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Modeling Cyanotoxin Production, Fate, and Transport in Surface Water Bodies Using CE-QUAL-W2

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Modeling Cyanotoxin Production, Fate, and Transport in Surface Water Bodies Using CE-QUAL-W2

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Abstract: Cyanobacteria are frequently associated with forming toxic blooms. The toxins produced by cyanobacteria, cyanotoxins, are harmful to both humans and animals. Rising temperatures due to global climate change are expected to increase the occurrence of cyanobacteria, and it is vital that we protect our drinking water supplies and natural water resources. Modeling the production, fate, and transport of these toxins is an important step in limiting exposure to them and evaluating management strategies to mitigate their impact. The research provided here offers an overview of some of the main cyanotoxins of concern and presents preliminary models for the transport and fate of these toxins. Cyanotoxins can be either intracellular or extracellular, and a model for each was developed. The models were incorporated into the two-dimensional (longitudinal and vertical) hydrodynamic and water quality model CE-QUAL-W2. The toxin models were tested using a model of Henry Hagg Lake (Oregon, USA). The models were able to produce similar trends as found in published data, but since the toxin data available at Henry Hagg Lake was minimal, no direct comparisons between model results and field data were made. Four scenarios were conducted to test the functionality of the toxin models in CE-QUAL-W2. The predicted results from each test scenario matched the expected outcomes based on the parameters used in each scenario. Further applications of the toxin models to other water bodies with more consistent toxin data will help verify the accuracy of the models. This research provides a first step at modeling cyanotoxins using CE-QUAL-W2 and provides a framework to further develop the models through continued research of the cyanotoxins.

Keywords: cyanotoxins; cyanobacteria; hydrodynamic and water quality modeling

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1. Introduction

Cyanobacteria are found throughout the world in both fresh and marine water, and since warmer temperatures have been shown to increase cyanobacterial growth, the occurrence of cyanobacteria could likely increase with the rise in global temperatures brought about by climate change [1–4]. While all bacterial and algal blooms can have harmful effects on the environment by reducing the availability of oxygen in water bodies, cyanobacterial blooms can also be harmful due to their ability to produce cyanotoxins, exposure to which may cause illnesses in humans [2,5,6]. The U.S. Environmental Protection Agency (EPA) Contaminant Candidate List 5 (CCL 5) lists cyanotoxins as contaminants in drinking water that may require future regulation [7]. By modeling the production and movement of these toxins, we can evaluate management strategies to mitigate their impact and help protect our drinking water supplies and natural resources.

Four of the main cyanotoxins studied (summarized in Table 1) are microcystins (MC), cylindrospermopsins (CYN), anatoxin-a (ATX), and saxitoxins (STX). Many publications have been produced in the last decade that provide reviews of the various cyanobacteria and cyanotoxins (see [5,8–14]).

Table 1. Summary of the cyanotoxins microcystins, cylindrospermopsins, anatoxin-a, and saxitoxins [9,11].

Toxin	Microcystins	Cylindrospermopsins	Anatoxin-a	Saxitoxins
Toxin Type	Hepatotoxin	Cytotoxin	Neurotoxin	Neurotoxin
Occurrence	Mostly found intracellularly. Extracellular portion likely due mainly to cell lysis.	High extracellular fractions observed. Extracellular portion likely due to active release from cells in addition to cell lysis.	Extracellular portion likely due mainly to cell lysis.	STX congeners may undergo reactions that can change their toxicity.

One of the difficulties with predicting cyanotoxin concentrations is that not all toxic cyanobacteria may produce toxins in all environments, and some species include both toxic and nontoxic cyanobacteria strains [9,12,13]. Additionally, some strains may be capable of producing multiple different toxins. A study by Ballot et al. [15] detected both microcystin and anatoxin-a in one strain of *Arthrospira fusiformis*. The toxins can be present as intracellular toxins (toxins bound within a cell) or extracellular toxins (dissolved in the water). Cell lysis is one of the pathways by which cyanotoxins become extracellular, and some studies have also suggested that release from live cells may also occur. The active release is thought to occur for some CYN-producing species due to the high extracellular concentrations that have been observed [16,17], whereas cell lysis is likely the main cause of extracellular microcystins [18]. However, the overall quantity of toxins present in a water body appears to be largely a factor in the overall abundance of the particular cyanobacteria strains present producing the toxins [9].

Various models have been proposed for the production and degradation of cyanotoxins. For example, Harris and Graham [19] analyzed twelve different "linear and nonlinear regression models" for their ability to predict cyanobacteria and toxin concentrations in a reservoir in Kansas; however, the models were generally unable to predict peak concentrations. Mechanistic models have also been developed that relate cyanobacteria cellular concentrations with a constant toxin production coefficient or cell quota (mass of toxin per cell) to estimate the toxin concentration [20–23]. Table 2 shows a summary of some of these mechanistic models of cyanotoxin production and degradation.

Table 2. Cyanotoxin models in the literature.

Equation	Parameters	References
$\frac{dC}{dt} = \mu_c N_c \varepsilon_c - k_c C$	$C = cyanotoxin\ concentration, \mu g L^{-1}$ $\mu_c = cyanobacteria\ growth\ rate, day^{-1}$ $N_c = cyanobacteria\ concentration, cell\ L^{-1}$ $\varepsilon_c = cyanotoxin\ production\ coefficient, \mu g\ cell^{-1}$ $k_c = first\ order\ cyanotoxin\ decay\ rate, day^{-1}$	[20,21]
$\frac{dM}{dt} = p\frac{dX}{dt} - d_M M$	$M = MC\ concentration, fg\ mL^{-1}$ $X = cyanobacteria\ concentration, cell\ mL^{-1}$ $p = MC\ production\ coefficient, fg\ cell^{-1}$ $d_M = first\ order\ intracellular\ MC\ depletion\ rate, day^{-1}$	[23]
$\frac{dC_e}{dt} = q\left(\frac{dN}{dt}\right) - k_{OH,MC} C_e C_{OH}$ $\frac{dC_i}{dt} = q\left(-\frac{dN}{dt}\right)$ $\frac{dC_t}{dt} = \frac{dC_e}{dt} + \frac{dC_i}{dt}$	$C_e = extracellular\ MC\ concentration, \mu g\ L^{-1}$ $C_i = cell-bound\ MC\ concentration, \mu g\ L^{-1}$ $C_t = total\ MC\ concentration, \mu g\ L^{-1}$ $q = MC\ cell\ quota, \mu g\ cell^{-1}$ $k_{OH,MC} = second\ order\ reaction\ rate\ constant\ between\ OH\ radical\ and\ MCs, M^{-1}s^{-1}$ $C_{OH} = OH\ concentration, M$ $N = cyanobacteria\ concentration\ of\ intact\ cells, cell\ L^{-1}$	[22]

The Hazen-Adams CyanoTOX (Cyanotoxin Tool for Oxidation Kinetics) tool has also been developed that predicts extracellular concentrations of various cyanotoxins after exposure to different oxidants [24,25]. The Version 3.0 of the CyanoTOX tool has been

updated to include the release of intracellular toxins through lysing or leakage using assumed release rates (none, slow, moderate, fast, or instantaneous).

While the factors that affect cyanobacteria and cyanotoxin production are complex, the objectives of this research were to develop a general framework to model cyanotoxin production, distribution, and degradation in surface water bodies that could be incorporated into CE-QUAL-W2 [26,27], two-dimensional (longitudinal and vertical) hydraulic and water quality model of rivers, lakes/reservoirs and estuaries. Even though we are using the CE-QUAL-W2 framework to evaluate cyanotoxin production and transport, the framework is applicable to any water quality model. CE-QUAL-W2 has been used extensively to model the water quality of many different water bodies throughout the world. CE-QUAL-W2 is suited for long and narrow water bodies under stratified conditions and has been used in many different regions, such as those with temperate or tropical climates. A water quality and hydrodynamic model of Henry Hagg Lake in Oregon, USA, was used to test the cyanotoxin model framework.

2. Methods

A modeling framework was developed for cyanotoxin production and degradation so that a predictive model could answer questions about toxic cyanobacterial blooms and their impact on water quality. Once the algorithms were developed, they were added, tested, and evaluated in the model CE-QUAL-W2 [26,27]. The growth and death kinetics of cyanobacteria are treated the same as algae in CE-QUAL-W2 [27]. A simplified equation of the sources and sinks of cyanobacteria in a batch reactor (no sources or sinks associated with inflows and outflows, nor settling since the reactor is well-mixed) based on those used in CE-QUAL-W2 are shown in Equation (1) [27].

$$\frac{da}{dt} = (k_g - k_r - k_e - k_d)a \quad (1)$$

where a is the concentration of cyanobacteria, k_g is the growth rate, k_r is respiration rate, k_e is excretion rate, and k_d is the death rate. Figure 1 shows some of the hypothesized sources and sinks of intracellular and extracellular cyanotoxins in a batch reactor based on the general growth and death kinetics of cyanobacteria as well as processes specific to cyanotoxins.

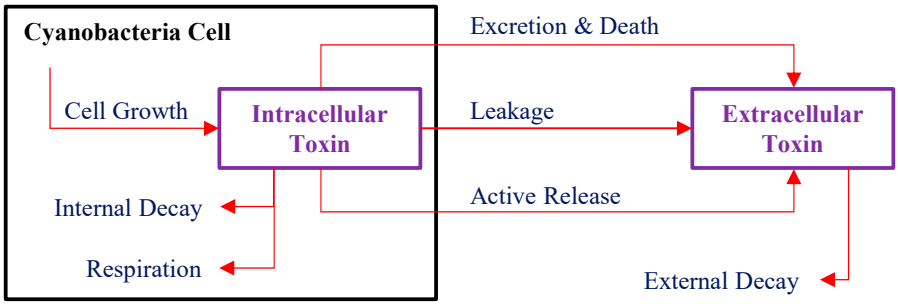

Figure 1. Hypothesized sources and sinks of intracellular and extracellular cyanotoxins.

It is assumed that during cellular respiration, when the cells release carbon dioxide [28], the cells may use the toxin for cell processes, thereby decreasing the amount of intracellular toxin while not adding to the amount of extracellular toxin. It is also assumed that during excretion, when the cells release nutrients and organic carbon [28], the cells may also release toxins to the water column, thereby adding to the extracellular toxin concentration and decreasing the intracellular concentration. Internal decay, leakage, and active release are other hypothesized processes that might occur. Referring to Figure 1, it is assumed that for a specific toxin in a batch reactor, the sources and sinks of intracellular toxin concentration can

be modeled by Equation (2), and the sources and sinks of extracellular toxin concentration can be modeled by Equation (3)

$$\frac{dC_{intra}}{dt} = \left(k_g - k_r - k_e - k_d\right)a\beta - k_{leak}C_{intra} - k_{decay\_intra}C_{intra} - k_{active}C_{intra} \quad (2)$$

$$\frac{dC_{extra}}{dt} = (k_e + k_d)a\beta + k_{leak}C_{intra} - k_{decay\_extra}C_{extra} + k_{active}C_{intra} \quad (3)$$

where C_{intra} is intracellular toxin concentration, $k_g a\beta$ is the increase in intracellular toxin during growth, $-k_r a\beta$ is the loss of intracellular toxin during respiration, $\pm k_e a\beta$ is the loss of intracellular toxin (an increase in extracellular toxin) to cell excretion, $\pm k_d a\beta$ is the loss of intracellular toxin (an increase in extracellular toxin) to death, β is the ratio of intracellular toxin mass to the mass of dry weight organic matter, $\pm k_{leak}C_{intra}$ is the loss of intracellular toxin (an increase in extracellular toxin) due to leakage from the cell, $-k_{decay\_intra}C_{intra}$ is the loss of intracellular toxin to internal decay, $\pm k_{active}C_{intra}$ is the loss of intracellular toxin (an increase in extracellular toxin) due to active toxin release from the cell, C_{extra} is the extracellular toxin concentration, and $-k_{decay\_extra}C_{extra}$ is the loss of extracellular toxin to extracellular decay. It is assumed that the value of β represents the intracellular toxin production rate (100 percent of the production rate) and that the extracellular toxin is only released from the intracellular toxin produced.

Since the distinction between various processes nor the rates of these different processes are not well known or measured, many processes were combined and simplified when applying this model to CE-QUAL-W2 as shown in Equations (4) and (5) for intracellular and extracellular toxin concentrations, respectively.

$$C_{intra} = \sum_{i=1}^{n\ algal\ groups} (CTP)\Phi_a\beta \quad (4)$$

$$\frac{dC_{extra}}{dt} = K_{am}\Phi_a\beta(CTP) + k_{release}C_{intra} - k_{decay}C_{extra} \quad (5)$$

where C_{intra} is the intracellular toxin concentration (mg toxin L^{-1}), Φ_a is the cyanobacteria concentration (mg dry weight organic matter L^{-1}), β is the ratio of intracellular toxin mass to the mass of dry weight organic matter (mg toxin mg dry weight^{-1}), C_{extra} is the extracellular toxin concentration (mg toxin L^{-1}), K_{am} is the cyanobacteria mortality rate (day^{-1}), $k_{release}$ is the combined toxin release rate of intracellular to extracellular representing any possible methods of excretion, leakage, and active release (day^{-1}), k_{decay} is the extracellular toxin decay rate (day^{-1}), and CTP (from CyanoToxin Produced) is described below. Separate toxin mass balances are used for each toxin of interest. In contrast to Equations (2) and (3), these simplified equations assume that the intracellular toxin concentration will not decrease as a result of toxin release (as determined by $k_{release}$), but rather the cell will manufacture any toxin lost to keep the ratio of intracellular toxin to dry-weight organic matter constant.

The CTP term mentioned above is the fraction of the algae group that produces the specific toxin. CTP represents which fraction of the modeled algae groups produce a toxin. For example, if a user selected one algae group to represent all the cyanobacteria in the reservoir, but only half of the group consisted of predicted microcystin producers, then CTP for Microcystin (CTP_MC) would be set to 0.5. If a group is not a toxin producer, then the CTP values for that group would be set to zero.

The intracellular toxin is calculated based on the concentration of cyanobacteria present in a model cell and the fraction of cyanobacteria producing that toxin. The intracellular concentration is summed for all the cyanobacteria groups that produce that toxin. There are no decay or other rates associated with the intracellular toxin concentration other than the scaling with the biomass algae concentration. The extracellular rate equation is a

function of the mortality rate of cyanobacteria, the release rate of intracellular toxin, and the extracellular decay.

Table 3 shows the literature values for dry weight ratios (toxin mass per dry weight biomass), toxin cell quotas (mass of toxin per cell), ratios of toxin to chlorophyll a, and percent of toxin as extracellular for specific species producing each of the four toxins. See Buratti et al. [8], Chorus and Welker [9], Cirés and Ballot [10], and Testai et al. [14] for similar tables with additional values. Table 4 shows the degradation rates of cyanotoxins exposed to various conditions. The taxonomy of some of the cyanobacteria species in Table 3 has been revised [9].

Table 3. Literature values for species producing cyanotoxins.

Species	Ratio of Toxin to Dry Weight (μg Toxin g^{-1} DW)	Cell Quota (fg Toxin $Cell^{-1}$)	Ratio of Toxin to Chlorophyll a (μg Toxin μg^{-1} chl a)	Percent Extracellular of Total Toxin (Intracellular + Extracellular)	References
Microcystins					
Anabaena	1300–3900				[29]
Microcystis aeruginosa	1180–6470		0.59		[30]
Microcystis aeruginosa		18–24	0.70–0.81 [a]	21–47	[31]
Microcystis aeruginosa	1500–9500 [b]	56–165			[32]
Microcystis aeruginosa	555–1113				[33]
Planktothrix agardhii	1170–4460	44–343		0–62	[34]
Planktothrix rubescens	320–4510	27–857		0–23	[34]
Cylindrospermopsins					
Aphanizomenon flos-aquae	2300–6600				[35]
Aphanizomenon flos-aquae				8–58	[17]
Aphanizomenon ovalisporum	90–6370	2–191	0.01–0.53	23–64	[36]
Cylindrospermopsis raciborskii		10–25 [b]		14–50	[37]
Anatoxin-a					
Anabaena circinalis	1396				[38]
Anabaena flos-aquae	1107–13,013				[38]
Aphanizomenon sp.	1562				[38]
Aphanizomenon issatschenkoi		100		21–39	[39]
Aphanizomenon issatschenkoi	6–1683			3–47	[40]
Oscillatoria sp.	4000				[41]
Oscillatoria sp.	2713				[38]
Saxitoxins					
Anabaena circinalis	1580 [c]				[42]
Anabaena circinalis	7–2553 [c]	<1–1105 [c]			[43]
Anabaena circinalis				36–38 [d]	[44]
Cylindrospermopsis raciborskii				53–63 [d]	[44]

DW, dry weight. [a] Value obtained by dividing toxin cell quota by chlorophyll a cell quota. [b] Value estimated from figure in the cited reference. [c] Total saxitoxins (paralytic shellfish poisoning toxins). [d] Saxitoxin congener only, value estimated from ratio of intracellular to extracellular toxins.

Table 4. Influences of different parameters on cyanotoxin degradation.

Parameter	Toxin Degradation Rates [a]	References
Microcystins		
Sunlight and range of water-extractable pigment concentrations from 0–5 mg mL^{-1}	0.006–0.159 day^{-1} (1–85% left after 29 days as estimated from figure in the cited reference)	[45]
Artificial equivalence to approximately 20 days of natural sunlight in surface water	0.071 day^{-1} (24% left after 144 h of light, equivalent to 20 days of natural sunlight)	[46]
254 nm UV light at 250 W m^{-2} in surface water	1326 day^{-1} (1% left after 5 min)	[46]
Chlorination by 3.0 mg L^{-1} NaOCl in surface water	798 day^{-1} (33% left after 2 min)	[46]
Biodegradation in water samples	0.039–0.173 day^{-1} (half-lives of 4–18 days)	[47]
Cylindrospermopsins		
Natural sunlight	4.2–11.1 day^{-1} (half-lives of 1.5 and 4 h for algal extract solutions) 0.046–0.063 day^{-1} (half-lives of 11 and 15 days for natural water samples)	[48]
Range of artificial light from 9–42 μE m^{-2} s^{-1} with initial toxin concentrations of 1 and 4 mg L^{-1}	0.005–0.025 day^{-1} (42–84% left after 5 weeks)	[48]
Range of pH values from 4–10 with initial toxin concentrations of 1 and 4 mg L^{-1}	0.004–0.005 day^{-1} (75–81% left after 8 weeks)	[48]
Range of temperatures from 4–35 °C with initial toxin concentrations of 1 and 4 mg L^{-1}	0.008–0.019 day^{-1} (77–89% left after 14 days)	[48]
257 nm UV light at 300 mW m^{-2}	10.3 day^{-1} (concentration decreased from 1.5 to 1.3 mg L^{-1} after 20 min)	[48]
257 nm UV light at 400 mW m^{-2}	0.924 day^{-1} (half-life of 18 h)	[48]
Artificial equivalence to approximately 20 days of natural sunlight in surface water	0.032 day^{-1} (53% left after 144 h of light, equivalent to 20 days of natural sunlight)	[46]
254 nm UV light at 250 W m^{-2} in surface water	27 day^{-1} (1% left after 250 min)	[46]
Chlorination by 3.0 mg L^{-1} NaOCl in surface water	2318 day^{-1} (4% left after 2 min)	[46]
Anatoxin-a		
Normal pH conditions and microbes	0.139 day^{-1} (half-life of 5 days)	[49]
Natural sunlight at pH range of 6–12 in aqueous toxin solution	3.0–10.4 day^{-1} (half-lives ranging between 96 and 330 min)	[50]
Natural sunlight at pH of 9 in algal lysate solution	1.4 day^{-1} (half-life of 690 min)	[50]
Headspace purged with nitrogen at pH of 9	0.069 day^{-1} (half-life of 10 days)	[50]
Headspace purged with oxygen at pH of 9	0.139 day^{-1} (half-life of 5 days)	[50]
Saxitoxins		
Temperature of 20 °C and pH values of 7 or 9 in sterile water	0.011 day^{-1} (total saxitoxins)	[51]
Temperature of 30 °C and pH value of 7 in sterile water	0.022 day^{-1} (total saxitoxins)	[51]
Temperature of 20 °C in culture medium under different sterilization and deproteinization conditions	0.089–0.240 day^{-1} (total saxitoxins)	[51]
Sterile water at 25 °C	0.015–0.033 day^{-1} (individual congeners)	[52]
Irrigation drain water at 25 °C	0.025–0.075 day^{-1} (individual congeners)	[52]

[a] Unless provided directly, the decay rate was estimated using the first-order decay equation $c = c_0 e^{-kt}$, where c is the toxin concentration after time (t), c_0 is the initial toxin concentration, and k is the decay rate [28].

Table 5 lists suggested ranges of values to use for each parameter from Equations (4) and (5) based on the literature values and model tests. CTP has been described previously, CTB (from CyanoToxin Biomass) represents the β term, CTR (from CyanoToxin Release) represents the $k_{release}$ term and CTD (from CyanoToxin Decay) represents the k_{decay} term.

Table 5. Suggested model parameter ranges for CE-QUAL-W2 toxin models.

Parameter	MC	CYN	ATX	STX
CTP, fraction of cyanobacteria concentration producing toxin	Water body dependent, determined by species present			
CTB, ratio of intracellular toxin to dry weight biomass (mg-toxin mg-DW^{-1})	0.0005–0.01	0.0001–0.01	0.0001–0.015	0.0001–0.005
CTR, release rate day^{-1}	Approx. 0–1 times excretion rate	Approx. 0–2 times excretion rate	Approx. 0–1 times excretion rate	Approx. 0–1 times excretion rate
CTD, extracellular decay day^{-1}	0.01–0.2	0.001–0.1	0.05–1.0	0.01–0.1

DW, dry weight.

3. Results and Discussion

The CE-QUAL-W2 model with the toxin code was applied to a previously developed and calibrated model of Henry Hagg Lake in Oregon, USA [53]. The model simulated eight years of continuous water quality from 1 January 2013 through 31 December 2020, and the model was calibrated to temperature, nutrient, chlorophyll a, and organic matter data from this time period. The first two years of the model simulation (2013 and 2014) were used in this paper to show annual trends. The climate of Oregon near Henry Hagg Lake shows distinct seasonal patterns with hot and dry (summer) weather typically from July through September and cold and wet (winter) weather typically from December through February.

There were minimal toxin data available at the lake, so the model was only used to test the proof of concept of the toxin equations within the code. Two toxins (MC and CYN) were included in the model simulations to test the model's ability to predict multiple toxin concentrations and to test the toxin dynamics between MC and CYN. The model was set up using three algal groups where group 1 represented cooler temperature algal species (diatoms), group 2 represented warmer temperature algal species (greens), and group 3 represented cyanobacteria.

Four model scenarios were conducted to test the functionality of the code updates. The first model scenario tested the toxin decay in the reservoir without any toxin production by the algal or cyanobacterial groups within the lake. This was achieved by adding an initial toxin to the lake and setting all the CTP (fraction of group producing a toxin) values to zero for all the groups. The second model scenario tested toxin decay and production by cyanobacteria death only and for only one group (group 3). The third scenario tested toxin decay and production by cyanobacteria death and release for only group 3. The fourth scenario tested toxin decay and production with all three groups set as toxin producers to test the ability of the model to add together intracellular toxins for multiple groups. The third scenario was chosen as a representative model that would likely be used in further applications of the CE-QUAL-W2 model, and additional results are provided for this scenario. This scenario was chosen because it modeled all four of the toxin parameters for two toxins typically of concern in lakes and from only the cyanobacteria group. Table 6 summarizes the control file parameters chosen for each scenario based on Equations (4) and (5).

Table 6. Summary of toxin model test scenarios for Henry Hagg Lake.

Parameter	Scenario 1		Scenario 2		Scenario 3		Scenario 4	
	MC	CYN	MC	CYN	MC	CYN	MC	CYN
CTP, fraction of cyanobacteria producing toxin, fraction	0	0	0.1	0.3	0.1	0.3	0.1	0.3
CTB, ratio of intracellular toxin to dry weight biomass, fraction	0	0	0.01	0.005	0.01	0.005	0.01	0.005
CTR, release rate, day^{-1}	0	0	0	0	0.01	0.03	0.01	0.03
CTD, extracellular decay rate, day^{-1}	0.1	0.05	0.1	0.05	0.1	0.05	0.1	0.05
Initial concentration	10 ng mL^{-1}		0 ng mL^{-1}		0 ng mL^{-1}		0 ng mL^{-1}	

Total toxin concentrations, as predicted by CE-QUAL-W2, at the surface of the lake for each of the four scenarios are shown in Figures 2–5.

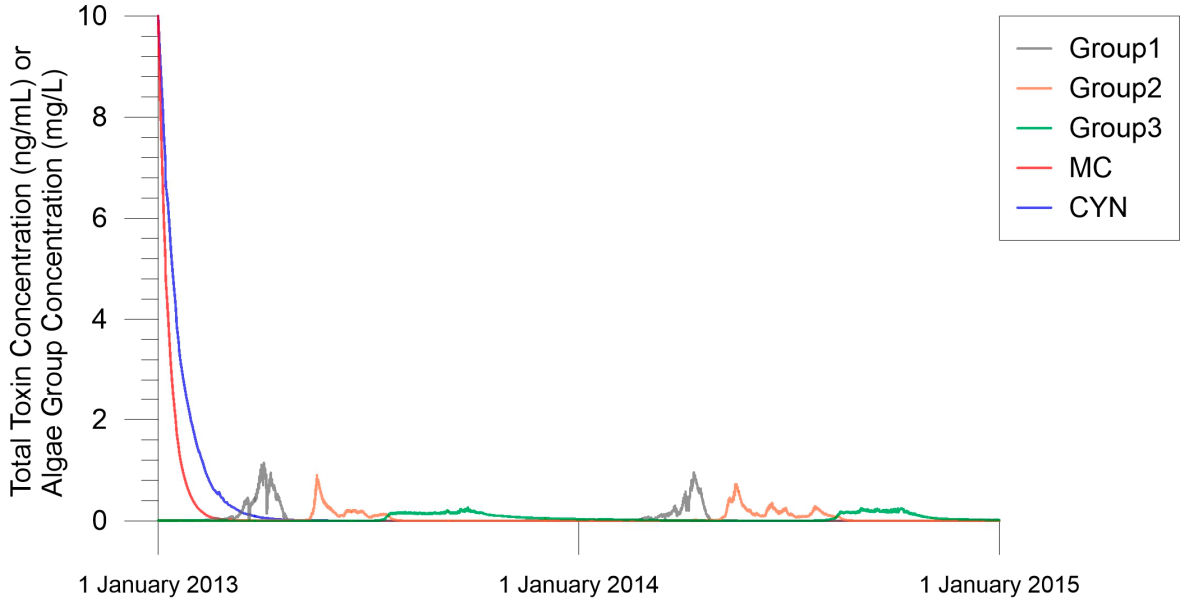

Figure 2. CE-QUAL-W2 toxin and algae predictions for scenario 1 at the surface of Henry Hagg Lake. Microcystins: MC, Cylindrospermopsins: CYN, cooler temperature algal species: Group1, warmer temperature algal species: Group2, cyanobacteria: Group3.

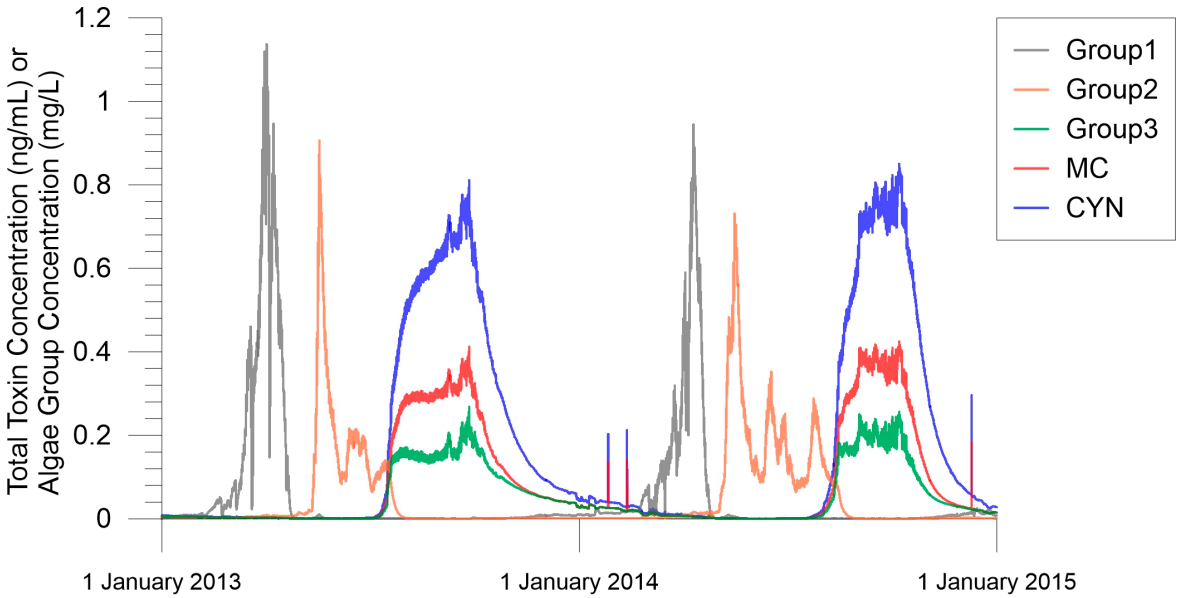

Figure 3. CE-QUAL-W2 toxin and algae predictions for scenario 2 at the surface of Henry Hagg Lake. Microcystins: MC, Cylindrospermopsins: CYN, cooler temperature algal species: Group1, warmer temperature algal species: Group2, cyanobacteria: Group3.

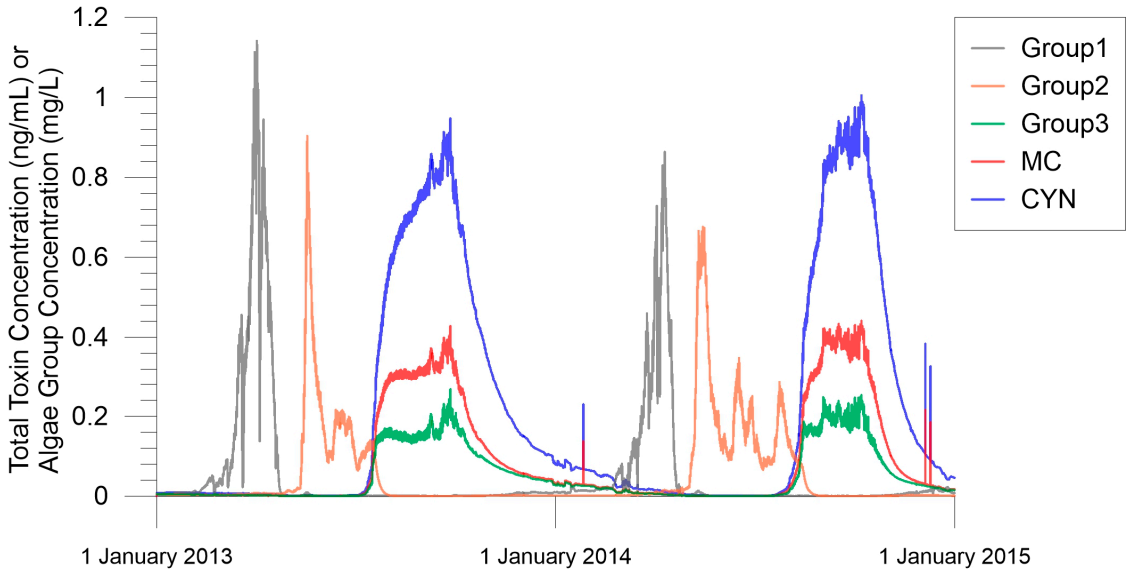

Figure 4. CE-QUAL-W2 toxin and algae predictions for scenario 3 at the surface of Henry Hagg Lake. Microcystins: MC, Cylindrospermopsins: CYN, cooler temperature algal species: Group1, warmer temperature algal species: Group2, cyanobacteria: Group3.

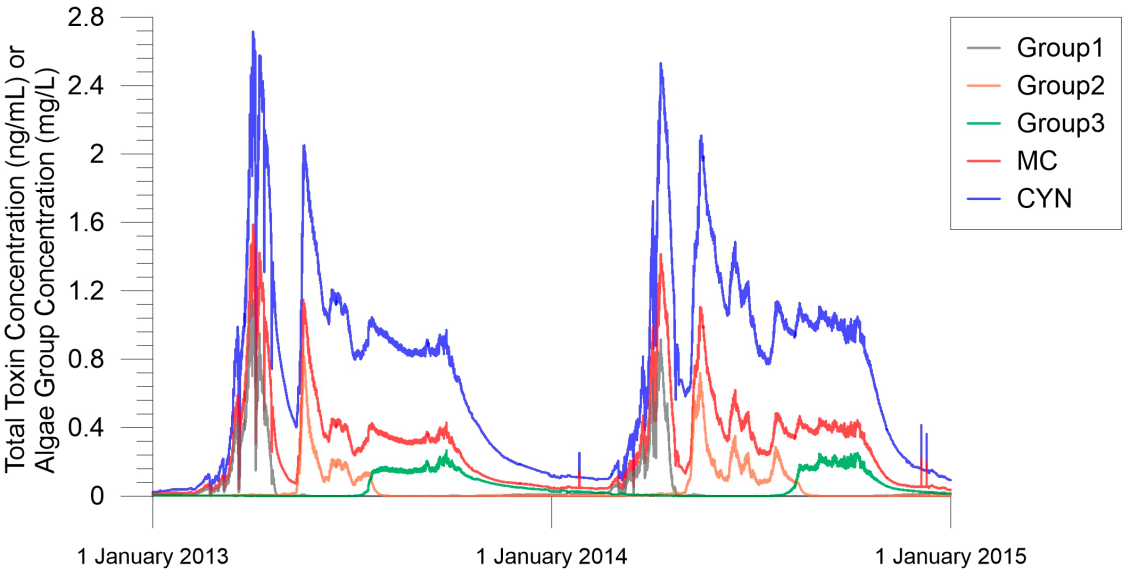

Figure 5. CE-QUAL-W2 toxin and algae predictions for scenario 4 at the surface of Henry Hagg Lake. Microcystins: MC, Cylindrospermopsins: CYN, cooler temperature algal species: Group1, warmer temperature algal species: Group2, cyanobacteria: Group3.

The results from each scenario matched the predicted outcomes based on the toxin parameters chosen for each scenario. The results for the first scenario showed a faster decay for MC than CYN, which agrees with the higher decay rate for MC chosen than CYN. The results for the second scenario showed peaks of extracellular concentrations in the summer, with concentrations close to zero in the winter months. CYN was given a larger CTP value and a slower decay rate which resulted in higher CYN concentrations than MC concentrations. The results for the third scenario showed peaks at similar time periods to the second scenario, but the overall concentrations were higher. Since both toxins in the third scenario had release rates, the concentrations in this scenario were higher than in the second scenario. The fourth scenario showed an even greater increase in concentrations for both toxins compared to the other scenarios since all three groups were turned on as toxin

producers. Figure 6 shows the concentration and percent of intracellular and extracellular MC, and Figure 7 shows the concentration and percent of intracellular and extracellular CYN for the third scenario.

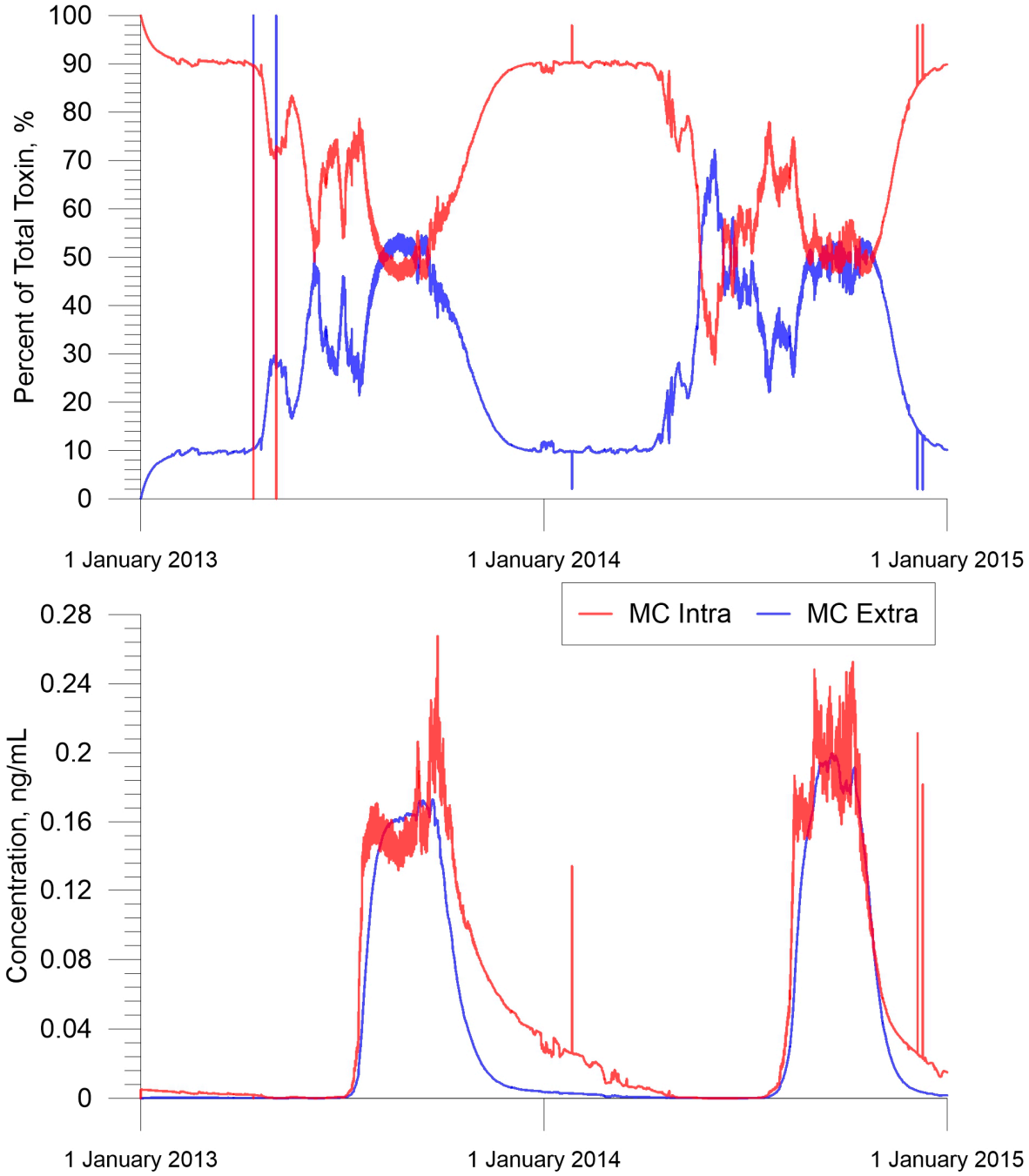

Figure 6. Scenario 3 toxin predictions for microcystin: (**top**) intracellular and extracellular percent of the total toxin concentration, (**bottom**) intracellular and extracellular toxin concentrations.

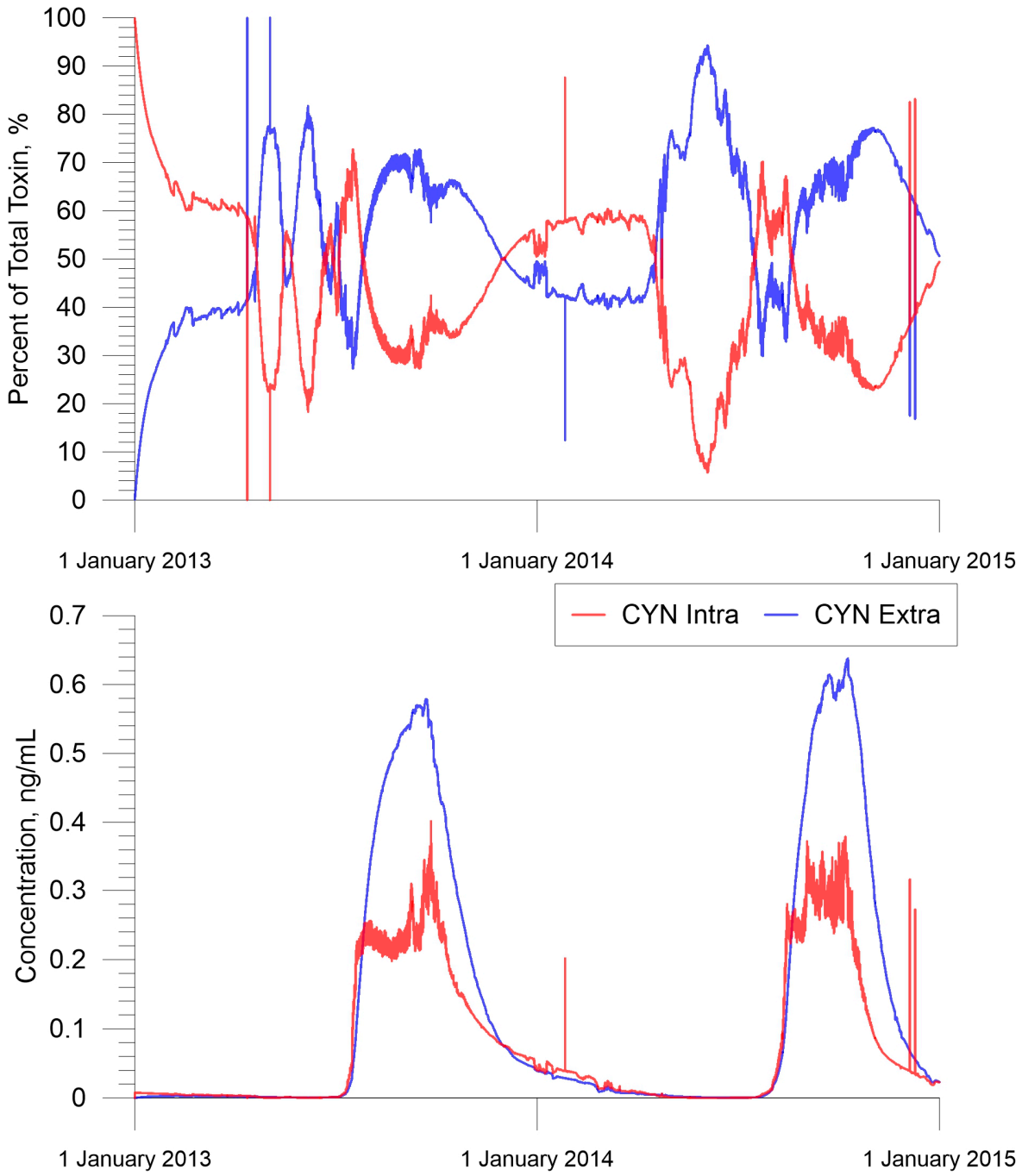

Figure 7. Scenario 3 toxin results for cylindrospermopsin: (**top**) intracellular and extracellular percent of the total toxin concentration, (**bottom**) intracellular and extracellular toxin concentrations.

The model results showed peak MC concentrations of about 0.4 ng mL^{-1} in the summer months and peak CYN concentrations of about 1 ng mL^{-1} in the summer months. The relative fractions of the intracellular and extracellular components of each toxin were similar to the literature values reported for microcystins and cylindrospermopsins. The intracellular fraction of MC is often found at a higher percentage than the extracellular fraction. Figure 6 shows that the model predicted approximately equal fractions of intracellular and extracellular MC during the summer months and predicted the intracellular toxin to be about 90 percent of the total toxin concentration during the winter months. In contrast to MC, CYN is often found at higher extracellular concentrations than intracellular concentrations. Figure 7 shows that the model predicted extracellular CYN to be about

70 percent of the total toxin concentration in the summer and fall. This agrees with CYN being found at high extracellular concentrations. The model predicted that most of the toxin would be present as the intracellular toxin in the winter for both MC and CYN. This is likely due to increased flows and more mixing in the winter that led to the dilution and removal of the extracellular toxin that exceeded the amount of toxin being produced during those months.

Toxin data were available at Henry Hagg Lake in the spring of 2019 for microcystin and cylindrospermopsin [54]. All the recorded values for CYN were classified as non-detects, and all but three measurements for MC were recorded as non-detects (0.15, 0.2, and 0.43 ng mL^{-1}). The model results showed peak MC concentrations of about 0.4 ng mL^{-1} in the summer months and peak CYN concentrations of about 1 ng mL^{-1} in the summer months. The peak microcystin concentrations, as predicted by the model, were in the approximate range of those measured in the lake, but since there was minimal field data, it is difficult to adequately compare these values. Even though no cylindrospermopsin concentrations were detected in the lake, the toxin was still included in the model simulations to test the model's ability to predict multiple toxin concentrations and to test how the dynamics vary between MC and CYN.

4. Conclusions

The goals of this research were to develop models for the transport and fate of cyanotoxins in surface water bodies and to incorporate the models into the two-dimensional (longitudinal and vertical) hydrodynamic and water quality model CE-QUAL-W2. The models were developed with various parameters that can be adjusted depending on the type of toxin being produced or the species producing the toxin, such as the leakage and loss rates, decay rates, and the ratio of intracellular toxin to dry-weight organic matter (β). These parameters allow for flexibility in applying the model to different cyanobacteria species and different toxins. While the model allows for flexibility in choosing values for each parameter, the values are fixed for each toxin. Future research could help to develop each of the model parameters as functions of changing environmental conditions such as light, temperature, and pH; however, as mentioned previously, the quantity of toxins present in a water body is due mainly to the abundance of toxin-producing strains.

The toxin models were incorporated into the CE-QUAL-W2 code and tested on a previously developed model of Henry Hagg Lake. Four scenarios were used to test the functionality of the toxin models in CE-QUAL-W2. The model results from each scenario matched the predicted outcomes based on the toxin parameters chosen for each scenario. There were limited field data available for the toxins, so the model was only used to test the proof of concept of the toxin equations within the code, and no direct comparisons between model results and field data were made. The model results of the relative fractions of intracellular and extracellular microcystin and cylindrospermopsin concentrations were similar to what would be expected based on the literature data for these two toxins. The model results showed that intracellular concentrations of both toxins were higher than the extracellular concentrations in the winter months. This is likely due to higher flows and more mixing in the winter leading to dilution of the extracellular concentration. Further applications of the model to water bodies with more consistent toxin data will help verify the accuracy of the CE-QUAL-W2 model in predicting toxin concentrations. The toxin models developed as part of this project are a first step in modeling toxin fate and transport in surface water bodies using CE-QUAL-W2.

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