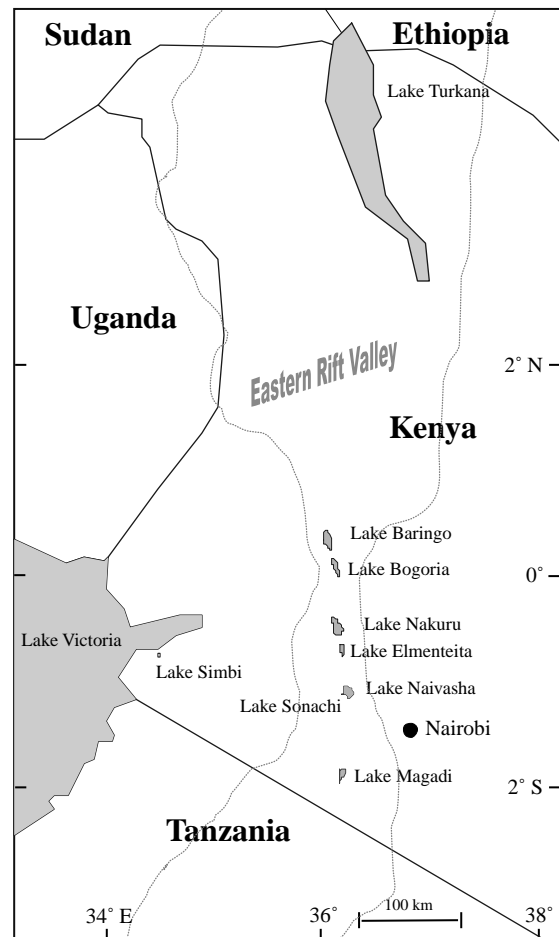


Figure 1 | Map showing the location of some of the water bodies investigated.

conditions and phytoplankton composition. These water bodies were selected from among the various categories of inland waters present in the country including the Rift Valley lakes, the Plateau lakes, Crater lakes, reservoirs and impoundments, small water bodies and sewage oxidation ponds. An output of the preliminary stage was the documentation of waters with cyanobacteria known to have a toxin producing potential.

Toxin analyses

Toxin analyses were carried out in water bodies with blooms of known toxin producing cyanobacteria and on the cyanobacterial mats from the hot springs of Lake Bogoria. Samples for toxin analyses were collected from each water body on diverse dates (Table 3). At each sampling site, a 1 litre

sample of water was filtered using 0.45 pore size Whatman Glass Fiber (GF/C) filters in the field. The seston retained by the filters was next air-dried and stored in the dark on transport to Germany and Scotland for toxin analyses. Water soluble toxins were fixed and enriched in the field by passing the filtrate above through Sep-Pak cartridges Plus tC18 (Milford, USA). Toxins in the filter materials were extracted with 70% ethanol while the enriched Sep-Pak cartridges were eluted with 90% methanol. Toxin concentrations were determined by the high performance liquid chromatography with photodiode detection array (HPLC-PDA) and matrix laser desorption/ionization time flight mass spectroscopy (MALDI-TOF). Details of the analytical procedures are provided in Ballot *et al.* (2003), Ballot *et al.* (2004a, b) and Krienitz *et al.* (2002, 2003). In total, five cyanotoxin samples were collected during the study period from one representative site of lakes Baringo, Sonachi, Bogoria and Elmenteita. Lake Nakuru was also sampled 5 times but on two separate study sites. Toxin samples from lakes Victoria and Simbi were collected once and twice respectively from a representative site of each lake. Mat samples for toxin analyses from Lake Bogoria hot springs were collected twice each from two different hot spring sites.

On June 13, 2001 and March 29, 2002, dying and dead flamingos were observed around Lake Bogoria hot springs and in Lake Nakuru. Known weights of tissue samples from

dead flamingos that included stomach contents, intestines, liver and fecal pellets were collected for toxin investigations. Toxin extraction was carried out in 70% methanol with 1% trifluoroacetic acid (Krienitz *et al.* 2003) and their analyses conducted at the School of Life Science, University of Dundee and at the Leibniz Institute of Freshwater Ecology and Inland Fisheries (working group, Biogeochemical Regulation), Berlin.

Isolated culture strains of *A. fusiformis* whose biomass dominated the phytoplankton of the saline lakes were also tested for cyanotoxins. To assess the potential risk of drinking lake water directly in Lakes Victoria and Baringo, an estimate of the toxin content per unit volume of lake water was computed using information on cyanobacteria biovolume and the toxin concentration.

RESULTS

Physico-chemical properties

In general, water temperature and dissolved oxygen fluctuated widely among and within lakes. The greatest fluctuation in dissolved oxygen occurred in the saline lakes where supersaturation was common following periods of relatively calm weather and near anoxic levels following sediment disturbance mostly by the flamingoes (Table 1).

Table 1 | Range in levels of the physico-chemical properties of the water bodies investigated for cyanotoxins

	Date	Temp. (°C)	DO (% sat.)	Median pH	Conductivity ($\mu\text{S cm}^{-1}$)	TA (meq l^{-1})	TN (mg l^{-1})	TP (mg l^{-1})
Nyanza Gulf	5/11/01	29.9	120	8.1	186	2.0	21.5	1.9
Baringo	6/12/01–26/5/02	24.9–26.3	58–88	8.8–9.1	1391–1659	13.3–15.9	0.5–8.0	0.6–1.3
Bogoria –HS	13/6/01	35–boiling	–	9.0	6410	78	≈0.5	0.02
Bogoria –lake	13/6/01–29/9/02	25.8–32.1	70–352	9.9–10.3	60000–72500	970–1724	0.6–8.2	5.4–33.6
Sonachi	18/6/01–1/6/02	21.3–30.1	260–500	10.0–10.4	15800–17330	196–420	0.5–5.2	0.9–5.6
Nakuru	9/6/01–29/5/02	17.1–32.1	3–503	10.1–10.6	5500–46900	166–815	3.3–11.3	2.4–40.0
Simbi	11/4/01–5/10/02	28.3–33.1	322	10.0–10.4	16200–17820	182–266	1.0–2.7	4.3–5.5
Elmenteita	15/6/01–31/5/02	22.5–32.3	30.3–170	9.8–10.5	23800–30300	226–400	1.7–18.5	0.6–7.6

Temp: Temperature; DO: Dissolved oxygen; TA: Total alkalinity; Sat: Saturation; TN: Total nitrogen; TP: Total phosphorus; HS: Hot Spring.

Water pH in all the lakes was generally high with the highest values being characteristic of the alkaline saline lakes. Electrical conductivity and total alkalinity had the widest variation with very low values in freshwater lakes and much higher values in the saline waters. A common characteristic of the lakes investigated for toxins was a high and variable concentration of nutrients (Table 1).

Distribution of cyanobacteria with toxin producing potential

Samples from twelve water bodies and one hot spring out of the more than 46 water bodies investigated had species of cyanobacteria known for toxin production. *Microcystis aeruginosa* was the most common cyanobacteria with the potential for toxin production in freshwater lakes such as Lakes Baringo and Naivasha. *Anabaena* spp. were common in samples of Nyanza Gulf of Lake Victoria. Other toxic cyanobacteria present in freshwater lakes were members of the genera *Synechococcus* and *Chroococcus*. The dominant cyanobacterium in the saline-alkaline Rift Valley lakes was *Arthrospira fusiformis*, which was often accompanied by members of the genera *Anabaena* and *Anabaenopsis* (Table 2). In hot springs mats on the shores of Lake Bogoria, the dominant toxin producing cyanobacteria are members of the genera *Phormidium*, *Oscillatoria* and *Synechococcus*.

Cyanotoxin findings

Out of the 12 water bodies and the hot spring that had toxic cyanobacteria, only seven natural lakes had cyanobacterial blooms. The seven lakes and the hot spring mats of Lake Bogoria were investigated for cyanotoxins. These lakes can be grouped into two categories; freshwaters serving as important sources of drinking water and alkaline saline waters, most of which are the habitats of the Lesser Flamingoes (*Phoeniconaias minor* Geoffrey) and other avian species. The freshwaters bodies studied were Lake Baringo and the Nyanza Gulf (Dunga Beach) of Lake Victoria while the alkaline saline waters were Lakes Elmenteita, Sonachi, Nakuru, Bogoria and Simbi (Figure 1). The majority of these lakes were characterized by a typical surface scum that imparted upon the lakes various shades of blue green to yellow colours depending on the dominant

cyanobacteria. The only exception was Lake Baringo whose colour was mostly dominated by clay suspensoids. Total phytoplankton biomass and cyanobacteria biomass were typically high in all these water bodies (Table 3). Detectable concentrations of toxins were recorded in all the water bodies investigated (including the hot spring mat samples) except Lake Elmenteita whose concentrations of microcystins and anatoxin-a were below the limit of detection by the methods used (Table 3). Dissolved cyanotoxins were below the limit of detection ($1 \mu\text{g l}^{-1}$) in all lake samples. Toxin concentration in the stomach contents of dead flamingos averaged $0.196 \mu\text{g}$ microcystin LR equivalent g^{-1} of Fresh Weight (FW) and $4.349 \mu\text{g}$ anatoxin-a g^{-1} FW (Krienitz *et al.* 2003). In liver samples, total microcystin ranged from 0.21 – 0.93 microcystin LR equivalent g^{-1} FW and from 1.76– 5.82 μg anatoxin-a g^{-1} FW (Codd *et al.* 2003a).

Microcystin concentrations of 2.2 and $15.02 \mu\text{g g}^{-1}$ DW were recorded from monoculture strains of *Arthrospira fusiformis* isolated from Lakes Sonachi and Bogoria respectively. Anatoxin-a concentrations of 0.3, 10.38 and $0.14 \mu\text{g g}^{-1}$ DW were measured from culture strains of *A. fusiformis* isolated from Lakes Sonachi, Bogoria and Nakuru in the same order. Toxins were not, however, detected in cultures of isolates of *A. fusiformis* from Lakes Simbi and Elmenteita.

Microcystin concentration of lake water at the Nyanza Gulf (L Victoria) was estimated at $1.065 \mu\text{g l}^{-1}$. In Lake Baringo microcystin concentration (microcystin LR equivalent) ranged from 0.08 to $3.25 \mu\text{g l}^{-1}$ while anatoxin-a varied from 0.05–0.2 $\mu\text{g l}^{-1}$. Hence in both water bodies, the cyanotoxin concentration sometimes exceeded the upper limit of $1.0 \mu\text{g l}^{-1}$ for drinking water set by the World Health Organization (Hitzfeld *et al.* 2000).

DISCUSSION

Toxic cyanobacteria in alkaline saline lakes

The presence of cyanotoxins in some of Kenya's alkaline saline lakes confirms long held suspicions that cyanotoxins could have played a major role in the mortality of flamingos in Lakes Bogoria and Nakuru. Observations made during

Table 2 | Distribution of cyanobacterial species globally known for toxin production in Kenyan water bodies

Water body, geographical position and altitude	Cyanobacteria	Abundance/comments
Lake Nakuru Pos. S 00°19.583'; E 36°05.325' Alt. 1782 m	<i>Anabaenopsis abijatae</i> <i>Anabaenopsis arnoldii</i> <i>Anabaena</i> sp. <i>Synechococcus</i> sp. <i>Arthrospira fusiformis</i> * <i>Synechocystis</i> sp.	Dominance and subdominance changes involving <i>A. abijatae</i> , <i>A. arnoldii</i> , <i>Anabaena</i> sp., <i>Synechocystis</i> sp and <i>A. fusiformis</i> were noted
Lake Baringo Pos. N 00°36.786'; E 36°01.395' Alt. 998 m	<i>Microcystis aeruginosa</i>	<i>M. aeruginosa</i> is dominant for a better part of the year
Bogoria Hot springs	<i>Synechococcus bigranulatus</i> <i>Oscillatoria willei</i> <i>Phormidium terebriformis</i>	Mats dominated by each species occur at different parts of the hot spring rivulets
Lake Bogoria Pos. N 00°36.786'; E 36°01.395' Alt. 1013 m	<i>Arthrospira fusiformis</i> * <i>Synechococcus</i> sp. <i>Synechocystis</i> sp. <i>Oscillatoria</i> sp.	<i>A. fusiformis</i> is dominant throughout the year with the rest occurring in small numbers
Lake Elmenteita Pos. S 00°27.345'; E 36°15.333' Alt. 1795 m	<i>Anabaenopsis abijatae</i> <i>Anabaenopsis arnoldii</i> <i>Synechococcus</i> sp.	<i>A. abijatae</i> and <i>A. arnoldii</i> are occasionally dominant
Lake Naivasha Pos. S 00°48.915'; E 36°18.897' Alt. 1942 m	<i>Microcystis</i> sp.	Occasionally subdominant.
Lake Oloiden Pos. S 00°49.025'; E 36°15.834' Alt. 1942 m	<i>Chroococcus</i> sp. <i>Anabaenopsis</i> sp. <i>Arthrospira fusiformis</i> *	Occasionally subdominant
Athi Basin Dam Pos. S 01°24.857'; E 36°55.687' Alt. 1556 n	<i>Microcystis</i> sp. <i>Anabaena</i> sp.	Both species occasionally subdominant
Lake Victoria (Nyanza Gulf) Pos. S 00°05.671'; E 34°42.362' Alt. 1143 m	<i>Anabaena flos-aquae</i> , <i>Anabaena discoidea</i> <i>Microcystis aeruginosa</i>	<i>A. flos-aquae</i> was dominant at the time of sample collection
Uhuru Park Pos. S 01°17.369'; E 36°48.995' Alt. 1688 m	<i>Microcystis</i> sp.	Occasionally dominant
Magadi Pos. S 02°00.168'; E 36°15.747' Alt. 627 m	<i>Arthrospira fusiformis</i> * <i>Anabaena</i> sp.	
Sonachi Pos. S 00°47.333'; E 36°15.150' Alt. 884 m	<i>Arthrospira fusiformis</i> <i>Anabaenopsis arnoldii</i>	<i>A. fusiformis</i> is most dominant
Simbi Pos. S 00°22.297'; E 34°37.904' Alt. 1145	<i>Arthrospira fusiformis</i> * <i>Anabaenopsis abijatae</i>	<i>A. fusiformis</i> is most dominant

*Cases where *A. fusiformis* is believed to be involved in toxin production.

Table 3 | Phytoplankton biomass and toxin concentration in Kenya's water bodies tested for cyanotoxins

	Date	Phytoplankton biomass (mg l⁻¹)	Cyanobacteria biomass (mg l⁻¹)	Microcystin (µg g⁻¹ DW) – LR equivalent	Microcystin variants	Anatoxin-a (µg⁻¹ DW)
Nyanza Gulf- a	5/11/01	282.2	269.1	39.15–41.4	LR, RR, LF & LA	Undetectable
Baringo- b	6/12/01–26/5/02	1.54–8.17	0.22–5.45	310–19800	LR, RR & YY	270–1260
Nakuru- c	9/6/01–29/5/02	6.7–104.3	5.3–96.4	129–4594	LR, RR, LF, LF & YR	5–223
Bogoria lake- c	13/6/01–29/9/02	62.2–777.1	61.1–769.1	16–227	LR, RR, LF, LF & YR	0.3–9
Bogoria HS- d	13/6/01	–	–	221–845	LR, RR, LF & YY	10–18
Elmenteita- c	15/6/01–31/5/02	17.5–202.0	14.1–196.7	Undetectable	–	Undetectable
Sonachi- e	18/6/01–1/6/02	312.8–3158.9	312.8–3158.4	1.6–12.2	RR	0.5–2.0
Simbi- e	11/4/01–5/10/02	61.2–348.3	61.2–348.3	19.7–39.0	LR, RR, LA & YR	1.4

*References to the detailed results; a – Krienitz *et al.* (2002); b – Ballot *et al.* (2003); c – Ballot *et al.* (2004a); d – Krienitz *et al.* (2003); e – Ballot *et al.* (2004b).
DW: Dry weight; HS: Hot spring.

the present study that support the contribution of cyanotoxin poisoning to flamingo deaths include a high concentration of toxins in Lake Bogoria hot spring algal mats, presence of toxins in the stomach contents of dead flamingos (Krienitz *et al.* 2003), lethal levels of anatoxin-a in liver samples of dead flamingoes and the behavioral pattern of the dying animals, staggering and opisthotonos, (Codd *et al.* 2003b). A high concentration of cellular cyanotoxins in the waters of Lakes Sonachi, Simbi (Ballot *et al.* 2004a), Nakuru and Bogoria (Ballot *et al.* 2004b) is additional evidence in support of cyanotoxin contribution to flamingo die offs. Hence bird mortalities may have possibly resulted from toxins acting alone or in synergy with other stress factors such as heavy metals (Kairu, 1996; Nelson *et al.* 1998) and mycobacteria infection (Kock *et al.* 1999).

Blooms of the known toxin producing *Anabaenopsis abijatae*, *A. arnoldii* and *Anabaena* sp. present in Lake Elmenteita appeared to be atoxygenic (non toxic). This is possibly because the lake has atoxygenic strains or its environment does not support toxin production. However, the same species could have contributed to toxin production in Lakes Nakuru and Simbi. Toxin presence in Lakes Bogoria and Sonachi, both dominated by *Athrospira fusiformes* suggests that toxic strains of *A. fusiformes* are present in Lakes Bogoria, Sonachi and possibly Nakuru. This finding was supported by the presence of toxins in culture strains of *A. fusiformes* from the three lakes (Ballot *et al.* 2004a, b). Although *A. fusiformes* has long been considered to be atoxygenic, recent observations suggest a possible existence of toxigenic strains (Gilroy *et al.* 2000; Iwasa *et al.* 2002). The analysis of a *Spirulina* based commercial human dietary supplement by Gilroy *et al.* (2000) found a microcystin concentration of 0.15 to 2.12 $\mu\text{g g}^{-1}$ DW. However, it was not clear whether the source was indeed *A. fusiformis* or perhaps some other cyanobacterial cells present in the supplement. Another incident was the liver injury sustained by a Japanese male after eating *Spirulina* (Iwasa *et al.* 2002), which suggested a possible hepatotoxic effect.

Flamingo mortality linked to cyanobacteria toxin poisoning appears not to be confined to the saline lakes of the East African Rift Valley. Toxin related mortalities have been reported for the Greater Flamingos (*Phoenicopterus ruber*)

in Spain (Alonso-Andicoberry *et al.* 2002) and for the Chilean flamingoes (*P. chilensis*) in Florida USA (Chittick *et al.* 2002). The high susceptibility of the flamingos possibly results from their feeding habits, which mostly targets the surface floating cyanobacteria (Codd *et al.* 2003b).

Implication of cyanotoxin presence in freshwater bodies

The occurrence of cyanotoxins at concentrations above the acceptable upper limit (1.0 μg microcystin LR equivalent l^{-1}) in water bodies used for various domestic chores presents new challenges to the management of freshwater resources in Kenya, a country already designated water scarce, with an annual freshwater availability of less than 1 000 m^3 per person, (Ongwen, 1996). Firstly, the presence of cyanotoxins means that the existing water quality assessment and monitoring strategies that employ microbial and physico-chemical criteria will no longer be adequate in the assessment of water quality, especially in water bodies showing evidence of progressive eutrophication. Secondly, although there are no confirmed records of human health problems that have been linked to toxic cyanobacteria in Kenya, the toxin levels recorded in some of the waters serving as sources of domestic water for some rural communities means that incidences of human health problems cannot be ruled out. Poor medical services and records in rural communities that rely on natural water bodies means that the health problems associated with consumption of contaminated water can easily go unnoticed or wrongly attributed to other causes. Finally, the presence of cyanotoxins in some of Kenya's freshwaters will further diminish the amount of clean water available to its citizens in a country where a significant proportion of the population already lacks access to clean water.

Although the maximum microcystin concentration recorded in the present study (less than 3.25 $\mu\text{g l}^{-1}$ in Lake Baringo) is comparatively lower than that measured at Caruaru, Brazil (19.5 $\mu\text{g l}^{-1}$; Carmichael *et al.* 2001), which resulted in the death of 76 patients (Pouria *et al.* 1998; Rinehart *et al.* 2001), there is still reason to be concerned. This study did not find significant concentrations of dissolved cyanotoxins, however, a separate study carried out recently by Mwaura *et al.* (2004) recorded a dissolved microcystin concentration of up

to $2.85 \mu\text{g l}^{-1}$ based on ELISA tests in a highland reservoir of Kenya during the dry season. Hence occurrence of dissolved toxins cannot be totally ruled out.

Presence of cyanobacteria with the potential for toxin production in some of Kenya's freshwater bodies is another cause of concern for a number of reasons. Firstly, this is a potential problem since there is a possibility that an increase in nutrient input into these waters or water column stability changes can lead to bloom development by these cyanobacteria and its associated problems. Secondly, these waters could have been subjected to progressive eutrophication and the observations made are signs of impending bloom related problems unless the situation is promptly addressed. In the absence of a long term monitoring program for these water bodies, the correct position is not certain. However, the progressive increase in nutrient concentrations in Lake Naivasha since the early 1980s (Boar *et al.* 1999) indirectly supports progressive eutrophication in the larger water bodies. Hence the need to institute measures aimed at reducing nutrient loading into inland waters in general. Despite the enormous size of the exorheic Lake Victoria, it has taken only some fifty years of nutrient loading to reach the present stage of algal bloom formation (Verschuren *et al.* 1998; Verschuren *et al.* 2002). The smaller size of the investigated water bodies, some of which are endorheic suggests that much shorter times are needed to reach the stage of bloom formation. These coupled with increasing land degradation consequent to rising demographic pressure and reliance on subsistence agriculture will with time accelerate the rate of eutrophication.

CONCLUSIONS

- Cyanotoxin poisoning plays a major role in the episodes of flamingo die offs that have been recorded in the Rift Valley lakes. These die offs possibly result from toxins acting alone or in synergy with other stress factors that stem from human activities.
- The presence of cyanotoxins at concentrations above the acceptable upper limit ($1.0 \mu\text{g}$ microcystin LR equivalent l^{-1}) has been demonstrated for some water bodies used for various domestic chores. This means that the water stressed communities in Kenya; especially those in the

rural areas, as well as domestic and wild animals are presently exposed to cyanotoxin related problems.

- Presence of cyanobacteria known for toxin production in a number of water bodies further amplifies the already clear signs of a progressive deterioration of water quality in the country. There is also an imminent danger that such water bodies can develop blooms of these toxic algae leading to human and animal health problems.

RECOMMENDATIONS

In many parts of the world, especially in the developing nations, the gap between supply and demand for freshwater is ever widening hence increasing the stress on these resources. In Africa, rapid population growth, pollution from pesticides and fertilizers, and industrial effluent all reduce availability of clean and safe drinking water, hence contributing to water stress. Thus, the entry of cyanotoxins into the field will further aggravate the situation.

Reducing the risk of exposure

Urgent measures must be taken to prevent human and animal problems associated with the consumption of toxin contaminated waters and occurrence of HABs in Kenyan freshwater lakes. The long-term solution in places where cyanobacterial toxins are prevalent is the provision of clean drinking water throughout the year. A continuous monitoring program is necessary to advise on areas that require the supply of drinking water. An immediate step is to inform affected communities on the dangers inherent in the use of toxin-laden waters, create awareness on early signs of bloom formation and provide practical ways of reducing exposure risks. Because cellular based toxin loads are considerably higher than those in solution (consistently below the limit of detection during the study), efforts that reduce the quantity of cyanobacterial cells can reduce the risk of toxin exposure to a large extent.

These include:

- Advising communities living around toxin contaminated water bodies against directly drinking the untreated water, whether a surface bloom is visible or not.

Livestock and pets should not be allowed to drink the water directly.

- Encouraging the collection of water for domestic use from shallow boreholes near the shore where toxins are suspected.
- Advising people against swimming or wading in the water when there is a visible cyanobacterial bloom.
- Encouraging people to form a habit of applying simple pretreatment of water, for example flocculation of suspended solids, filtration with suitable material such as cheesecloth. As a rule drinking water must be boiled because of the added risks from protozoan, worm, bacterial and viral illnesses.
- Introducing low cost materials that can adsorb cyanotoxins such as activated carbon in places where algal blooms are prevalent and popularizing their use.
- Prohibiting all forms of cleaning operations inside an agreed radius from the standing water body.

In addition to these, there is need for a review of the drinking water quality regulations to incorporate cyanobacteria and cyanotoxins as parameters that must be monitored for water quality control.

Eutrophication control

In most nations, particularly in developing regions, management of freshwater resources has largely focused more on water availability (quantity) rather than water quality. The problems of water quality can often be as severe as those of availability. Similarly, a decline in water quality can lead to a decline in water availability. The first step in the prevention of eutrophication related water quality deterioration is to carry out an investigation on the various sources of nutrients. Where effluents from sewage treatment plants find their way into the water body, effluent water quality standards should strictly be enforced and the treatment facilities maintained regularly. In agricultural countries like Kenya, nutrient and pollution runoff from the land is a common source of eutrophication in the rural areas. Hence preventing these wastes from getting into the water is important in greatly reducing, the frequency, toxicity and duration of harmful algal blooms. Special attention should be given to phosphorus, as an important nutrient for

cyanobacterial dominance. In cases of already eutrophic waters, schemes for purification and reducing nutrient loads, where feasible, should be initiated. However, this should be preceded by a careful evaluation of the available approaches to prevent other problems from setting in.

Research and monitoring

Some dedicated research focusing on HABs should be initiated. It is important to confirm the identity and distribution of toxin producing algae in Kenyan waters. The role of eutrophication and other environmental factors involved in the suppression or promotion of algal bloom development as well as toxin production needs to be determined. For example, the impact of changes in nitrogen phosphorus (N:P) ratios and concentration on toxin production, specific mortality by pathogens and grazing on HAB species. In the pursuit of natural control options, research should also focus on identifying the natural enemies of HAB species such as bacteria and viruses and conditions under which they are most effective. The success of combating HABs is vested in a good understanding of their growth limiting factors in each water body. Research should also focus on coming up with low cost materials that can be used to remove toxins from contaminated waters.

Monitoring is important for the acquisition of the necessary data and information for making informed water resource management decisions. For each water body involved, ecological and water quality indicators of lake health must be carefully selected and a monitoring schedule established. Research efforts should focus on establishing useful indices of water quality change. For example, organisms sensitive to water quality deterioration that disappear when the quality declines, can be used as possible sentinels of deleterious changes.

The scientific studies of inland waters in Kenya suffer from a lack of coordination. This has resulted in a wide disparity in the amount of scientific attention paid to different lakes. Many NGOs, individuals, government agencies, academic institutions, research organizations and so on are involved in the pursuit of diverse research goals. Because on many occasions, these research initiatives operate independently, duplication is not uncommon. The data generated over time

may be characterized by an overlap with intervening periods of no data! Hence the need for an umbrella organization that can collate research data, disseminate findings and identify areas in need of further research.

Capacity building

Capacity building and institutional development are essential for successful implementation of the measures proposed above and should focus at water research institutions, local communities as well as those authorities responsible for the management of these waters. It is also important to strengthen the capabilities of community groups to use and manage water resources prudently.

A common problem in most developing countries is the availability of funds. To reduce the cost of monitoring programs and make them sustainable, capacity building should aim at developing the necessary skills at the community level. Low cost but effective data collection for monitoring purposes involving citizen volunteers, local authorities and schools, for instance, should be considered. The initial training of community members and formulation of monitoring guidelines can be carried out by collaborative efforts of government authorities, research institutions, universities and international partner institutions.

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REFERENCES

- Agence France-Press 2002 Algae killing fish off east African shoreline identified. *Agence France-Press*, February 15, 2002 Nairobi, Kenya.
- Alonso-Andicoberry, C., Garcia-Villada, L., Lopez-Rodas, V. & Costas, E. 2002 Catastrophic mortality of flamingos in a Spanish national park caused by cyanobacteria. *Vet. Rec.* **151**, 706–707.
- Ballot, A., Pflugmacher, S., Wiegand, C., Kotut, K. & Krienitz, L. 2003 Cyanobacterial toxins in Lake Baringo, Kenya. *Limnologica* **33**, 2–9.
- Ballot, A., Krienitz, L., Kotut, K., Wiegand, C., Metcalf, J. S., Codd, G. A. & Pflugmacher, S. 2004a Cyanobacteria and cyanobacterial toxins in three alkaline lakes of Kenya – Lakes Bogoria, Nakuru and Elmenteita. *J. Plankt. Res.* **26**, 925–935.
- Ballot, A., Pflugmacher, S., Wiegand, C., Kotut, K. & Krienitz, L. 2004b Cyanobacteria and cyanobacterial toxins in the alkaline crater lakes Sonachi and Simbi. *Harmful algae* **4**, 139–150.
- Boar, R. R., Harper, D. M. & Adams, C. S. 1999 Biomass allocation in *Cyperus papyrus* in a tropical wetland, Lake Naivasha, Kenya. *Biotropica* **31**, 411–421.
- Byth, S. 1980 Palm Island mystery disease. *M. J. Australia* **2**, 40–42.
- Carmichael, W. W. 1992 Cyanobacteria secondary metabolites – the cyanotoxins. *J. Appl. Bacteriol.* **72**, 445–459.
- Carmichael, W. W. 1997 The cyanotoxins. *Adv. Bo. Res.* **27**, 211–256.
- Carmichael, W. W., Azevedo, S. M. F. O., An, J. S., Molica, R. J. R., Jochimsen, E. M., Lau, S., Rinehart, K. L., Shaw, G. R. & Eaglesham, G. K. 2001 Human fatalities from cyanobacteria: chemical and biological evidence for cyanotoxins source. *Environ. Health Perspect.* **109**, 663–668.
- Chittick, E., Puschner, B., Walsh, M., Gearhart, S., St Leger, J., Skocelas, E. & Branch, S. 2002 Blue-green algae microcystin toxicosis in captive Chilean flamingos. In: *Proceedings of the American Association of Zoo Veterinarians*. Milwaukee, USA, pp. 115–116.
- Codd, G. A. & Beattie, K. A. 1991 Cyanobacteria (blue green alga) and their toxins: awareness and action in the United Kingdom. *PHLS Microbiology Digest Supplement* **8**, 82–86.
- Codd, G. A., Metcalf, J. S., Morrison, L. F., Krienitz, L., Ballot, A., Krause, E., Kotut, K., Putz, S., Wiegand, C. & Pflugmacher, S. 2003a Cyanobacterial toxins in the mass mortalities of flamingos at Kenyan lakes. *Phycologist Abstracts of the annual winter meeting*, Galway, 2–5 January 2003.
- Codd, G. A., Metcalf, J. S., Morrison, L. F., Krienitz, L., Ballot, A., Wiegand, C., Pflugmacher, S. & Kotut, K. 2003b Susceptibility of flamingos to cyanobacterial toxins via feeding. *Vet. Rec.* **152**, 722–723.
- Dow, C. S. & Swoboda, U. K. 2000 Cyanotoxins. In: *The Ecology of the Cyanobacteria* (eds. Whitton, B. A. & Potts, M.), Kluwer, Dordrecht, pp. 613–632.
- Gilroy, D. J., Kauffman, K. W., Hall, R. A., Huang, X. & Chu, F. S. 2000 Assessing potential health risks from microcystin toxins in blue green algae dietary supplements. *Environ. Health Persp.* **108**, 435–439.

- Gunn, G. J., Rafferty, A. G., Rafferty, G. C., Cockburn, N., Edwards, C., Beattie, K. A. & Codd, G. A. 1992 Fatal canine neurotoxicosis attributed to blue-green algae (cyanobacteria). *Vet. Rec.* **4**, 301–302.
- Harding, W. R. 1997 New regional emergence of cyanobacterial toxicosis. *Harmful Algae News* **16**, 12–13.
- Harding, W. R., Rowe, N., Wessels, J. C., Beattie, K. A. & Codd, G. A. 1995 Death of a dog attributed to the cyanobacterial (blue-green algal) hepatotoxin nodularin in South Africa. *J. S. Afr. Vet. Ass.* **66**, 256–259.
- Hepperle, D. 2000 *Opticount* (distributed by the author). Available at <http://domix0.tripod.com>
- Hitzfeld, C. B., Höger, S. J. & Dietrich, D. R. 2000 Cyanobacterial toxins: removal during drinking water treatment, and human risk assessment. *Environ. Health Persp.* **108**, 113–122.
- Iwasa, M., Yamamoto, M., Tanaka, Y., Kaito, M. & Adachi, Y. 2002 *Spirulina* associated hepatotoxicity. *Am. J. Gastroenterol.* **97**, 3212–3213.
- Kairu, J. K. 1996 Heavy metals residues in birds of Lake Nakuru, Kenya. *Afr. J. Ecol.* **34**, 397–400.
- Kock, N. D., Kock, R. A., Wambua, J., Kamau, G. J. & Mohan, K. 1999 *Mycobacterium avium*-related epizootic in free-ranging Lesser Flamingos in Kenya. *J. Wildlife Dis.* **35**, 297–300.
- Krienitz, L., Ballot, A., Wiegand, C., Kotut, K., Codd, G. A. & Pflugmacher, S. 2002 Cyanotoxin producing bloom of *Anabaena flos-aquae*, *Anabaena discoidea* and *Microcystis aeruginosa* (Cyanobacteria) in Nyanza Gulf of Lake Victoria, Kenya. *J. App. Bot.* **76**, 179–183.
- Krienitz, L., Ballot, A., Wiegand, C., Kotut, K., Codd, G. A. & Pflugmacher, S. 2003 Contribution of hot spring cyanobacteria to the mysterious deaths of lesser flamingos at Lake Bogoria, Kenya. *FEMS Microbiol. Ecol.* **43**, 141–148.
- Lawton, L. A. & Codd, G. A. 1991 Cyanobacterial (Blue-green-algal) toxins and their significance in UK and European waters. *J. Inst. Water Environ. Manage.* **5**, 460–465.
- Luckas, B. 2000 Phycotoxins in the marine environment: control of marine organisms for contamination with algal toxins. *Int. J. Environ. Pollut.* **13**, 148–172.
- Matsunaga, H., Harada, K. I., Sennma, M., Ito, Y., Yasuda, N., Ushida, S. & Kimura, Y. 1999 Possible cause of unnatural mass death of wild birds in a pond in Nishinomiya, Japan: sudden appearance of toxic cyanobacteria. *Nat. Toxins* **7**, 81–84.
- Mwaura, F., Koyo, A. O. & Zech, B. 2004 Cyanobacterial blooms and the presence of cyanotoxins in small high altitude tropical headwater reservoirs in Kenya. *J. Water Health* **2**, 49–57.
- Nasri, A. B., Bouaicha, N. & Fastner, J. 2004 First report of a microcystin-containing bloom of the cyanobacteria *Microcystis* spp. in Lake Oubeira, eastern Algeria. *Arch. Environ. Con. Tox.* **46**, 197–202.
- Nelson, Y. M., Thampy, R. J., Motelin, G. K., Raini, J. A., Disante, C. J. & Lion, L. W. 1998 Model for trace metal exposure in filter-feeding flamingos at alkaline Rift Valley lakes, Kenya. *Environm. Toxicol. Chem.* **20**, 2302–2309.
- Okoko, T. 2000 Lake Flamingos Threatened by Effluent. *Panfrican News Agency*, December 17, 2000, Nairobi, Kenya.
- Ong'wen, O. 1996 NGO experience, intervention, and challenges in water strain, demand, and supply management in Africa. In: *Water Management in Africa and the Middle East: Challenges and Opportunities* (eds. Rached, E., Rathgebers, E. & Brooks, D. B.). IDRC, Ottawa, Canada, pp. 295.
- Oudra, B., Loudiki, M., Vasconcelos, V., Sabour, B., Sbiyyaa, B., Oufdou, K. & Mezrioui, N. 2002 Detection and quantification of microcystins from cyanobacteria strains isolated from reservoirs and ponds in Morocco. *Env. Toxicol.* **17**, 32–39.
- Plumley, F. G. 1997 Marine algal toxins: Biochemistry, genetics, and molecular biology. *Limnol. Oceanogr.* **421**, 252–1264.
- Pollack, M.P. 1999 *Toxic Algae, Alligator & Bird Deaths*. <http://lama.kcc.hawaii.edu/praise/news/eh38.html> (viewed in April 2004).
- Pouria, S., Deandrade, A., Barbosa, J., Caavalcanti, R. L., Barreto, V. T. S., Ward, C. J., Prieser, W., Poon, G. K., Neild, G. H. & Codd, G. A. 1998 Fatal microcystin intoxication in haemodialysis unit in Caruaru, Brazil. *Lancet* **352**, 21–26.
- Rinehart, K. L., Shaw, G. R. & Eaglesham, G. K. 2001 Human fatalities from cyanobacteria: Chemical and biological evidence for cyanotoxins. *Environ. Health Persp.* **109**, 663–668.
- Scott, W. E. 1991 Occurrence and significance of toxic cyanobacteria in Southern Africa. *Water Sci. Technol.* **23**, 175–180.
- Stephen, C., Keft, M. L. & Dawe, S. C. 1993 Hepatic megalocytosis in wild and farmed chinook salmon *Oncorhynchus tshawtscha* in British Columbia, Canada. *Dis. Aquat. Organ.* **16**, 35–39.
- Tomlinson, C. 2002 Fish deaths confirmed to be caused by toxic algae, expected to clear naturally. *Associated Press*, February 08, 2002, Nairobi, Kenya.
- Van Dolah, F. M. 2000 Marine algal toxins: Origins, health effects, and their increased occurrence. *Environ. Health Persp.* **108**, 133–141.
- Van Ginkel, C.E. 2003 *A national survey of the incidence of cyanobacterial blooms and toxin production in major impoundments*. Internal Report No. N/0000/00/DEQ/0503. Resource Quality Services, Department of Water Affairs and Forestry, Pretoria, v + 44 pp + 4 pp appendix.
- Van Halderen, A., Harding, W. R., Wessels, J. C., Scheider, D. J., Heine, E. W. P., vanderMerwe, J. & Fourie, J. M. 1995 Cyanobacterial (Blue green algae poisoning of livestock in Western Cape Province of South Africa. *J. South Afr. Vet. Ass.* **66**, 260–264.
- Verschuren, D., Edgington, D. N., Kling, H. J. & Johnson, T. C. 1998 Silica depletion in Lake Victoria: Sedimentary signals at offshore stations. *J. Great Lakes Res.* **24**, 118–130.
- Verschuren, D., Johnson, T. C., Kling, H. J., Edgington, D. N., Leavitt, P. R., Brown, E. T., Talbot, M. R. & Hecky, R. E. 2002 History and timing of human impact on Lake Victoria, East Africa. *P. Roy. Soc. Lond. B Bio.* **269**, 289–294.
- Wanjiru, J. 2001 *Kenya's Pink Flamingos Weighted Down by Heavy Metals*. ENS, July 16, 2001, <http://www.worldlakes.org/News/Kenya%27s%20Pink%20Flamingos.html> (viewed in August 2002).
- Wicks, R. J. & Theil, P. G. 1990 Environmental factors affecting the production of peptides in floating scums of cyanobacterium

- Microcystis aeruginosa* in a hypertrophic African reservoir. *Environ. Sci. Technol.* **24**, 1413–1418.
- Wild Net Africa 2002 WWF concerned by massive fish die-off in Kenyan waters. Stellenbosch, February 05, 2002, <http://www.wildafrica.net/latest/messages/441.html> (viewed in August 2002).
- Yoo, R. S., Carmichael, W. W., Hoehn, R.C., Hruday, S. C. 1995 *Cyanobacterial (Blue green algae) Toxins: a Resource Guidebook*. American Water Works Research Foundation, Denver.
- Zilberg, B. 1966 Gastroenteritis in Salisbury European Children-A Five Year Study. *Centr. Afr. J. Med.* **12**, 164–168.