

The occurrence and response to toxic cyanobacteria in the Pacific Northwest, North America

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Abstract

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Toxic cyanobacteria are of increasing concern in the Pacific Northwest region of North America. Toxic blooms have been documented in Idaho, northern California, Oregon, Washington, and British Columbia and have caused animal poisonings, lake closures, and public health concerns. Microcystins are the most commonly detected cyanotoxins in this region and have been found at water concentrations from <1 µg/L to 30 mg/L. Anatoxins have also been measured in water bodies throughout the region and have been implicated in the deaths of both domestic and wild animals. Environmental factors associated with the occurrence of toxic blooms have been studied in several western Washington lakes. In these lakes, toxic blooms occurred during conditions of high phosphorus, water temperatures, and water column stability. Migration of cyanobacteria from the sediments contributed to toxic blooms in one of the lakes. Although regulations have not been formally adopted by the states and province in this region, some local jurisdictions use the World Health Organization proposed drinking water guideline of 1 µg/L microcystin-LR to close lakes to recreational use and for drinking water supplies. Recommendations currently advocated by Oregon take into account genera-specific differences, as well as separate, less stringent guidelines for recreational water bodies not used for drinking water.

Key words: anatoxin, cyanobacteria, cyanotoxins, drinking water, microcystins, recreational lakes, toxicity

Toxic cyanobacteria have been increasingly observed in the Pacific Northwest, which is defined herein as Idaho (ID), northern California (N. CA), Oregon (OR), and Washington (WA) in the USA, as well as the Canadian province of British Columbia (BC). These toxic blooms have caused animal poisonings and closures of drinking water and recreational lakes. Despite the widespread occurrence of toxic cyanobacteria in this region, few measurements of cyanotoxin concentrations and very few in-depth studies of the causes and temporal/spatial occurrence of cyanotoxicity have been conducted to date. Furthermore, efforts to manage toxic blooms and regulate human use of these water bodies have been isolated, without the benefit of uniform state or national guidelines.

In this paper, we summarize the available information on toxic cyanobacteria from this region and describe the approaches being used in several states to reduce human

exposure to cyanotoxins. Although intensive surveys of toxic cyanobacteria have been conducted in several lakes in WA, OR and CA (Jacoby *et al.* 1994, Jacoby *et al.* 2000, Johnston and Jacoby 2003, Kann 2005, Kann and Corum 2006), most measurements of cyanotoxins have been isolated events undertaken by lake managers or health officials in response to heavy bloom conditions or animal poisonings. None of the regions in the Pacific Northwest have attempted state- or province-wide monitoring and management of toxic cyanobacteria. However, OR has formulated guidelines with respect to issuing public health advisories based upon cell density of potentially toxigenic species (ODHS 2005, Stone and Bress 2007). The approach being advocated by OR is presented in this paper and may be a useful model for the other states and the province in the region.

Methods

Information on toxic cyanobacteria was compiled from the region by contacting lake managers, laboratories, state and federal agencies, university researchers and others involved in water quality, limnological, and toxicological studies. These people were asked to provide information on the toxic episodes with which they were involved by completing a table that listed the lake, its location, sampling date, major cyanobacterial species observed, cyanotoxin detected (and its concentration if measured), analysis method and laboratory, and the investigator. Although we attempted to compile a comprehensive dataset, there may be other information on cyanotoxicity in the region of which we are unaware.

A variety of methods were used to measure cyanotoxins including mouse assay, high performance liquid chromatography (HPLC), enzyme-linked immunoassay (ELISA), and protein phosphatase inhibition assay (PPIA). No attempt to evaluate the quality of the data was made. The data are reported as presented by the investigator and the laboratories that performed the analyses.

The methods used to analyze cyanotoxins in the in-depth studies conducted in WA were previously reported in Jacoby *et al.* (1994), Jacoby *et al.* (2000), and Johnston and Jacoby (2003). Determination of anatoxin-a for the OR lakes discussed below used Liquid Chromatography/Mass Spectrometry (LC/MS) techniques (*e.g.*, Harada *et al.* 1999), and microcystin analyses for both OR and N. CA samples used ELISA methodology (Chu *et al.* 1990, Carmichael and An 1999). Cell density of toxigenic species for OR and N. CA was determined microscopically on samples preserved in Lugol's Iodine (APHA *et al.* 1992).

Results and discussion

Cyanotoxin data compiled from the region are summarized for each state and the BC province in the following sections, followed by an overview of the efforts taken by some states to reduce public health risks from exposure to toxic cyanobacteria.

Most monitoring for cyanotoxins has been conducted in OR and WA (Table 1) with isolated monitoring in the other states and BC. Microcystins, which are hepatotoxins, were the most frequently analyzed and detected cyanotoxins. To a much lesser extent, anatoxins, which are neurotoxins, were also detected in several lakes in the region.

Cyanotoxicity occurrence in ID

Although toxic blooms have been implicated in animal poisonings, cyanotoxins have been measured in only two lakes in Idaho. The first documented toxic blooms occurred

in Black Lake in fall 1985 and 1986. Dogs, cattle, and deer died after drinking water with a heavy *Anabaena flos-aquae* bloom. Anatoxin-a was indicated by mouse bioassay (Kann and Falter 1987). In 1993, 23 cattle died after consuming water from Cascade Reservoir. The Idaho Division of Environmental Quality continued to measure microcystins during summer-fall 1994-1996. Concentrations of total microcystins ranged from 0.3 ng/mg to 100 µg/mg during this period (Table 1). No other monitoring for cyanotoxins has been conducted in Idaho.

Cyanotoxicity occurrence in N. CA

Although several dog deaths were reported from two Humboldt County waterways (Table 1), the extent of toxic cyanobacterial monitoring in N. CA has been limited. The exception is one year of intensive monitoring on the Klamath River, including Copco and Iron Gate Reservoirs (two reservoirs on PacifiCorp's Klamath River Hydropower Project). These systems experienced dense toxic blooms of the cyanobacteria *Microcystis aeruginosa* in 2004 and 2005. A summary of cell density data during 2005 shows a preponderance of reservoir stations exceeding the World Health Organization (WHO) Moderate Probability of Adverse Health Effect Level (MPAHEL) of 100,000 cells/ml (Chorus and Bartrum 1999) for the majority of the August-October period (Fig. 1a). During the peak cell density period of mid-August to mid-September, several stations exceeded this level by more than 100×, with the maximum cell count at 163 million cells/ml (>1000× the MPAHEL) on 20 September, 2005 (Fig. 1a). No *M. aeruginosa* was detected in the Klamath River inflow to Copco on any of the measured sample dates (station KRAC; Fig. 1a, black circles). However, levels in the river below the reservoirs increased coincident with reservoir increases, and a maximum density of 42,577 cells/ml of *M. aeruginosa* was detected in the Klamath River below Iron Gate on 8 September (station KRBI; Fig. 1a).

Microcystin toxin levels followed the general seasonal trend of cell counts, peaking during the August-September period and declining in October (Fig. 1b). During the August-September period toxin levels exceeded the WHO MPAHEL of 20 µg/L microcystin at the majority of stations, and levels were frequently greater than 10× the MPAHEL, peaking at 1,994 µg/L at CRCC on 20 September, 2005. Likewise, the Tolerable Daily Intake (TDI; 0.04 µg microcystin/kg/day as defined in WHO 1998) for an 18 kg child ingesting 100 ml was commonly exceeded by more than 10-100× throughout the August-September period (Fig. 1c). Despite the occurrence of *M. aeruginosa* 50-75 miles upstream in the Upper Klamath Lake system (Agency Lake; Table 1), the non-detects at KRAC (directly above Copco Reservoir), even when reservoir stations showed substantial concentrations of both toxin and *M. aeruginosa* cell density, clearly indicate the role of the reservoirs in providing ideal habitat conditions for *M.*

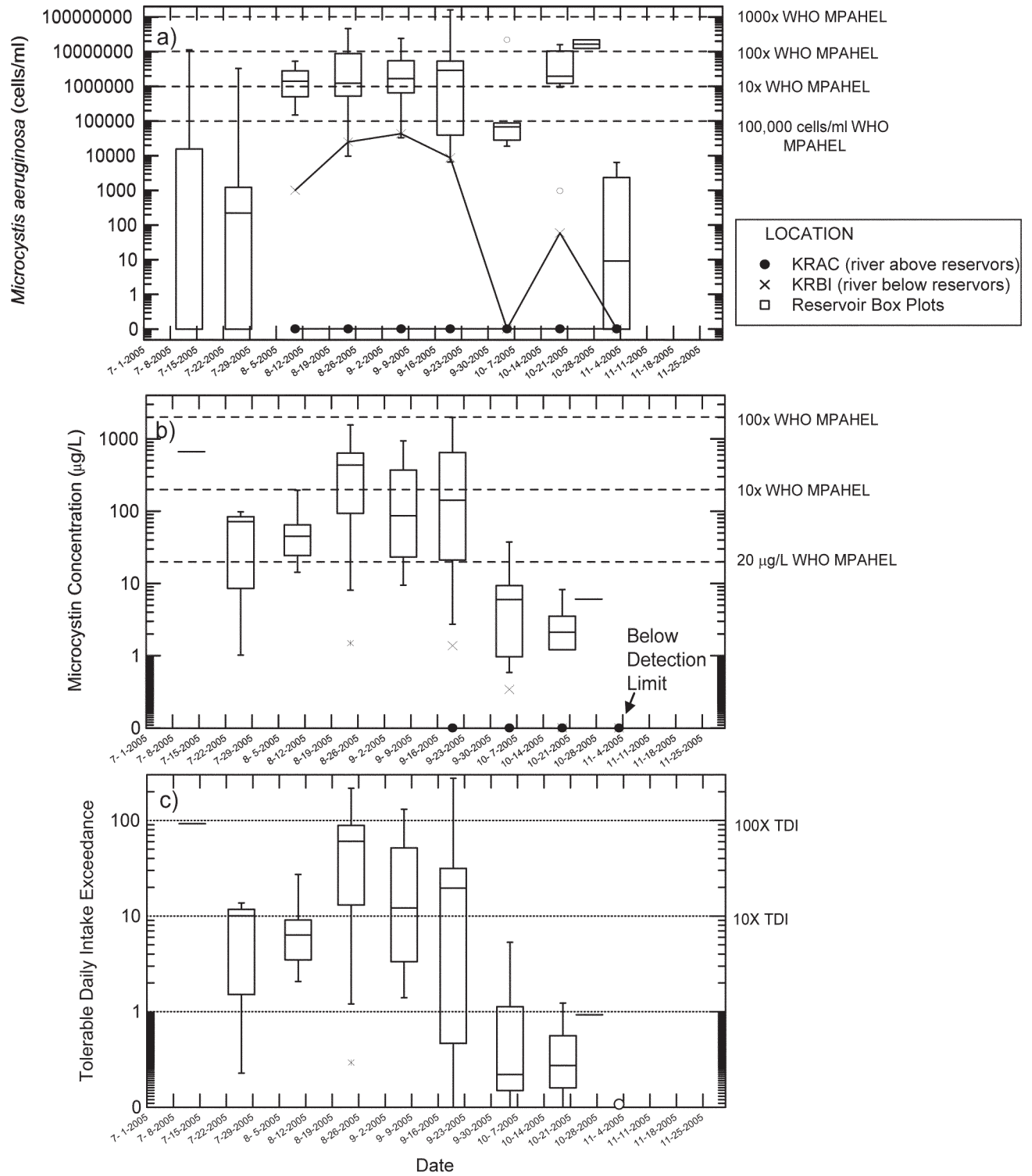


Figure 1.-*M. aeruginosa* cell density (a), microcystin concentration (b), and tolerable daily intake (c) in Copco and Iron Gate Reservoirs and Klamath River stations above Copco Reservoir (KRAC) and below Iron Gate Reservoir (KRBI), 2005. The WHO MPAHEL is 100,000 cells/ml for *M. aeruginosa* and 20 µg/L for microcystin (Falconer *et al.* 1999).

Table 1.-Toxic cyanobacteria detected in the Pacific Northwest, USA.

County	Lake	Date	Major Species	Toxin/Concentration	Analysis Method	Collected by	Animal Deaths	Beach Closure/ Advisory
ID								
Kootenai	Black	Fall 1985 & 1986	<i>Anabaena flos-aquae</i>	anatoxin-a	Mouse assay	Kann & Falter (1987) (dogs, cattle, deer deaths)	X	X
Valley	Cascade Reservoir	7/14-8/18/94 9/12/94 10/5/94 8/17/95 9/12 & 9/13/95 10/3 & 10/4/95 10/8/96	<i>Anabaena spiroides</i> <i>Microcystis aeruginosa</i>	Total MC: 0.3-100 ng/mg 5-600 ng/mg 85-250 ng/mg 0.7-2.4 ng/mg 1.6-100.0 µg/mg 18.3-94.0 µg/mg 33.1-50.6 µg/mg	ELISA ¹	ID Division of Environmental Quality (23 cattle deaths in summer 1993)	X	X
Northern CA								
Humboldt	Big Lagoon	2001	Not Determined	Dog deaths		Humboldt Co. Dept of Public Health	X	X
Humboldt	Eel River	2002-2004	<i>Anabaena flos-aquae?</i>	Dog deaths		Humboldt Co. Dept of Public Health	X	X
Siskiyou	Copco Reservoir	2004-2006	<i>Microcystis aeruginosa</i>	max density>393,000,000 cells/ml; max MC 12,176 µg/L	ELISA ¹	Karuk Tribe, J. Kann (Aquatic Ecosystem Sciences)	X	X
Siskiyou	Iron Gate Reservoir	2005-2006	<i>Microcystis aeruginosa</i>	max density>17,458,000 cells/ml; max MC 2032 µg/L	ELISA ¹	Karuk Tribe, J. Kann (Aquatic Ecosystem Sciences)	X	X
Siskiyou, Humboldt	Klamath River (below Iron Gate to Estuary)	2005	<i>Microcystis aeruginosa</i>	9020-1,355,627 cells/ml; max MC 46.73 µg/L	ELISA ¹	Karuk Tribe, Yurok Tribe, U.S. Fish & Wildlife Service		
OR								
Clackamas	Lake Oswego	1998, 2001	<i>Microcystis aeruginosa</i>	Visible scum			X	X
Coos	Eel Lake	2002	<i>Microcystis aeruginosa</i>			Tennile Lakes Basin Partnership	X	X

Table 1.-Continued.

County	Lake	Date	Major Species	Toxin/Concentration	Analysis Method	Collected by	Animal Deaths	Beach Closure/Advisory
Coos	Tennile Lakes (used for potable water after treatment by individual homeowners)	1997, 2001, 2002, 2004	<i>Microcystis aeruginosa</i>	>11,000 cells/ml 2001; >40,000 cells/ml 2002; >2 µg/L MC1997 and 2000; 0.7-1.61 µg/L 2001-2002	Mouse assay ² 1997; ELISA ³	Tennile Lakes Basin Partnership, J. Kann (Aquatic Ecosystem Sciences)		X
Deschutes	Crane Prairie	2003, 2004, 2005	<i>Anabaena flos-aquae</i> (possibly <i>A. lemmermannii</i>)	>915,000 cells/ml 2004; >100,000 cells/ml 2005; 0.19-4.92 µg/L MC 2004	ELISA ¹	Deschutes National Forest		X
Deschutes	Lava Lake	2004	<i>Anabaena flos-aquae</i> (<i>lemmermannii</i> ?)	>81,000 cells/ml 2004; 0-0.68 µg/L MC 2004	ELISA ¹	Deschutes National Forest		X
Deschutes	Paulina	2003, 2004	<i>Microcystis aeruginosa</i> ; <i>Anabaena</i> sp.	>1,900,000 cells/ml <i>Anabaena</i> sp. 2004; 0.009-84.0 µg/L MC 2004	ELISA ¹	Deschutes National Forest		X
Deschutes	Wickiup Reservoir	2004	<i>Anabaena flos-aquae</i>	> 50,000 cells/ml 2004; 0-2.9 µg/L MC 2004	ELISA ¹			X
Douglas	Diamond Lake	2001, 2002, 2003, 2004	<i>Anabaena flos-aquae</i> ; <i>A. circinalis</i>	max. anatoxin-a 262.3 µg/L 2001, max. MC 2.54 µg/L 2003; max. <i>Anabaena</i> sp. 558,000 cells/ml 2001	ELISA ¹	Umpqua National Forest, J. Eilers (Max Depth Aquatics), J. Kann (Aquatic Ecosystem Sciences)		X
Jackson	Fish Lake	2002	<i>Anabaena</i> sp.					
Jefferson	Suttle Lake	2004	<i>Anabaena flos-aquae</i>	>102,000 cells/ml 2004; 0.36-0.51 µg/L MC 2004	ELISA ¹	Deschutes National Forest		X
Josephine	Lake Selmac (used for potable campground water after treatment by Jackson Co. Parks)	2004	<i>Microcystis aeruginosa</i> ; <i>A. spiroides</i> , <i>A. circinalis</i>	>17,000,000 cells/ml; 0.19-13.5 µg/L MC raw lake water; 0.01-2.38 µg/L treated drinking water	ELISA ¹	Josephine County Parks (S. Schalk), J. Kann (Aquatic Ecosystem Sciences)		X

Table 1.-Continued.

County	Lake	Date	Major Species	Toxin/Concentration	Analysis Method	Collected by	Animal Deaths	Beach Closure/Advisory
Klamath	Odeil Lake	2004, 2005	<i>Anabaena flos-aquae</i> (<i>A. lemmermannii</i> ?)	>598,000 cells/ml 2004; >100,000 cells/ml 2005; 0.03-5.01 µg/L MC 2004; 10 µg/L MC-LR dissolved 2005	ELISA ¹ ; HPLC ⁴ 2005	J. Eilers (Max Depth Aquatics)		X
Klamath	Upper Klamath Lake (Agency Lake)	July 1996	<i>Microcystis aeruginosa</i>	30 mg/L in shoreline scum; 600 µg/L offshore MC-LR	ELISA ¹	J. Kann (Aquatic EcosystemSciences)		X
Lane	Cougar Reservoir	2002				Willamette National Forest		X
Lane	Dexter Reservoir	2002	<i>Anabaena</i> sp.			City of Lowell		X
Lane	Hills Creek Reservoir	2002, 2005	<i>Microcystis aeruginosa</i>	Visible scum		Willamette National Forest		X
Lane	Lookout Point Reservoir	2005	Not determined	Visible scum		Willamette National Forest		X
Lane	Mercer	2002, 2003		Visible scum				
Lincoln	Big Creek Reservoir	8/25/95	<i>Microcystis aeruginosa</i>	MC-LR	HPLC ²	D. Wagner (Devils Lake Improvement District)		
Lincoln	Devils Lake	1995		Visible blue-green algae bloom: No cell counts or toxin assays reported.				
Malheur	Owyhee Reservoir	2002	<i>Microcystis aeruginosa</i>	14.6 µg/L MC; 11.8 µg/L anatoxin-a	ELISA ¹	Oregon Dept. of Fish and Wildlife; W. Van Dyke; ~20 big horn sheep deaths reported	X	X
Multnomah	Blue Lake	2003		Visible scum				
Newport	Big Creek Reservoir	8/25/95	<i>Microcystis aeruginosa</i>	MC-LR	HPLC ²	D. Wagner (Devils Lake Improvement District)		
Wasco + Wheeler	John Day River	2001-2003		Visible scum		Bureau of Land Management; Dog deaths reported	X	

Table 1.-Continued.

County	Lake	Date	Major Species	Toxin/Concentration	Analysis Method	Collected by	Animal Deaths	Beach Closure/Advisory
WA								
Clark	Vancouver	8/20/03 8/28/03 9/4, 9/8/03	<i>Microcystis aeruginosa</i> <i>Microcystis aeruginosa</i> few <i>Aphanizomenon flos-aquae</i>	18.5, 0.91, >2.5 µg/L MC 7.0, 9.5 µg/L MC <0.5 µg/L MC	ELISA ⁵	M. McGuinn (Clark County)		X
Jefferson	Anderson	6/05/06	<i>Anabaena</i> (76%) <i>Aphanizomenon</i> (15%) <i>Microcystis</i> (9%) (1.5 × 10 ⁶ cells/mL)	20 µg/L anatoxin-a	HPLC ⁹	N. Harrington (Jefferson County Natural Resources) (2 dog deaths and 1 dog poisoning 5/27-6/2/06)	X	X
King	Garrett (Hicks)	7/29/04 8/25/04 8/10/04 8/30/04 9/7/04 9/21/04	NA	µg/L MC-LR equivalents: 0.25 0.85 0.26 0.75 0.74 0.68 0.86	ELISA ⁸ ELISA ⁸ ELISA ⁸ ELISA ⁸ ELISA ⁸ PPIA ⁸ ELISA ⁸	B. Budka [King County Water and Land Resources Division (WLRD)]		
King	Green	8/99-10/99 8/02 -1/03 8/03 -9/03	<i>Microcystis aeruginosa</i> <i>Microcystis aeruginosa</i> <i>Microcystis aeruginosa</i>	1-32 µg/L MC 3-100 µg/L MC 0.17-2.2 µg/L MC (n=10)	ELISA ⁶ ELISA ⁵ ELISA ⁵	J. Frodge (King County WLRD); Jacoby (2003) M. Joubert (Seattle Public Utilities); Jacoby (2003) E. Johnson (Seattle Public Utilities)		X X
King	Sammamish	9/97 8/99-9/99 Mar-Nov: 2003 2004 2005 2006	<i>Microcystis aeruginosa</i> <i>Microcystis aeruginosa</i> <i>Anabaena</i> , <i>Anacystis</i> , <i>Gomphosphaera</i> , <i>Aphanizomenon</i> Same as above <i>Anabaena</i> , <i>Aphanizomenon</i> NA	478-588 µg/g total MC 1-43 µg/L MC Maxi. µg/L MC-LR equivalents 0.13 (n=30) 0.17 (n=36) 0.16 (n=84) <MDL of 0.10 (n=89)	HPLC ⁷ ELISA ⁶ PPIA ⁸	J. Frodge (King County WLRD) Johnston & Jacoby (2003) ³ King County WLRD	X? X	X

Table 1.-Continued.

County	Lake	Date	Major Species	Toxin/Concentration	Analysis Method	Collected by	Animal Deaths	Beach Closure/ Advisory
King	Union	Mar-Nov: 2003	<i>Anabaena</i> , <i>Anacystis</i> , <i>Oscillatoria</i>	Maxi. µg/L MC-LR equivalents 0.14 (n=15)	PPIA ⁸	King County WLRD		
		2004	<i>Anabaena</i>	0.15 (n=18)				
		2005	NA	0.12 (n=17)				
		2006	<i>Anabaena</i>	0.14 (n=17)				
King	Washington	Mar-Nov: 2003	<i>Anacystis</i> , <i>Coelosphaerium</i> , <i>Gomphosphaeria</i>	Maxi. µg/L MC-LR equivalents 0.18 (n=45)	PPIA ⁸	King County WLRD		
		2004	<i>Aphanizomenon</i> , <i>Anacystis</i>	0.62 (n=54)				
		2005	<i>Anabaena</i> , <i>Anacystis</i> , <i>Aphanizomenon</i> , <i>Oscillatoria</i> , <i>Coelosphaerium</i>	0.68 (n=223)				
		2006	<i>Anabaena</i>	1.15 (n=230)				
		8/06	<i>Anabaena</i>	5.09, 52.6 (maxi. beach bloom samples)	PPIA ⁸			
Kitsap	Kitsap	10/22/01	<i>Anabena</i> sp. – 88% <i>Microcystis</i> sp. – 12%	anatoxin-a = 10 µg/L MC <1.0 µg/L	HPLC ⁹	S. Ultican (Kitsap County Health Department)		X
		9/30/06	<i>Gleotrichia</i> sp. – 95% <i>Aphanizomenon</i> sp. – 3% <i>Anabena</i> sp. – 1% <i>Microcystis</i> sp.	MC = 4.0 µg/L	HPLC ⁹			X
Kitsap	Long	9/20/02	<i>Anabena</i> sp. – 11% <i>Microcystis</i> sp. – 88% <i>Aphanizomenon</i> sp. – 1%	anatoxin-a < 2.0 µg/L MC < 0.5 µg/L	HPLC ⁹ & Mouse assay ²	S. Ultican (Kitsap County Health Department)		X

Table 1.-Continued.

County	Lake	Date	Major Species	Toxin/Concentration	Analysis Method	Collected by	Animal Deaths	Beach Closure/ Advisory	
Pierce	American	12/1/89,	<i>Anabaena flos-aquae</i>	anatoxin-a	Mouse assay ² and HPLC ¹	R. Hanowell [Tacoma-Pierce County Health Department, (TPCHD)], KCM 1992-1993; Jacoby <i>et al.</i> (1994)	X [11 animal poisonings (5 cat fatalities)]	X	
		12/14/89,							
		2/14/89,							
		2/4/92,							
		2/20/92,							
		12/3/92,							
		1/7/93,							
		2/4/93,							
		12/15/93							
		2/14/96							
Pierce	Clear	2/90	<i>Anabaena flos-aquae</i>	anatoxin	Mouse assay ²	R. Hanowell (TPCHD)	X	X	
		3/24/97,	<i>Aphanizomenon flos-aquae</i>	anatoxin	Mouse assay ²	R. Hanowell (TPCHD)	X	X	
		2/9/98,	<i>Anabaena flos-aquae</i>						
		2/24/98,	<i>Anabaena circinalis?</i>						
		1/06/99	<i>Coelosphaerium</i> sp.						
			<i>Anabaena</i> noted on all dates						
Pierce	Spanaway	8/18/95	<i>Microcystis aeruginosa</i>	MC	Mouse assay ²	R. Hanowell (TPCHD)	X	X	
		8/23/95	<i>Coelosphaerium</i> sp. (minor)			(several dog deaths reported during <i>Anabaena</i> bloom in spring 2005)			
		10/4/95	<i>Anabaena flos-aquae</i> (trace)						
Pierce	Steilacoom	10/07/92	<i>Anabaena</i>	MC	Mouse assay ²	R. Hanowell (TPCHD)	X	X	
			<i>Microcystis aeruginosa</i>						
		Aug.-Oct. 94	<i>Microcystis aeruginosa</i>	209-1385 µg/g MC	HPLC ⁷	Jacoby <i>et al.</i> (2000)	X	X	
		10/16/95	<i>Aphanizomenon flos-aquae</i>	MC	Mouse assay ² and HPLC ¹	M. Crayton [Pacific Lutheran University (PLU)]	X	X	
			<i>Microcystis aeruginosa</i>						
			<i>Coelosphaerium</i> sp.						
	<i>Anabaena</i>								
		10/6/98	<i>Microcystis aeruginosa</i>	MC	Mouse assay ²	R. Hanowell (TPCHD)	X	X	
		8/99	<i>Microcystis aeruginosa</i>	13 µg/L MC	ELISA ⁶	R. Hanowell (TPCHD) (dog death reported in fall 2004)	X	X	

Table 1.-Continued.

County	Lake	Date	Major Species	Toxin/Concentration	Analysis Method	Collected by	Animal Deaths	Beach Closure/ Advisory
Pierce	Wapato	8/1/97	<i>Microcystis aeruginosa</i>	MC	Mouse assay ²	R. Hanowell (TPCHD)	X	X
Pierce	Waughop	7/26/96	<i>Microcystis aeruginosa</i> <i>Coelosphaerium</i> sp.	MC	Mouse assay; HPLC ² ELISA ⁶	M. Crayton (PLU)		X
San Juan	Moran State Park Lake	12/30/04	<i>Aphanizomenon flos-aquae</i> (dominant) <i>Anabaena</i>	0.8 µg/L MC 0.13 µg/L MC-LR equivalents	ELISA ⁸	R. Hanowell (TPCHD) B. Budka [King County (WLRD)]		
Snohomish	Cassidy	Sept 2005	<i>Microcystis aeruginosa</i> <i>Aphanizomenon flos-aquae</i>	0.5-3.0 µg/L MC	ELISA ¹⁰	G. Williams/H. Reynolds [Snohomish County Surface Water Management (SWM)]		
Snohomish	Ketchum	July 2000	<i>Anabena flos-aquae</i> <i>Aphanizomenon flos-aquae</i> <i>Microcystis</i> sp.	25 µg/L anatoxin-a	HPLC ⁹	D. Dorling (Allied Aquatics)		
		Aug 2005	<i>Microcystis aeruginosa</i> <i>Aphanizomenon flos-aquae</i> *	0.5-3.0 µg/L MC	ELISA ¹⁰	G. Williams/H. Reynolds (Snohomish County SWM)		
		10/04/06	<i>Anabaena</i> <i>Microcystis</i>	4 µg/L saxitoxin	HPLC ⁹	G. Williams/H. Reynolds (Snohomish County SWM)	X	
Snohomish	Loma	6/05	<i>Microcystis aeruginosa</i>	>3.0 µg/L MC	ELISA ¹⁰	G. Williams/H. Reynolds (Snohomish County SWM)		X
Thurston	Lawrence	Nov 2004	<i>Microcystis</i> sp. <i>Aphanizomenon</i> sp.	2.73 - >830 µg/L MC-LR equiv.	ELISA ⁸	S. Davis (Thurston County Public Health Department) Possible net-pen fish kill	X	X?
Thurston	McIntosh	7/25/06	NA	0.99, 0.54, 0.51 µg/L MC-LR 1.52, 0.54, 0.48 MC-LR equiv.	ELISA ⁸ PPIA ⁸	B. Budka [King County (WLRD)]		
Yakima	Wenas	9/31/95	<i>Microcystis</i> ?	MC	HPLC ²	P. Freeborn (WA State Department of Ecology)		

Table 1.-Continued.

Province	Lake	Date	Major Species	Toxin/Concentration	Analysis Method	Collected by	Animal Deaths	Beach Closure/ Advisory
BC	Bouchie	8/30/95		364 µg total MC/g (1.17 µg cellular MC/L)	HPLC ⁷	R. Nordin (University of Victoria-Biology)		
	Douglas	8/27/95		25 µg total MC/g (0.16 µg cellular MC/L)				
	Eaglet	8/29/95		1363 µg MC-LR/g (119.29 µg cell. MC-LR/L)				
	Leighton	8/28/95		2 µg total MC/g (0.27 µg cellular MC/L)				
	Nulki	8/30/95		129 µg MC-LR/g				
	Putataekut	8/30/95		315 µg total MC/g (1.29 µg cellular MC/L)				
	Spokin	8/29/05		463 µg total MC/g (76.6 µg cellular MC/L)				
	Tabor	8/29/05		35 µg MC-LR/g (6.28 µg cellular micro-LR/L)				
	Tachick	8/29/05		220 µg MC-LR/g (0.43 µg cellular micro-LR/L)				
	Tunkwa	8/28/05		2 µg total MC/g (0.02 µg cellular MC/L)				

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⁴ Oregon Department of Environmental Quality, Portland, OR

⁵ Seattle Public Utilities Water Quality Lab, Seattle, WA (E. Johnson, M. Joubert)

⁶ Seattle University, Department of Civil & Environmental Engineering, Seattle, WA (J. Jacoby)

⁷ Algal Tox International, Edmonton, Alberta (B. Kotak)

⁸ King County Environmental Lab, Seattle, WA (D. Bouchard, G. Hannach)

⁹ Water Management Lab, Tacoma, WA

¹⁰ Envirologix QuickTube MC Kit (Snohomish County)

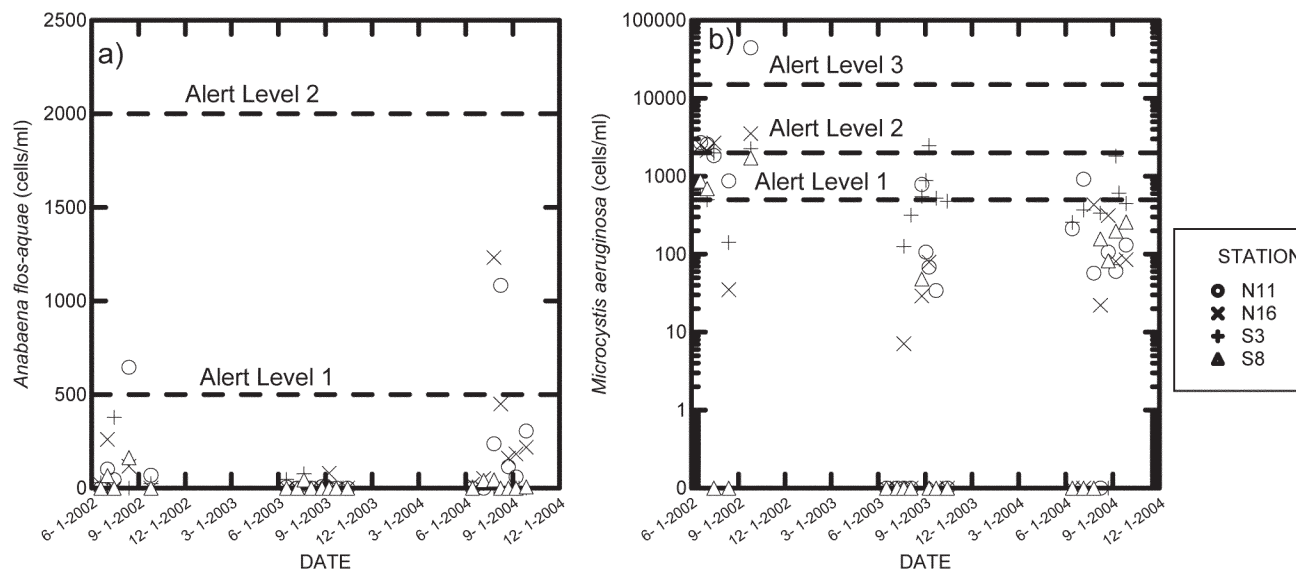


Figure 2.—*A. flos-aquae* (a) and *M. aeruginosa* (b) cell density levels at 4 stations in Tenmile Lakes Oregon, 2002–2004. Alert levels refer to drinking water levels outlined in Yoo (1995) and are Alert Level 1 = 500 cells/ml; Alert Level 2 = 2000 cells/ml, and Alert Level 3 = 15,000 cells/ml.

aeruginosa and serving as continued inoculum downstream in the Klamath River. Given existing guidelines, Copco and Iron Gate Reservoir conditions in 2005 represented a public health risk with respect to water contact recreation. Similar large concentrations of *M. aeruginosa* and microcystin were observed in 2006, with a maximum cell density of >393 million cells/ml and a maximum microcystin concentration of 12,176 µg/L (Table 1).

Cyanotoxicity occurrence in OR

The only reported animal deaths in Oregon were ~20 big horn sheep near Owyhee Reservoir in 2002 and several dog deaths on the John Day River between 2001 and 2003. Although the deaths were not definitively linked to toxic cyanobacteria, both microcystin and anatoxin-a were measured in water samples collected from Owyhee Reservoir several days after the dead sheep were found (Table 1).

Cyanotoxins and/or the presence of potentially toxigenic cyanobacterial species occurring at levels that prompted issuance of health advisories were documented in at least 26 lakes in OR (Table 1). Cyanotoxins were detected in 13 lakes, and toxic algae alerts were issued in the other 13 lakes on the basis of cell counts or the presence of a scum. The most commonly occurring toxin was microcystin, and in only one of the lakes, Diamond Lake, were health advisories issued for anatoxin-a. The highest measured microcystin value for the state was 30 mg/L, which occurred in a shoreline scum of *M. aeruginosa* in Agency Lake. Multiple advisories that occurred on recreational lakes in the Deschutes and Wil-

lamette National Forests were based upon a combination of cell density measurements, toxin measurements, and visual determination of shoreline scums. Two of the OR lakes, Tenmile Lakes and Lake Selmac, are used for potable water, and thus health advisories were issued both in response to recreational and potable water uses.

Tenmile Lakes

A toxic bloom of *M. aeruginosa* was first documented in 1997, at which time Oregon Human Services issued the first health advisory for toxic cyanobacteria. Because ~200 homes around the lakes use lake water directly for household purposes, public health advisories included recommendations for water treatment systems. Intermittent monitoring occurred between 1998 and 2001, and advisories were issued in 2000 and 2001. Since 2002, the local watershed council (Tenmile Lakes Basin Partnership) implemented a regular monitoring program at 4 sample stations specifically to assess cell density (and toxin concentration when cell density exceeds 2000 cells/ml) with respect to guidance levels for drinking water (Yoo *et al.* 1995, Falconer *et al.* 1999). Although *A. flos-aquae* was detected in Tenmile Lakes, cell density remained low (Fig. 2a). However, based on *M. aeruginosa* cell density that exceeded the Alert Level 2 guideline (Yoo *et al.* 1995) of 2000 cells/ml (Fig. 2b), additional advisories were issued in 2002 and 2003. Corresponding with the elevated cell density, on several occasions microcystin exceeded the WHO drinking water guidance level of 1 µg/L (Table 1; WHO 1998).

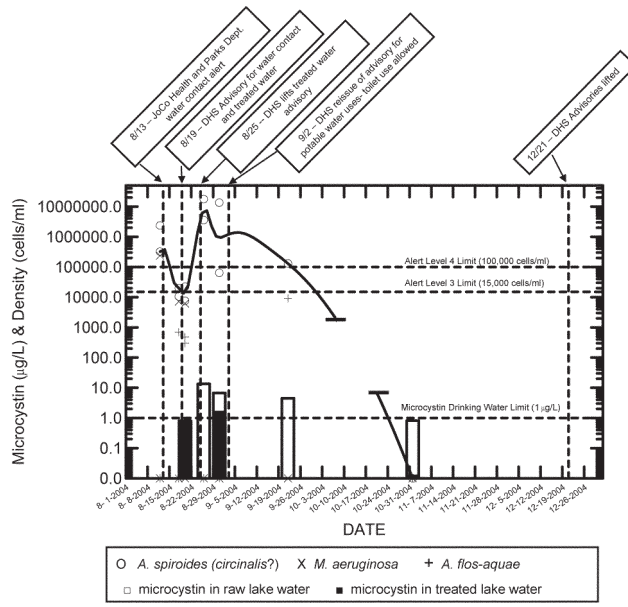


Figure 3.-Microcystin toxin concentration in raw and treated lake water (bars) and cell density for *Anabaena* sp. and *M. aeruginosa* (symbols; trend line is for the dominant *A. Spiroides*), Lake Selmac, 2004. Timeline shows Jackson County Health and Parks Departments (JoCo) and Oregon Department of Human Services (DHS) management advisories. Alert levels are after Yoo *et al.* (1995), with Alert Level 4 = 100,000 cells/ml.

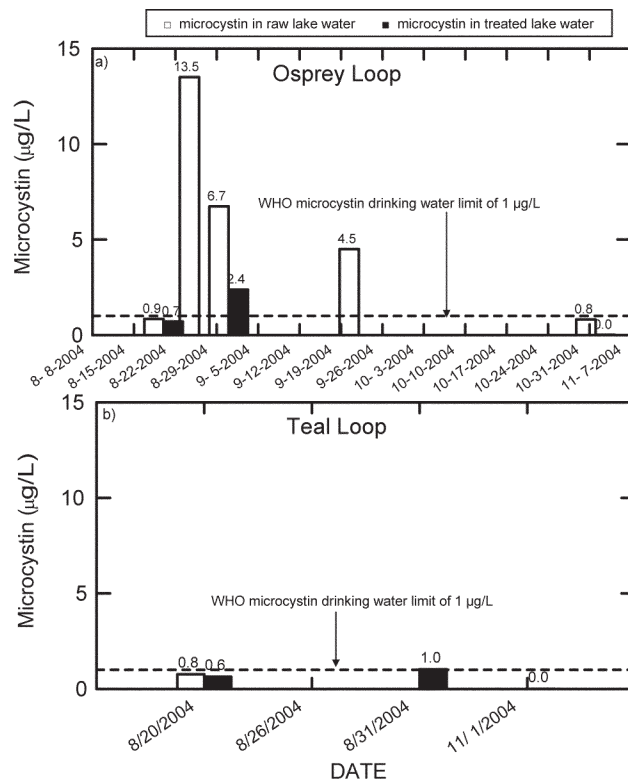


Figure 4.-Microcystin toxin concentration in raw and treated lake water at Osprey (a) and Teal Loop (b) campgrounds, Lake Selmac, 2004.

Lake Selmac

The first documented toxic cyanobacterial bloom in Lake Selmac occurred in 2004, and the initial early-August bloom was composed primarily of various *Anabaena* sp. and *M. aeruginosa* (Fig. 3). Sampling stations were located near the intake locations for treated drinking water systems supplying Osprey and Teal Loop campgrounds, and the configuration of the treatment systems consisted of preliminary clarification, filtration, carbon filtration, and finally chlorination before being routed to storage tanks that gravity feed the campground facilities.

M. aeruginosa declined from initial counts >200,000 cells/ml to <10,000 cells/ml in mid-August, and *Anabaena* cell density initially declined but then increased and remained high (>13,000,000 cells/ml) through early September (Fig. 3). During this period anatoxin-a was not detected; however, a maximum value of 13.5 µg/L of microcystin was measured in raw lake water on 26 August 2004. Samples of both raw and finished drinking water were lower than the WHO recommended level for drinking water on 18 August (Fig. 4). However, on 31 August the Osprey system removed only 65% of the microcystin (from 6.74 µg/L in the raw water to 2.38 µg/L in the finished water), and the remaining concentration was still more than 2× the WHO recommended level of 1 µg/L (Fig. 4a). This occurred despite probable reduction of algal cells by an infiltration system located at the Osprey intake.

The microcystin concentration of 6.74 µg/L in the lake represented a low risk in terms of contact recreation and indicated that high-density blooms of *Anabaena* sp. (3.5 million cells/ml on 31 August at Osprey Loop) in Lake Selmac in 2004 did not appear to be associated with microcystin or anatoxin levels that posed a substantial risk for contact recreation. These results are consistent with low levels of microcystin associated with some *Anabaena* species. However, even though raw water microcystin was not extreme, water treatment removal efficiency ranging between 16 and 65% was not sufficient to reduce levels in treated water to below the WHO guideline. These data indicate that under conditions of substantially higher raw water microcystin concentration, the potential exists to greatly exceed the WHO guideline in the treated water supplied to the campgrounds. A minimum one-week lag time for receiving toxin results necessitated curtailing potable water uses based on cell count data; subsequent laboratory results confirmed the efficacy of these decisions.

Diamond Lake

The first documented toxic cyanobacterial bloom occurred in Diamond Lake during summer 2001, when a major bloom, identified as *A. flos-aquae*, was associated with the neurotoxin anatoxin-a (Jones *et al.* 2007). Diamond Lake in 2001 was

unique both in terms of the cell density achieved and anatoxin concentration (Fig. 5a). Time-series plots of cell density and toxin data showed high levels of anatoxin coinciding with peak *Anabaena* cell densities, with an anatoxin-a peak of 262 µg/L occurring during the cell density peak of >550,000 cell/ml (Fig. 5). Except for July 2003 (Fig. 5c) when *Anabaena* cell densities peaked at ~255,000 cells/ml (28 July), *Anabaena* cell density was generally <50,000 cells/ml for other years. Moreover, despite this high July *Anabaena* value and others >25,000 cells/ml, both anatoxin and microcystin were either low or not detectable during the 2002-2004 period (Fig. 5b, c and d). *Microcystis* showed occasional concentrations of <1000 cells/ml (Fig. 5).

A plot of *Anabaena* density vs. anatoxin indicates that toxin concentration was positively related to cell density only during the healthy bloom period in early August 2001 ($r^2 = 0.97$; $p < 0.001$) with no relationship evident after bloom decline (Fig. 6a). No relationship between cell density and toxin was evident for any of the other years. However, during the 2003 bloom, the highest microcystin values (although still relatively low) tended to coincide with higher *Anabaena* values (Fig. 6b). A possible explanation for the lack of anatoxin production from 2002 to 2004 is that although initial identifications of the dominant *Anabaena* were as *A. flos-aquae*, subsequent split samples indicated that *A. lemmermannii* may have replaced *A. flos-aquae* as the dominant *Anabaena* in Diamond Lake (St. Amand, A., pers. comm., 4 April 2005). Although it has not been resolved whether the 2002-2004 blooms actually consisted of a different species, given the apparent change in toxin production between 2001 and 2002, either a new species dominated, or within-species genetic changes shifted away from toxin production. The low levels of microcystin present without anatoxin during 2002-2004 are more consistent with non-*flos-aquae* species of *Anabaena*.

Cyanotoxicity occurrence in WA

Cyanotoxins, mostly microcystins, have been detected in approximately 20 lakes in Washington (Table 1). In several of these lakes cyanotoxin concentrations were not directly measured but were implicated by mouse assay or the presence of cyanobacterial species known to produce toxins and animal deaths. The first documented toxic episodes occurred in eastern WA. In September 1976, 4 dogs died after drinking water with a heavy *A. flos-aquae* bloom in Long Lake, Spokane (Soltero and Nichols 1981). In October 1982, 2 dogs died at Moses Lake, which was experiencing a dense cyanobacterial bloom at the time (Edmondson 1991). More recent animal deaths have been associated with the detection of anatoxin-a in several WA lakes (e.g., American Lake in Pierce County during December 1989; Kitsap Lake in Kitsap County during October 2001; and Anderson Lake in Jefferson County during June 2006). Saxitoxin was also measured at

4 µg/L in Ketchum Lake (Snohomish County) in October 2006. In previous years, anatoxin-a and microcystins were measured in this lake (Table 1).

American Lake

Most of the recent toxic episodes have been documented in western WA, beginning with wintertime toxic blooms of *A. flos-aquae* in American Lake (near Tacoma) in 1989. This bloom persisted into spring 1990 and caused 11 animal poisonings, including the deaths of 5 cats. Anatoxin-a was measured using mouse bioassay and HPLC (Jacoby *et al.* 1994). Toxic *A. flos-aquae* blooms also occurred in this lake during the winters of 1992, 1993, and 1996. Toxic blooms in this lake were anomalous because the lake is only moderately productive and because the blooms occurred during the winter. Jacoby *et al.* (1994) hypothesized that these blooms were associated with increased nutrient, especially phosphorus (P), following winter turnover. Phosphorus released from anoxic lake sediments during stratification apparently fueled winter blooms of cyanobacteria. High water-column total P (30-35 µg/L) and low iron content (<5 µg/L) following lake turnover indicated that iron levels were insufficient to completely remove this released P from the water column.

Steilacoom Lake

In nearby Steilacoom Lake, environmental factors associated with toxic cyanobacterial blooms were investigated during summers 1994 and 1995 (Jacoby *et al.* 2000). A prolonged toxic bloom of *M. aeruginosa* occurred during summer 1994 but not 1995, providing a unique opportunity to compare the environmental conditions that occurred these two years. Total microcystin concentrations, measured by HPLC, ranged from 209 to 1,385 µg/g (Fig. 7). Lake characteristics associated with the toxic bloom in 1994 were: decreased water transparency, high water column stability, high surface water temperature and pH, and decreased lake flushing. Decreased water transparency during 1994 may have been due to significantly lower zooplankton abundance. Jacoby *et al.* (2000) hypothesized that decreased transparency was caused by increased planktivory by higher numbers of coho salmon fingerlings during 1994 and/or inhibition of zooplankton grazing by *Microcystis*.

Lake Sammamish

The environmental factors associated with toxic cyanobacteria blooms were also investigated in Lake Sammamish (32 km east of Seattle) in summer-fall 1999 (Johnston and Jacoby 2003) following a heavy toxic bloom of *M. aeruginosa* in fall 1997. Despite low cyanobacterial abundance in 1999, microcystins were detected using ELISA from late August to mid-September at concentrations that ranged between 0.19 and 3.8 µg/L throughout the lake, with surface (0.5 m

The occurrence and response to toxic cyanobacteria in the Pacific Northwest, North America

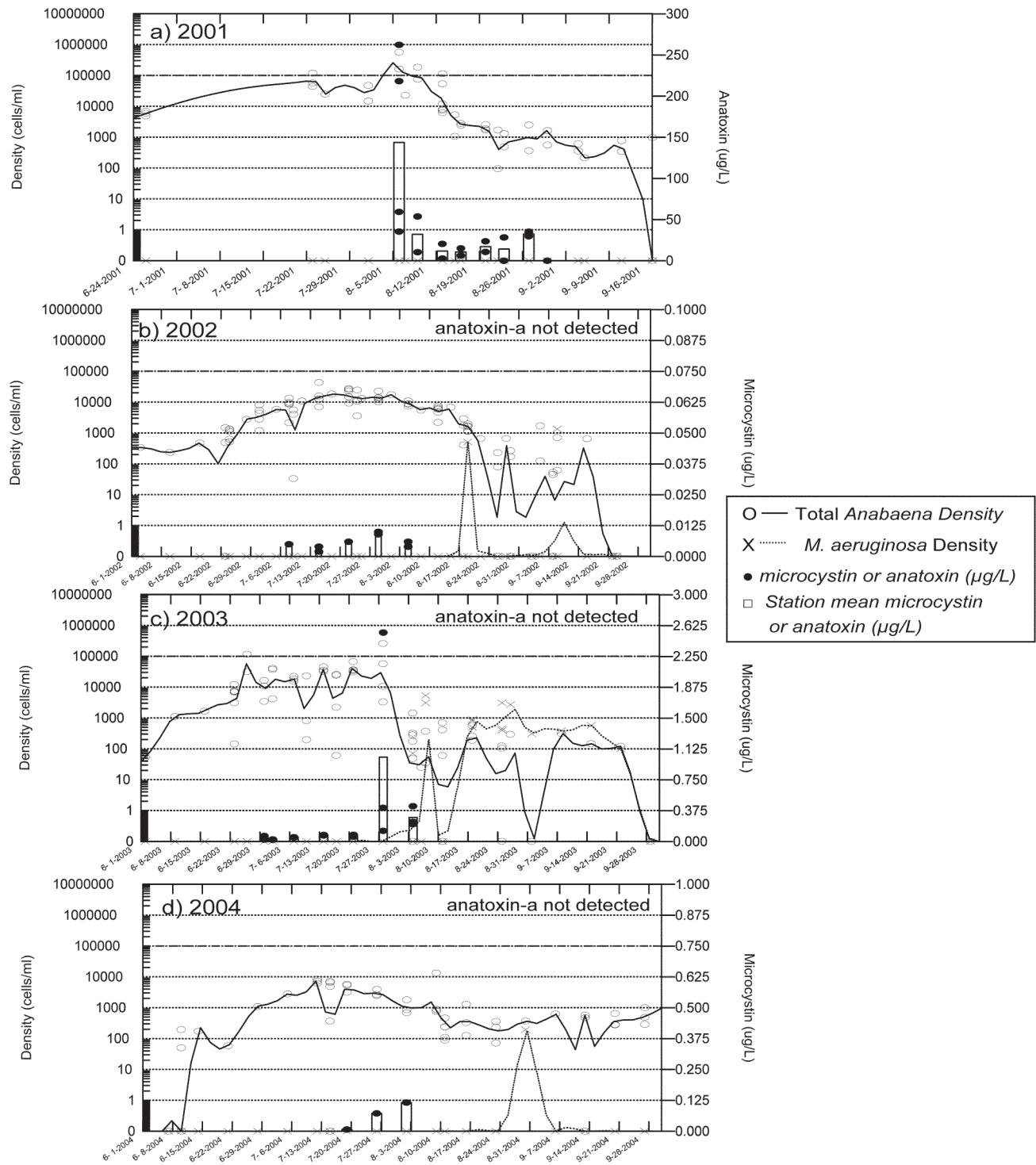


Figure 5. Time-series of total *Anabaena* (all *Anabaena* genera combined) cell density, *M. aeruginosa* cell density, and anatoxin-a or microcystin concentration in Diamond Lake, 2001(a); 2002(b); 2003(c); 2004(c).

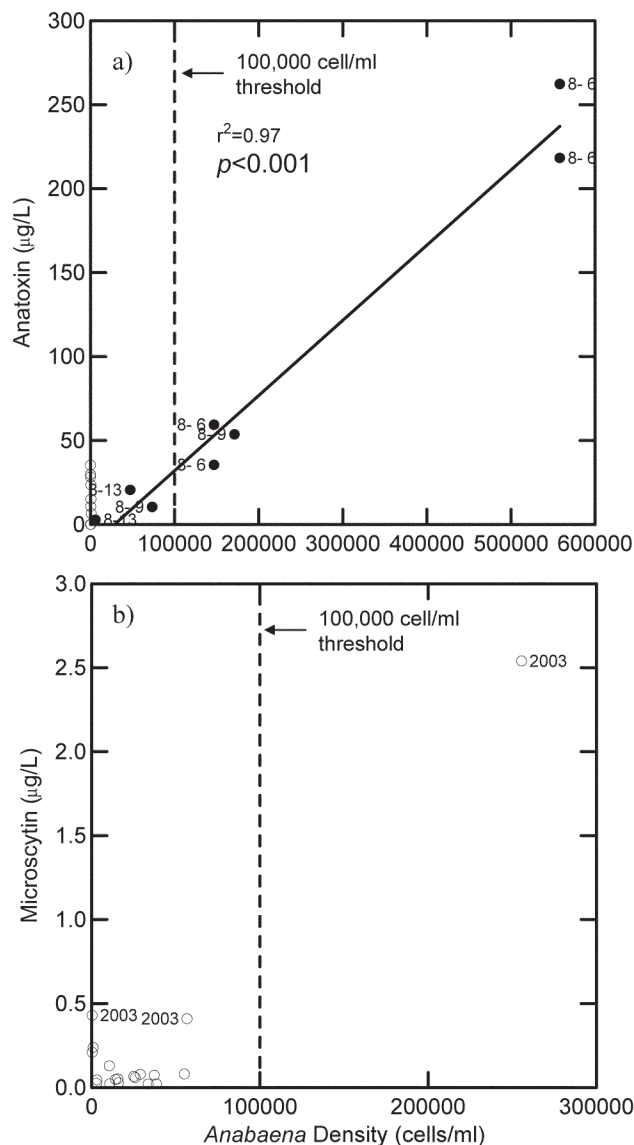


Figure 6.-Relationship between *A. flos-aquae* cell density and anatoxin-a in 2001 (a) and between *A. flos-aquae* and microcystin concentration (b) 2002-2004, Diamond Lake, Oregon.

depth) concentrations that ranged from 1.5 to 3.5 µg/L (Fig. 8). One substantially higher concentration (43 µg/L) was measured near the shoreline. Conditions associated with the toxic episodes in 1997 and 1999 indicate that *M. aeruginosa* was associated with a stable water column, increased surface total phosphorus concentrations (>10 µg/L), and surface temperatures >22°C. Using traps suspended above the sediments, cyanobacterial migration from the sediments to the overlying water was also measured weekly during this study. Migration of *M. aeruginosa* and *Anabaena* sp. occurred in both the deep and shallow portions of the lake. *M. aeruginosa* dominated (89-99%) the migrating cyanobacteria with higher migration rates from the 9-m depth station (1,678 mm³/m²-d) than the

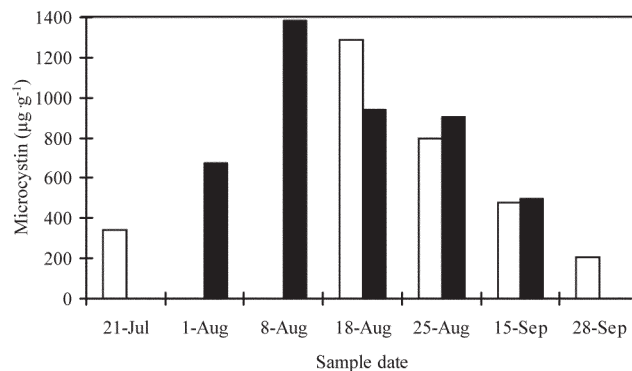


Figure 7.-Microcystin concentrations in phytoplankton biomass in Steilacoom Lake at stations 1 (open bars) and 2 (solid bars) during 1994 (from Jacoby *et al.* 2000; reprinted with permission from NRC Research Press).

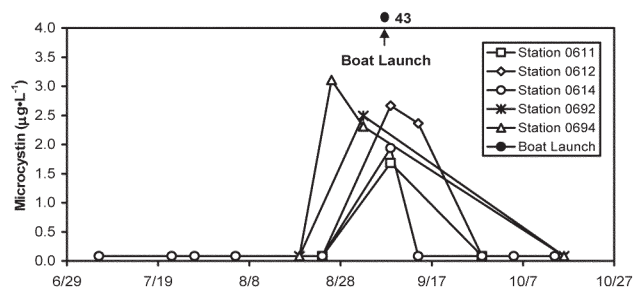


Figure 8.-Microcystin concentrations at 0.5-m water depth in Lake Sammamish during summer 1999 (from Johnston and Jacoby 2003; reprinted with permission of Springer Science and Business Media).

deeper station at 19-m depth (754 mm³/m²-d) during a one-week period in October 1999. External loading of nutrients due to the large rainfall preceding the 1997 toxic episode likely provided the nutrients needed to fuel that bloom. However, microcystins were measured in 1999 despite the lack of rain and subsequent external nutrient loading to the lake. The migration of *M. aeruginosa* from the nutrient-rich sediments may have been the inoculum for the toxic population detected in 1999 (Johnston and Jacoby 2003).

In-depth studies of cyanobacterial toxicity have not been conducted in other lakes in WA. Furthermore, no systematic monitoring for cyanotoxins is being conducted by the state. Monitoring has primarily been prompted by heavy blooms, public concern, or animal poisonings (Table 1). However, King County has recently implemented a long-term monitoring program for cyanotoxins as part of its large lakes program (King County Department of Natural Resources and Parks 2006). Since 2003, microcystins have been routinely analyzed throughout the summer using ELISA and PPIA in Lakes Sammamish, Washington, and Union (see station locations at: <http://dnr.metrokc.gov/wlr/waterres/lakes/LocatorTable>).

aspx.). In Lakes Sammamish and Washington, shoreline samples are collected from the swimming beaches, as well as the deeper routine monitoring stations. To date, microcystin concentrations have been relatively low in all 3 lakes ($< 1 \mu\text{g/L}$) except in several samples collected in beach samples during blooms (Table 1). Multi-year monitoring has been conducted in several other lakes in the state that are “repeat offenders” that regularly exhibit toxic blooms, but the measurements are episodic and reactive to bloom conditions.

Cyanotoxicity occurrence in BC

Microcystins have been detected in approximately 10 lakes in BC (Table 1). Historically there have been cattle kills from toxic cyanobacteria in the central interior (Tunkwa, Leighton, Mamit, and Monte lakes in the Kamloops area) and in the Vanderhoof area (Nulki Lake) of the north central part of the province (Nordin, R., pers. comm., 10 October 2005). In a 1995 survey of 32 lakes in BC, microcystins were detected in 10 of these lakes using HPLC (Nordin 1996). Five of these lakes showed high levels of microcystin-LR (Douglas, Ealget, Nulki, Tabor, and Tachiuk) and 4 had high concentrations of total microcystins (Bouchie, Douglas, Putataekut, and Spokin). In the lakes where microcystins were present, high levels of chlorophyll were also measured (all $>60 \mu\text{g/L}$ with 3 lakes $>2,000 \mu\text{g/L}$).

Several water utilities in BC also monitor water supplies for microcystins. Microcystin concentrations in Cusheon and St. Mary Lakes (Saltspring Island) occasionally exceeded the $1.5 \mu\text{g/L}$ microcystin-LR standard used as a drinking water guideline in BC and were closed for all uses on at least one occasion. Microcystins are also monitored in the water supplies to the Greater Victoria and Vancouver areas and have not been detected (Nordin, R. pers. comm., 12 October 2005).

Responses to toxic blooms

Toxic cyanobacterial blooms are a challenge for managing both drinking water and recreational lakes. The World Health Organization (WHO 1998) has recommended that microcystin-LR concentrations should not exceed $1 \mu\text{g/L}$ in drinking waters. This guideline served as the basis for guidelines developed by several countries to close drinking water supplies to human consumption (*e.g.*, Australia, Brazil, Canada, the Czech Republic, France, and Poland). However, national thresholds for microcystins in drinking waters of the U.S. have not yet been developed. Furthermore, neither the U.S. nor Canada has developed national guidelines to reduce public health risk in recreational waters. Several cities and counties in the Pacific Northwest have used the WHO drinking water guideline of $1 \mu\text{g/L}$ to close lakes to recreational activities; however, this level may be overly protective for lakes that are not being used for drinking water.

Human health effects from exposure to cyanobacteria in recreational waters are diverse and include skin rashes and lesions, nausea, vomiting, headaches, gastroenteritis, conjunctivitis, eye and ear irritations, fevers, and sore throats (Pilotta *et al.* 1997, Chorus *et al.* 2000, Codd *et al.* 2005). While these conditions have been observed in individuals following recreational exposures to cyanobacteria, information on cyanobacterial species, cell densities, and the toxins to which individuals are exposed is typically lacking (Chorus 2005, Codd *et al.* 2005). Although liver damage has been associated with microcystin in drinking water supplies (Falconer *et al.* 1983), we are not aware of epidemiological studies that have evaluated liver damage following incidental recreational ingestion of microcystin tainted waters. Thus, characterization of the hazards to humans during recreational activities has been primarily derived from the potential for adverse health outcomes using human case histories, animal poisonings, limited human epidemiological data (Pilotto *et al.* 1997), and guidance values for microcystins in drinking water (Codd *et al.* 2005).

In heavily used recreational lakes, lake closures can constitute significant economic hardship to local businesses dependent on these uses. In the case of Green Lake in Seattle (WA), a prolonged toxic bloom occurred from early August 2002 through January 2003, with microcystin concentrations from 3 to $100 \mu\text{g/L}$, with most measurements within $30\text{--}65 \mu\text{g/L}$ (Jacoby 2003). Green Lake is the most popular park in the city with several million visitors annually. Due to human health concerns, Seattle Parks and Recreation closed the lake to all contact activities during the toxic bloom until February 2003. During this time, user enjoyment of the lake decreased markedly and local businesses and activities related to recreation were negatively affected. Green Lake was treated with aluminum sulfate/sodium aluminate in spring 2004, and cyanobacterial blooms have subsequently subsided.

Although no state-wide management plan exists for Oregon, the Umpqua National Forest, which manages Diamond Lake, implemented a fish removal program both to restore the game fishery and to control toxic blooms in September 2006. The cyanobacterial algal blooms that occurred in Diamond Lake during the summers of 2001–2006 were likely the result of optimal climatic conditions in conjunction with trophic changes stemming from large populations of non-native tui chub. Two possible pathways for the currently large populations of tui chub to enhance the blooms are (1) by reducing the number of larger sized zooplankton that can efficiently filter algal cells from the water column, and (2) by increasing the water column nutrient concentration through excretion of nitrogen and phosphorus in forms available for algal growth. Eilers *et al.* (2001) showed that initial *Anabaena* akinete increases in Diamond Lake sediments coincided with dense trout stocking, and that further increases occurred as the non-native tui chub greatly increased in population size.

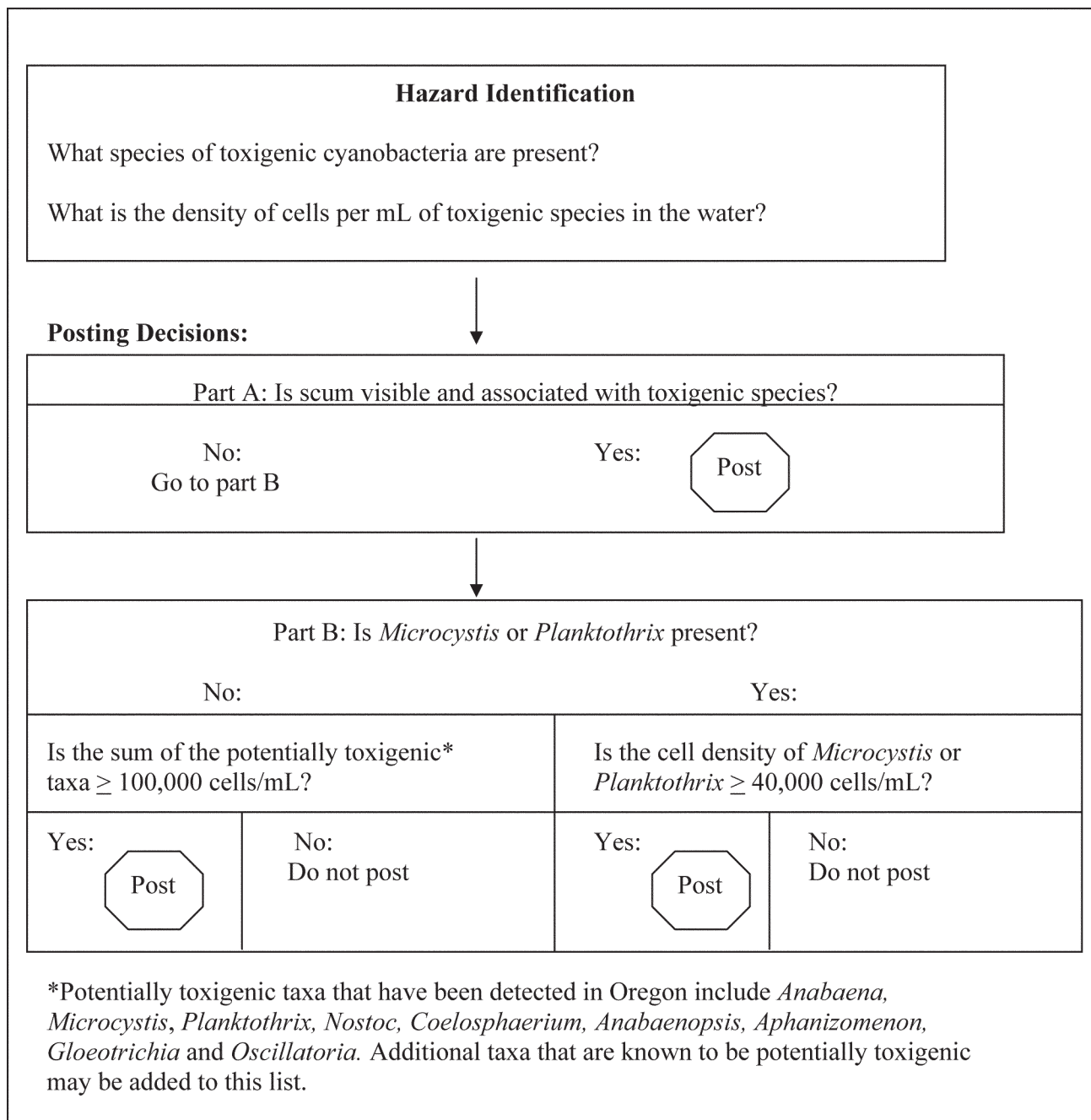


Figure 9.-Oregon Department of Human Services guidance flow chart for issuing health advisories for recreational contact with cyanobacteria (from Stone and Bress 2007; reprinted by permission of Alliance Communications Group, a division of Allen Press, Inc).

Complete fish removal through drawdown and rotenone treatment was successfully completed in Diamond Lake in September 2006.

Partly in response to the need to distinguish between drinking water health advisories and recreational advisories, the Oregon Department of Human Services (DHS) issued a guidance document for issuing and lifting advisories in recreational

waters when toxigenic cyanobacteria are detected (ODHS 2005, Stone and Bress 2007). In 2004 and previous years, lakes were posted when toxigenic cell densities exceeded 15,000 cells/ml, a level that corresponded to an Alert Level III using Yoo *et al.* (1995) recommendations. However, the risk to recreational users at this cell density is considered low and includes symptoms such as skin irritation and gas-

traintestinal disorders. Consequently, the focus of advisory postings for OR is on the risk posed by cyanotoxins and the potential for systemic effects.

DHS uses a flowchart of guidelines to assist in deciding whether or not to post a water body (Fig. 9). Due to high cost and lag time for receiving results, the issuance of advisories is based solely on cell density determinations and is not dependent upon the analysis of toxins. When possible, DHS recommends toxin analysis to better understand the systems being monitored, the potential health implications, and to document historical trends for future advisories (ODHS 2005, Stone and Bress 2007). DHS recommends that if *Microcystis* or *Planktothrix* is not the dominant species in a sample, advisories should be posted if cell densities of total toxigenic cyanobacteria equal or exceed 100,000 cells/ml, or if scums containing toxigenic cyanobacteria are observed. Because *Anabaena* species can produce both anatoxin and microcystins, and based on the taxonomic issues described above for Diamond Lake and multiple co-occurring *Anabaena* species in Lake Selmac, the guideline level of 100,000 cells/ml does not distinguish among the various *Anabaena* species.

Moreover, because *Microcystis* and *Planktothrix* are more likely to produce microcystin toxin compared to other genera, such as *Anabaena* (Chorus and Bartrum 1999, Codd *et al.* 2005), and the observation that almost all *Microcystis* strains are toxic (Carmichael 1995), a lower guideline of 40,000 cells/ml was recommended for issuing advisories based on cell densities that are dominated by these genera. Oregon guidelines for *Microcystis* are similar to recent guidelines developed by Australia, where a value of 50,000 cells/ml is used when a water body is considered unsuitable for primary contact recreation (NHMRC 2005). DHS recommends that an advisory should remain in place until a final quantitative sample confirms the decreasing trend of potentially toxigenic cyanobacteria, and restrictions should remain in place whenever scums are visible. A statewide website for tracking current and past advisories has been posted by DHS (www.oregon.gov/DHS/ph/envtox/maadvisories.shtml).

Statewide guidelines for issuing advisories during cyanobacterial blooms in Washington do not currently exist; however, some counties will post advisories based on the presence of scums or high cell numbers of toxigenic cyanobacteria (*e.g.*, Clark and Pierce counties). The Washington State Department of Ecology (Ecology) recently received funding from the state legislature (\$250,000 annually) to develop an Algae Control Program for the State of Washington. Based on public comments and input from local health districts and lake managers, Ecology and the Washington Department of Health (DOH) will use these funds to develop statewide guidelines for cyanobacterial blooms/toxins and work with a laboratory for toxicity testing. A mail-in service to identify cyanobacterial blooms will be implemented. If those blooms

are composed of toxigenic species, cyanotoxin concentrations will be analyzed. Local health districts and lake managers will be notified when blooms are reported and of the results of the algal identification and toxin analyses. Ecology is also developing a database for historical and new cyanotoxin data.

California Department of Health Services (CDHS) is also in the process of developing statewide guidelines to reduce risk to recreational users when toxigenic cyanobacteria are present. Their approach is likely to be similar to the framework adopted by OR. CDHS has a website with general information about cyanobacteria, possible health effects, and ways to reduce risks from exposure to cyanobacteria (<http://www.dhs.ca.gov/ps/ddwem/bluegreenalgae/default.htm>).

Conclusions

Toxic cyanobacteria blooms have been detected throughout the Pacific Northwest and are of increasing concern in both drinking water and recreational lakes. Microcystins are the most commonly detected cyanotoxins in this region and have been found at water concentrations from <1 µg/L to 30 mg/L. Anatoxins have also been measured and have been associated with the deaths of domestic and wild animals. Despite increased concern for human health and environmental quality, monitoring for cyanotoxins has been very limited. Most of the monitoring conducted to date has been in response to heavy blooms or animal poisonings. Neither the United States nor the Canadian province in this region have a systematic monitoring program in place for cyanotoxins. However, several more intensive studies of toxic episodes have been conducted in N. CA, OR, and WA and have increased our knowledge of the environmental factors associated with toxic blooms and their spatial and temporal patterns. In addition, some local jurisdictions (*e.g.*, King County, WA) and water suppliers are conducting routine monitoring of their water bodies for microcystins.

The relatively high cost of cyanotoxin analyses and limited availability of analytical laboratories that can perform the analyses are impediments to routine, systematic monitoring. Furthermore, standard quality assurance/quality control (QA/QC) procedures have not been developed on a statewide or regional level. There is also very little information on inter-laboratory variability of toxin analyses.

Increased monitoring for cyanotoxins in the Pacific Northwest is crucial to developing guidelines that will decrease human exposures to these potent toxins. Improved laboratory access and QA/QC are also needed to improve data quality and the basis upon which lake closures are made. The WHO drinking water threshold of 1 µg/L microcystin-LR is likely too low to use for closure of recreational lakes. The approach being used in OR to issue health advisories and close recreational lakes should be considered by other states as a model.

Acknowledgments

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