OBJECTIVES:
Toxin-releasing algae have consistently increased in Lake Erie over recent decades due to anthropogenic nutrient loads and meteorological conditions [Michalak et al., 2013]. Microcystin, a hepatotoxin excreted by cyanobacteria, is one of the most common and harmful toxins released by algae [Graham et al., 2010; Hotto et al., 2008]. The World Health Organization has set a drinking water guideline of 1 ug/L and recreational water guideline of 20 ug/L. Peak concentrations in Lake Erie have likely exceeded 1000 ug/L [Michalak et al., 2013]. There is a clear need to understand the transport and fate of microcystin in Lake Erie and other surface water bodies.

The goal of this study is to determine the specific role of interactions between lake water and lakebed sediments in attenuating microcystin plumes. In laboratory experiments, microcystin degrades faster in aerobic sediments than the water column [Chen et al., 2008; Song et al., 2014]. Microcystin also has a moderate sorption affinity for sediments [Chen et al., 2006; Wu et al., 2011]. We therefore hypothesize that the exchange of lake water through shallow lakebed sediments enhances the removal and attenuation of microcystin in the water column. We will test this hypothesis through a numerical modeling study that will explore the necessary sediment properties and hydrodynamic conditions for efficient removal of microcystin in shallow lakebed sediments.

METHODOLOGY:
We will conduct a numerical sensitivity test to quantify how surface water-pore water exchange impacts removal of microcystin-LR from surface water. Specifically, we will develop spatially explicit models of reactive microcystin transport in idealized lakebed sediments with varying rates of surface water-pore water exchange (Fig. 1). Similar modeling approaches have been used to quantify the fate of conservative solutes and non-conservative solutes such as nitrate near sediment-water interfaces [Bardini et al., 2012; Cardenas et al., 2008; Sawyer and Cardenas, 2009]. Briefly, the simulation approach first involves solution of the groundwater flow equation in shallow lakebed sediment. Flow paths through the sediment will be prescribed based on surface water depth, wave
height, currents, and bed topography according to existing mathematical solutions [Elliott and Brooks, 1997; King et al., 2009; Sawyer et al., 2013]. The basal boundary will be treated as a no-flow boundary.

Fig. 1. Preliminary simulation of microcystin transport in rippled lakebed sediment. Surface water flows left to right. Pore water flow is shown with black streamlines. Colors show microcystin-LR concentrations in shallow lakebed. The proposed study will quantify removal rates due to sorption and biodegradation in sediments under various current and wave conditions.

Next, the velocity field from the groundwater flow simulation will be used to solve the unsteady, two-dimensional advection-dispersion-reaction equations for dissolved oxygen and microcystin-LR. Transport of dissolved oxygen must be simulated to account for substantially lower degradation rates of microcystin in anaerobic sediments [Song et al., 2014]. Transformations of dissolved oxygen and microcystin will be treated using first-order kinetics [Chen et al., 2008; Song et al., 2014]. Sorption of microcystin will be represented with a linear isotherm model using distribution coefficients from the literature [Song et al., 2014; Wu et al., 2011]. Along the top of the domain (sediment-water interface), fixed concentrations of dissolved oxygen and microcystin will be assigned at downwelling zones to represent concentrations in lake water. Upwelling zones will be treated as advective-flux boundaries. The transport simulations will be run until concentrations in upwelling water (return flow to the lake) approach steady state. For each simulation, we will quantify the aerobic volume of sediment, masses of sorbed and mobile microcystin per unit area of lakebed, and degradation and removal rates per unit area of lakebed. We will also test the behavior of unsteady conditions (for example, changes in wave energy) on transport. These longer simulations will test whether fluctuations in wave energy may drive episodic sorption and release of microcystin from sediments. All models will be executed in COMSOL, a generic finite-element solver designed to represent multiphysics flow and transport processes.

We will vary the hydraulic conductivity of lakebed sediment to explore the effects of sandy or silty sediments on removal rates. We will also vary current and wave energies to explore the effects of hydrodynamics on surface water-pore water interactions and removal rates. The key product of these sensitivity studies will be plots of microcystin removal rates in lakebed sediment as a function of surface water hydrodynamics and lakebed sediment properties that can be used to assess the importance of lakebed sediments in the attenuation of microcystin plumes under a variety of environmental conditions.

RATIONALE:

Harmful algal blooms and algal toxins are a growing concern in Lake Erie and many inland waters. In August of 2014, a prolonged harmful algal bloom in western Lake Erie triggered a ban on tap water for 500,000 people in Toledo. In the US alone, 96% of all Midwestern lakes tested positive for microcystin [Graham et al., 2010]. Microcystin is also a global concern in European, Asian, and Australian lakes [Bruno et al., 2012; Ueno et al., 1996].

Algal toxin plumes are particularly challenging to manage because we understand little about the factors that lead to their occurrence and mechanisms that control their attenuation. A better understanding of attenuation mechanisms will improve our predictions of the severity and duration of algal toxin plumes and help develop treatment technologies or best management practices that optimize attenuation. Common attenuation mechanisms for organic toxins in the environment include photodegradation, sorption to sediments, biological degradation, and dispersion. Preliminary studies have suggested that photodegradation is minimal, in part because toxins often co-occur with dense macrophytes and algal blooms that reduce light penetration in the water column [Chen et al., 2008]. The sorption affinity of microcysin-LR is also moderate to minimal, depending on the organic and clay
content of sediments [Wu et al., 2011]. Biodegradation is therefore likely to be a primary attenuation mechanism, along with dispersion. Laboratory studies have shown that the greatest biodegradation rates occur in the presence of oxygen and sediment-dwelling bacteria [Song et al., 2014]. We therefore posit that the transport of microcystin through shallow, aerobic sediments significantly enhances the attenuation of microcystin in the water column.

The proposed numerical experiments will test the potential importance of this attenuation mechanism in lake settings such as Lake Erie. Although there are numerous compounds and consumers of algal toxins, we focus on microcystin-LR for two reasons: 1) it is one of the more common and harmful algal toxins [Graham et al., 2010], and 2) the sorption and degradation properties of microcystin-LR have been measured in multiple laboratory studies [Chen et al., 2006; Chen et al., 2008; Song et al., 2014; Wu et al., 2011]. At the end of the proposed modeling study, we will have a better understanding of how sediment-water interactions accelerate attenuation of microcystin plumes. We will also know the influence of lakebed permeability, wave conditions, and currents on this attenuation mechanism. Models will directly answer the following key questions: Are surface water-pore water exchange and biodegradation in shallow sediments responsible for removing substantial fractions of microcystin loads in lakes? For which sediment characteristics and hydrodynamic conditions is removal most efficient? Answers to these questions will directly improve our ability to predict the attenuation of microcystin in the environment and the conditions that promote either sustained or short-lived plumes.