HISTORICAL OCCURRENCE AND CURRENT STATUS OF HARMFUL ALGAL BLOOMS IN SUFFOLK COUNTY, NY, USA

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Coastal marine ecosystems are among the most ecologically and socioeconomically productive on the planet. Anthropogenic processes associated with eutrophication and climate change can make these coastal marine ecosystems more susceptible to ecological perturbations including harmful algal blooms (HABs). The spatial and temporal expansion and increased intensity of HABs is a globally recognized phenomenon and this expansion has already been observed in Suffolk County where HABs have become a human health, economic and environmental threat to local waters. While HABs were not reported in Suffolk County from 1954 – 1984, today, Suffolk experiences at least five types of HABs annually, a distinction potentially unmatched in the US. Brown tide (*Aureococcus*) have occurred across the east end and south shore and caused and/or contributed to the demise of these once thriving fisheries. Freshwater toxic cyanobacteria blooms have occurred in more than two dozen lakes across the County. Rust tides caused by the dinoflagellate *Cochlodinium polykrikoides* occur in late summer within Shinnecock and the Peconic Estuary. Paralytic shellfish poisoning (PSP) due to the dinoflagellate *Alexandrium* and the presence of saxitoxin contaminated shellfish over federal regulatory limits has occurred more than a dozen times across six locations and okadaic acid contaminated (diarrhetic shellfish poisoning (DSP) toxins) shellfish due to *Dinophysis acuminata* blooms have been found in multiple locations. Concurrent with these repeated occurrences of HABs have been the decline in shellfisheries and an increase in fish and animal kills associated with these HABs. These events have stimulated a need for action to protect our local waters. This report provides detailed information regarding each of these HABs including local and regional historical accounts of each type of HABs, impacts of these HABs, factors stimulating these events, and potential mitigation strategies that may be used to minimize their occurrence.

INTRODUCTION

Coastal marine ecosystems are among the most ecologically and socioeconomically productive on the planet, providing an estimated US$25 trillion, or about 77% of the global total, worth in ecosystem goods and services, annually (Martínez et al., 2007). About 40% of the global population lives within 100km of the coast (Martínez et al., 2007) and nearly 75% of the US population lives within 75km of the coastline, making these regions subject to a suite of anthropogenic induced stresses (de Jonge et al., 2002; Valiela, 2006) which in turn can lead to ecological perturbations such as harmful algal blooms and hypoxia (Cloern, 2001; Anderson et al., 2008; Heisler et al., 2008). Anthropogenic nutrient loading, ocean warming, and increases in CO_2 levels are all factors that are driven by urban development and industrialization (Nixon, 1995; Borges and Gypens, 2010; Cai et al., 2011) and have been cited as factors influencing harmful algal blooms (HABs) around the world (Hallegraeff, 1993; Anderson et al., 2008; Fu et al., 2008; Heisler et al., 2008; Fu et al., 2010; Hallegraeff, 2010; Fu et al., 2012), many of which can disrupt important ecosystem services and coastal industries (Cosper et al., 1987; Hoagland et al., 2002; Bricelj and MacQuarrie, 2007), as well as negatively affect human health (Anderson et al., 2012b).

Here, for the sake of uniformity, we adopted the Harmful algal bloom and hypoxia research and control act (HABHRCA, 2016) definition of a harmful algal bloom (HAB): A small subset of algal species – including diatom, dinoflagellate, and cyanobacterial blooms – that produce toxins or grow excessively, harming humans, other animals, and the environment. In accordance with this definition, a HAB need not reach visible proportions to be considered harmful (e.g. *Alexandrium*) or produce a toxin to produce deleterious environmental and/or economic effects (e.g. *Aureococcus*). Smayda (1997b) noted that while biomass is most often the criterion used to define a bloom for non-toxic species, for harmful species, the presence of toxins or biomass above background levels is most often used.

The spatial and temporal expansion and increased intensity of HABs is a globally recognized phenomenon (Hallegraeff, 1993; Glibert et al., 2005). HABs that synthesize biotoxins (e.g. *Alexandrium, Dinophysis*, freshwater cyanobacteria) and those considered ecologically destructive (e.g. *Aureococcus, Cochlodinium*) are a growing societal concern in many coastal regions due to their impacts on human health, the environment and the economy

(Anderson et al., 2008; Anderson et al., 2012b). Human health syndrome causing HABs are often associated with substantial economic losses due to the closure of shellfish beds containing toxic shellfish (Hoagland et al., 2002; Koukaras and Nikolaidis, 2004; Jin and Hoagland, 2008; Jin et al., 2008) as are ecologically destructive HABs due to loss of fisheries (Bricelj and MacQuarrie, 2007). HABs undergo several developmental phases including initiation, growth and maintenance, and termination (Steidinger and Garcés, 2006). Documenting the factors effecting the growth and maintenance period is of critical importance as this is the time period during which vegetative toxin-forming cells can accumulate in shellfish (e.g. *Alexandrium, Dinophysis*) or ecologically destructive HABs (e.g. *Cochlodinium*) can have detrimental effects on local wildlife. Any factor acting to extend this developmental phase can have subsequent effects on human health and coastal economies. Moreover, the need for species-specific, even strain-specific, information is of the utmost importance as it has become increasingly evident that a one-explanation-fits-all bloom populations scenario does not exist (Burkholder and Glibert, 2009; Pitcher, 2012). Given the human health and economic threats that these HABs pose and the global increase in these events (Hallegraeff, 1993; Van Dolah, 2000) more strain-specific documentation and research is needed to understand what promotes and aids in the maintenance of these blooms.

Anderson et al. (2012b) cited the mechanisms facilitating the global expansion of HABs as dispersal through human activities such as ballast water discharge and shellfish translocation, improved detection of HABs and their toxins due to better chemical instrumentation and improved communications among scientists, increased aquaculture operations in coastal waters, stimulation of HABs as a result of cultural eutrophication, and stimulation of HABs by climate change. It is likely that many of these mechanisms have contributed toward HABs in Suffolk County. Regarding eutrophication, a consensus on the relationship between eutrophication and HABs was discussed by Heisler et al. (2008) who indicated degraded water quality from increased nutrient pollution promotes the development and persistence of many HABs and is one of the reasons for their expansion in the U.S. Heisler et al. (2008) further indicated that high-biomass HABs must have exogenous nutrients to be sustained and that the management of nutrient inputs to watersheds can lead to significant reduction in HABs. The anthropogenic nutrient-HAB link is often location- and/or species-specific, however (Anderson et al., 2008; Heisler et al., 2008; Davidson et al., 2014).

AN OVERVIEW OF HABS IN THE MARINE WATERS OF SUFFOLK COUNTY, NY

Suffolk County has seen an increased incidence of HABs over the past several decades (Fig. 1). A full summary of the HABs affecting Suffolk County including their impacts, morphology, appearance, phenology, and regions of common occurrence are provided in Table 1. The first documented HABs on Long Island were green tides in the early 1950s that occurred within Moriches and Great South Bays. From 1954 through 1985, a time during which the largest hard clam and bay scallop fisheries on the US east coast were found on Long Island, there were no documented HABs. In 1985, the first ever brown tide (*Aureococcus*) occurred across the east end and south shore and subsequently caused and/or contributed to the demise of these once thriving fisheries. In 2003, the first freshwater toxic cyanobacteria bloom was documented in Lake Agawam, Southampton. This was followed by the first rust tide event caused by the dinoflagellate *Cochlodinium polykrikoides* in 2004 in the Peconic Estuary. In 2006, the first documented paralytic shellfish poisoning (PSP) closure in NY occurred in Northport Bay due to the dinoflagellate *Alexandrium* and the presence of saxitoxin contaminated shellfish over federal regulatory limits (National Shellfish Sanitation Program (NSSP) – Model Ordinance). Once again, in Northport Bay during the summer of 2011 okadaic acid contaminated (diarrhetic shellfish poisoning (DSP) toxins) shellfish were reported in Long Island, New York due to an extraordinary *Dinophysis acuminata* bloom. Since these first documented events, all of these HABs have recurred annually across Suffolk County. In addition, macroalgae blooms have occurred sporadically in different regions of Suffolk County for more than two decades. Figure 2 indicates the distribution of these HABs across Suffolk County during the last decade. With more than six different HABs occurring in Suffolk County on an annual basis, it may be argued that no other county on the US East or Gulf Coast or perhaps the nation experiences as many HABs as Suffolk. This report provides information regarding each of these HABs focusing on regional and local dynamics, distribution, and impacts as well as processes known to promote these HABs on both local and global scales, economic effects and potential remedial measures.

ECOLOGICALLY SIGNIFICANT BLOOMS IN SUFFOLK COUNTY

SMALL FORM, CHLOROPHYTE BLOOMS
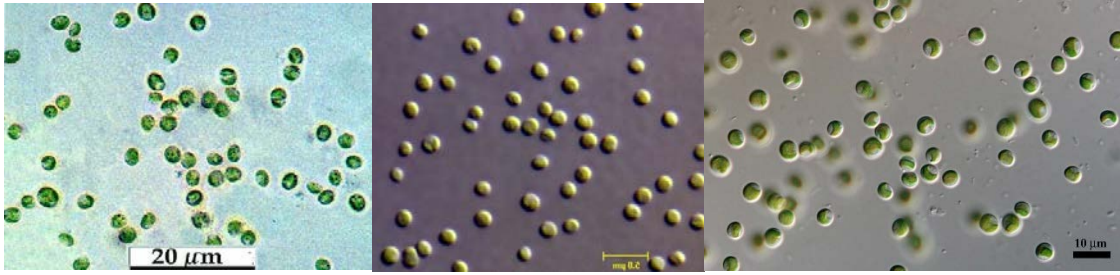
Nannochloris (source: http://cfb.unh.edu/phycokey/phycokey.htm), *Stichococcus* (Source:CCALA)

The first notable HAB in Suffolk County was that of the "small form" picoplankton, the chlorophytes *Nannochloris atomus*, and S*tichococcus* sp. that occurred within the waters of Moriches and Great South Bays in the early 1950s. These blooms had a severe detrimental effect on shellfish, especially the oyster fishery extant at that time. A series of reports produced by the Woods Hole Oceanographic Institution (Redfield, 1952; Ryther et al., 1958) linked the blooms to effluent from duck farms located on tributaries entering the bays, and the closure of Moriches Inlet effectively trapping the added nutrients and algae within the bays. The green algae could utilize organic (including uric acid, the major waste product of the ducks), as well as inorganic sources of nitrogen. In contrast, Redfield et al. (1958) noted that *Nitzschia*, a local diatom, grew well with nitrite and nitrate, but not with ammonia or organic forms of nitrogen.

Redfield noted that the low N:P ratio of the duck wastes, and the ratio in the Terrell River (~3.3:1) as compared to plant requirements (~16:1) resulted in the utilization of nitrogen by the plants and an excess of phosphorus in the water, and thus nitrogen-limited conditions. He also noted the desirability of reducing the introduction of organic wastes, as well as the discharge of the products of decomposition, into the bay. Glancy (1956) saw these small forms blooms in the context of a changing and developing Long Island as he stated "… the rapidly increasing growth in population and industry on Long Island will inevitably create additional pollution loads and it is obvious the degree of treatment must not only take care of bacterial and biological oxygen demand loads, but must reduce the phosphorus-nitrogen content to prevent harmful plankton blooms."

Ryther et al. (1958) noted that while the inlet was closed, most algal growth occurred within the tributary area of nutrient release, where all the available nitrogen was removed from the water, and that bloom conditions within the bay were a result of tributary transport.

However, in 1958 when Moriches Inlet was reopened and widened it was found that, although there was still little or no nitrogen in bay waters, the blooms ended and phytoplankton populations in the bay now differed from those in the rivers and coves. They surmised that the nutrients carried into the bays were now in the inorganic form, and subsequently assimilated by diatoms populations.   That the small-form blooms originated near the duck farms, with populations being diluted by tidal exchange via Great South Bay and Shinnecock Bay, was reiterated by Ryther and Dunstan (1971), and Ryther (1989).

BROWN TIDES
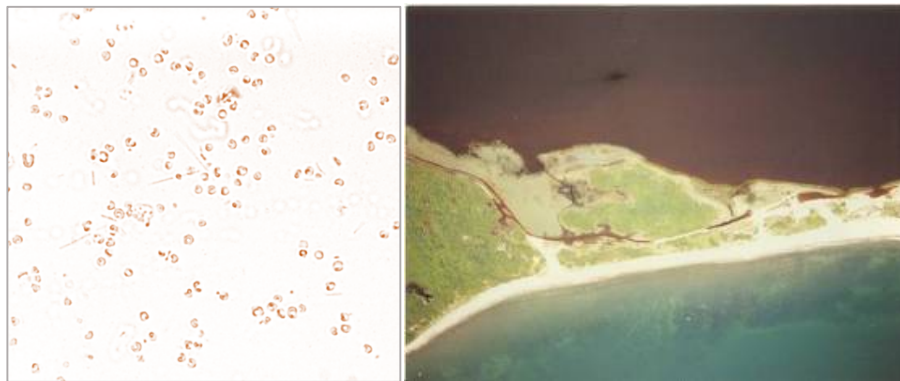
A light microscope image of *Aureococcus anophagefferens* (Photo credit: C.J. Gobler) and an aerial photograph of a brown tide in Great South Bay (Photo credit: E.M. Cosper)

Brown tides are caused by the small (2-3 µm) pelagophyte *Aureococcus anophagefferens* Hargraves et Sieburth. *Aureococcus* has caused destructive brown tide blooms in northeast and mid-Atlantic U.S. estuaries for more than 30 years (Nuzzi and Waters, 2004), and more recently in South Africa (Probyn et al., 2001; Probyn et al., 2010), and China (Zhang et al., 2012). This alga was unknown to science prior to the first documented bloom events and does not affect human health, but has since gained notoriety for its ability to disrupt and damage the coastal ecosystems in which they occur.

On Long Island, brown tides are a Suffolk County phenomenon as bloom densities have never been recorded across the County border. In eastern Suffolk, blooms occurred in and across the entire Peconic Estuary during six summers from 1985-1995 but more frequently on Shelter Island through 1998 (Gobler et al., 2005). Since this time, blooms have occurred sporadically

within the entire Suffolk County region of the NYS South Shore Estuary Reserve from Babylon to Southampton. Brown tides have been sporadic in Great South Bay occurring on average every other year since 1985. The most intense and frequent brown tides have occurred in the region between the Moriches and Shinnecock Inlets. These blooms experience their peak intensity in Quantuck Bay, located between Moriches and Shinnecock, and decline in density in regions closer to the ocean inlets. Blooms in this region have occurred in 29 of the past 31 years and have occurred annually since 2007 (Fig. 2). Kang et al. (2016) recently demonstrated that the most closely related brown tide pelagophyte to *Aureococcus*, *Aureoumbra lagunensis* forms resting cells that can persist in sediments for seven months and Popels and Hutchins (2002) demonstrated this alga has the ability to survive prolonged periods of darkness (over one month). Moreover, Doblin et al. (2004) described the discovery of vegetative *Aureococcus* cells in the bilge water of small boats throughout Delaware's inland bays and in the ballast water of several larger ships. These findings suggest the ability of this species to survive adverse conditions, as a resting cell allowing it to reform blooms annually in Suffolk County estuaries.

As part of the Suffolk County's ongoing response to the initial outbreaks of "brown tide", the Brown Tide Comprehensive Assessment and Management Program (BTCAMP) was initiated in 1988. Conducted by SCDHS and supported by three consulting firms and numerous researchers, the BTCAMP focused on plans for research, monitoring, data analysis, and modeling. This initial effort was highly focused on the Peconic Estuary System where the near immediate collapse of the bay scallop fishery was of large and significant concern. BTCAMP developed two major objectives, specifically to 1. Support research into the causes and impacts, identify appropriate remedial actions for brown tides and, 2. Investigate more conventional water quality problems so that corrective actions could be identified. During the subsequent decade, a series of research projects were supported by Suffolk County and NYSG to address these objectives.

Impacts

During the initial *Aureococcus* blooms in eastern and southern Long Island bays (1985-1987), high cell concentrations ($> 10^6$ ml^{-1}) substantially decreased light penetration, which caused a large-scale die-off of seagrass beds of *Zostera marina*, a critical habitat for scallops, larval fish, and other species (Cosper et al., 1987). The blooms caused mass mortality and recruitment failure in populations of bay scallops (*Argopecten irradians*), which resulted in the

collapse of the multi-million dollar scallop industry in eastern Long Island (Bricelj and Kuenstner, 1989). The *Aureococcus* blooms also negatively impacted populations of clams (*Mercenaria mercenaria*) in Great South Bay, which was formerly the most valuable fishery in the state of New York. While overharvesting was identified as the cause of a sharp reduction in clam populations during the early 1980s, chronic *Aureococcus* blooms during the late 1980s and early 1990s contributed toward the 99% reduction in landings that occurred during this period (Kraeuter et al., 2008).

Subsequent research has demonstrated that *Aureococcus* adversely impacts the growth, survival, and reproduction of many algal grazers, including larval, juvenile and adult hard clams (*M. mercenaria*; Bricelj et al., 2001; Greenfield and Lonsdale, 2002; Padilla et al., 2006; Bricelj and MacQuarrie, 2007; Newell et al., 2009), larval and adult bay scallops (*A. irradians*; Bricelj and Kuenstner, 1989; Gallagher et al., 1989), adult and juvenile blue mussels (*Mytilus edulis*; Bricelj and Kuenstner, 1989; Bricelj et al., 2001), and micro- and mesozooplankton (Lonsdale et al., 1996; Caron et al., 2004; Deonarine et al., 2006; Smith et al., 2008). Beyond the direct negative effects on fisheries, negative effects on zooplankton are likely to translate to decreased finfish productivity (Chasar et al., 2005). Although a toxin has never been isolated from *Aureococcus*, there is evidence for toxic activity, most likely within the extracellular polysaccharide (EPS) sheath surrounding the cells (Sieburth et al., 1988). This putative toxin deters feeding in bivalves by stimulating contractions of the branchial muscles and interfering with lateral and ventral groove ciliary beating (Gainey and Shumway, 1991; Robbins et al., 2010). In addition, it has also been argued that *Aureococcus* may be a poor source of nutrition for zooplankton (Lonsdale et al., 1996; Caron et al., 2004) and some bivalve larvae (Padilla et al., 2006; Bricelj and MacQuarrie, 2007). The loss of toxicity of one strain of *Aureococcus* (strain CCMP 1784) since its isolation permitted (Bricelj et al., 2004) the comparison of toxic and non-toxic clones of *Aureococcus* and unambiguously demonstrated that the detrimental effects of *Aureococcus* on bivalve growth were mainly attributable to cell toxicity, rather than cell size, high cell density, nutritional deficiency, or poor filtration retention of the small cells.

Nutritional and physical factors promoting brown tides

Although harmful algal blooms in coastal waters have been commonly attributed to nutrient loading, the role of nutrients in the occurrence of brown tides appears to be more complex than a simple nutrient stimulation of brown tide growth (Sunda et al., 2006). An examination of spatial and temporal patterns of concentrations of *Aureococcus* cells and inorganic nutrients indicated that blooms occurred when inorganic nutrient levels were low (Cosper et al., 1989; LaRoche et al., 1997, Fig. 3). Moreover, nitrate additions during laboratory, mesocosm, and bottle experiments in the field have consistently yielded reduced *Aureococcus* growth rates relative to those of competing algae (Keller and Rice, 1989; Gobler and Sanudo-Wilhelmy, 2001a; Gobler et al., 2002; Gobler et al., 2004a; Gobler et al., 2004b; Taylor et al., 2006). The nitrogen-uptake characteristics of *Aureococcus* suggest that this species is well adapted to low nutrient environments (Lomas et al., 1996). The ability of *Aureococcus* to attain high biomass levels when inorganic nutrient concentrations are low may be partly linked to its ability to utilize organic forms of N and P. Cultures (both axenic and non-axenic) and field populations of *Aureococcus* have been shown to obtain nitrogen from a variety of organic compounds, including urea, amino acids, proteins, chitobiose, and acetamide (Berg et al., 2002; Mulholland et al., 2002; Mulholland et al., 2009a), and to assimilate organic carbon from glucose and amino acids (Dzurica et al., 1989; Mulholland et al., 2002; Mulholland et al., 2009a). Genomic analyses of this species have revealed *Aureococcus* contains more genes involved in the transport and metabolism of organic carbon and nitrogen compounds (Gobler et al., 2011) and that *Aureococcus* strongly expresses genes associated with the use of dissolved organic nitrogen (DON) compounds such as urea and purines during blooms (Wurch et al., 2014). Experimental additions of dissolved organic matter (DOM; glucose and amino acids) have enhanced the growth and relative abundance of *Aureococcus* during field experiments (Gobler and Sanudo-Wilhelmy, 2001a). Dissolved organic nitrogen (DON) and carbon (DOC) are often elevated during bloom initiation (LaRoche et al., 1997; Gobler et al., 2002; Gobler et al., 2004a) and decreases in DOC and DON concentrations have been observed with bloom development, a pattern consistent with utilization of these substances by *Aureococcus* (LaRoche et al., 1997; Gobler et al., 2004a; Gobler et al., 2011, Fig. 3). In Chincoteague Bay (MD), the concentrations of DON doubled from 1996 to 2004 during which time *Aureococcus* blooms began and intensified with increasing DON levels (Glibert et al., 2007). While DON provides an important

N source to blooms, heterotrophic utilization of carbon may partly circumvent the need for photosynthetic carbon fixation, giving *Aureococcus* a competitive advantage over strictly autotrophic algae under severe shading during blooms.

Blooms of *Aureococcus* often occur after 'pre-blooms' of other algae, which both draw down inorganic nutrients to low levels and cause gradual increases DON and DOC pools that are less utilizable by other phytoplankton (Gobler and Sanudo-Wilhelmy, 2001b; Gobler et al., 2011, Fig. 3). Microbial degradation processes during and following these pre-blooms can result in enhanced DOM levels, which can serve as a source of DON, dissolved organic phosphorus (DOP), and DOC to subsequent *Aureococcus* blooms (LaRoche et al., 1997; Gobler and Sanudo-Wilhelmy, 2001b; Glibert et al., 2007).

Since these blooms usually occur in shallow bays, benthic fluxes are important sources of inorganic and organic nutrients (Lomas et al., 2004). In experiments, the enrichment of bloom water with benthic pore water significantly increased the absolute and relative abundance of *Aureococcus* (Taylor et al., 2006). Whether sediments serve as a net source or sink for nutrients often depends on the degree to which benthic primary producers (microalgae, seagrass, or macroalgae) utilize nutrients regenerated in sediments (MacIntyre et al., 2004). Brown-tide shading of the bottom causes a net loss of these benthic phototrophs, and this loss provides a source of nutrients and DOM via plant matter decay and recycling processes. It also increases net benthic flux of nutrients to the water column due to decreased utilization by benthic phototrophs. Both processes should promote the development of brown tide blooms (MacIntyre et al., 2004).

Clearly, organic nutrients play an important role in the occurrence of brown tides caused by *A. anophagefferens*. However, as this review points out, there are multiple co-occurring factors which also contribute to bloom initiation and maintenance, such as reduced pelagic and benthic grazing, reduced flushing, temperature, and light. Hence, it would seem that organic nutrients alone may not cause blooms per se, but rather that elevated levels of DOM is only one of many pre-requisite conditions necessary for intense blooms to occur. Interestingly, a comparison of two major Long Island estuarine systems that have supported brown tides indicates that Great South Bay has had higher levels of DOC and DON and experienced blooms more frequently than the Peconic Estuary which has had with lower DOC and DON concentrations and less frequent blooms (Gobler et al., 2005). Dissolved inorganic nitrogen

(DIN) and dissolved inorganic phosphorus (DIP) concentrations are not significantly different between these systems. Higher DOC and DON levels in Great South Bay may be due to the shallow depth (2 – 4 m) and hence stronger benthic-pelagic coupling in this system relative to the Peconic Estuary (3 – 20 m), as benthic fluxes have been identified as a critical source of DOM for brown tides (Lomas et al., 2004). These results suggest that in Great South Bay, higher levels of DOM may generally be available to support brown tides, and hence the occurrence of a bloom there may be more dependent on other factors. Although some of this higher DOM may be, in fact, caused by frequent brown tides (Gobler et al., 2004a), it is also likely that the higher DOM levels in GSB may also promote the greater frequency of blooms there (LaRoche et al., 1997).

Whether coming from sediments or (micro or macro) algae blooms occurring before brown tides, DON and DOP are the fuel on which brown tides grow. Ultimately, the source of this DON and DOP is inorganic nutrients being loaded from watersheds such as wastewater-derived nitrate in groundwater. The rate of nutrient loading in winter and spring months will control the intensity of spring micro- and macroalgal blooms that degrade in the late spring and provide to DON and DOP that support and fuel brown tides. Hence, efforts made to restrict the delivery of nitrogen and phosphorus from land to sea in Suffolk County will limit the accumulation of DON and DOP in estuaries and thus minimize the intensity of brown tides.

Collectively, therefore, the nutrient niche of brown tides appears to be ecosystems where inorganic nutrient (nitrate, phosphate) levels are low, but organic nutrient (DON, DOP) levels are elevated. These conditions, therefore, restrict the time and space within which blooms occur. Regarding space, during brown tides, *Aureococcus* is typically displaced by other phytoplankton with a greater affinity for high levels of inorganic nutrients such as *Prorocentrum* within creeks that are known to have high nutrient loading rates (SCDHS, 1976 - 2015). Inorganic and organic nutrients display seasonal cycles with nitrate levels being higher in winter and organic nutrient concentrations increasing only after initial blooms or growth of other phytoplankton occur.

Although many of the embayments which host brown tides have been traditionally viewed as nitrogen-limited (Ryther and Dunstan, 1971; Kaufman et al., 1983), there is some evidence that phosphorus can also influence the occurrence of brown tides. A multi-variate regression model by Gobler and Sanudo-Wilhelmy (2001b) found that dissolved phosphate

levels were inversely correlated with brown tide abundances during a two-year study of a bloom-prone estuary, potentially indicating rapid P uptake by *Aureococcus* during blooms. Research in eastern Great South Bay has shown that net growth rates of *Aureococcus* can be stimulated by experimental P additions during bloom initiation, but not during or after the bloom peaks (Gobler et al., 2002; Gobler et al., 2004b). More recent studies in Great South Bay and Quantuck Bay have repeated these results and have shown that *Aureococcus* populations express genes associated with phosphorus limitation during blooms (Wurch et al., 2014). Although the inorganic N:P ratios in most brown tide prone estuaries are typically below the Redfield ratio of 16:1, the total dissolved N:P (organic plus inorganic) ratios in eastern Great South Bay can be substantially above it (SCDHS, 1985 - 2002). These ratios suggest that autotrophic algae using strictly inorganic sources might be N-limited, but mixotrophs, such as *Aureococcus*, which may access both organic and inorganic nutrients, could at times be P-limited. In Suffolk County, a substantial increase in the volumes of wastewater discharged to groundwater has enriched the concentrations of N discharged from groundwater into estuaries by more than 200% since 1980 while P levels have largely remained unchanged (SCCWRMP, 2015). This unbalanced delivery of nutrients is likely a factor that has altered this once strongly N-limited system (Ryther and Dunstan, 1971; Kaufman et al., 1983) into one that today can drive dense blooms of *A. anophagefferens* into P deficiency. Further, this suggests a dual nutrient mitigation strategy that restricts the delivery of both N and P into estuaries may be most effective for mitigating blooms of this HAB (Conley et al., 2009).

Physical factors (salinity, temperature, light, and water residence times) influence brown tides. *Aureococcus* blooms are associated with high chlorophyll *a* (chl *a*) concentrations (30-60 µg L^{-1}) and associated severe light attenuation. *Aureococcus* can maintain near-maximum growth rates (at 20°C) at a light intensity of 50µmol photons m^{-2} s^{-1} (~ 2% of noon solar irradiance; MacIntyre et al., 2004). In addition, the shallow (<50 cm) photic zone observed during *Aureococcus* blooms combined with wind mixing creates large fluctuations in the light field experienced by cells, a condition that significantly increases the short-term carbon fixation and photosynthetic efficiency in *A. anophagefferens* compared to constant irradiance (Milligan and Cosper, 1997). *Aureococcus* possesses more genes involved in the harvesting of light than the competing phytoplankton (Gobler et al., 2011) that likely provide it the ability to grow

maximally at low light intensities may contribute to its ability to outcompete other phytoplankton during blooms (Milligan and Cosper, 1997; MacIntyre et al., 2004).

Aureococcus grows optimally at salinities ≥ 24 (Cosper et al., 1989) and the salinities of most bays that experience brown tide blooms rarely fall below 24 (SCDHS, 1976 - 2015). Aureococcus blooms under a wide range of temperatures (0 – 25°C) and shows optimal growth at ~20°C (Cosper et al., 1989; Gobler et al., 2002). Blooms typically initiate during May or June as temperatures approach 15 - 20°C, and decline when temperatures exceed 25°C during July or August (Nuzzi and Waters, 2004). Aureococcus blooms can also re-emerge and even initiate during the fall when temperatures fall below 20°C, and can persist during winter when temperatures reach freezing (Gobler et al., 2002; Nuzzi and Waters, 2004).

Bays that host frequent Aureococcus blooms are known to have long residence times (up to two months), which are associated with low freshwater inputs and low rates of mixing with coastal seawater (Hardy, 1976; Wilson, 1995). While long residence times clearly permit the accumulation of high algal biomass, they would not necessarily give a competitive advantage to one algal species over another. However, the high biomass and low inputs of external nutrients in these systems create large algal nutrient demand:supply ratios, and hence low concentrations of available inorganic nutrients. Moreover, intense nutrient recycling within these shallow, long-residence time systems promotes the accumulation of DOM (Lomas et al., 2004). The resulting low concentrations of available inorganic nutrients and low ratios of inorganic to organic pools of N and P likely favor the net population growth of brown tide species.

Sources of cell mortality

Because of its small size, Aureococcus should be efficiently grazed by micro- and nanozooplankton, whose rapid growth rates should prevent blooms from developing (Sherr and Sherr, 2002). However, grazing rates on the brown tide have been found to be low due to unpalatability, toxicity, or physical interference with grazing, a factor that facilitates the initiation and persistence of brown tides (Gobler et al., 2002; Sunda et al., 2006; Smayda, 2008). While some zooplankton are able to actively graze during Aureococcus blooms, specific grazing rates on Aureococcus can be considerably lower (e.g. 70% lower; Gobler et al., 2002) than

grazing rates on competing algae and lower than concurrent cellular growth rates for *Aureococcus* (Gobler et al., 2002; Caron et al., 2004; Gobler et al., 2004b). Reduced grazing may be partly facilitated by the EPS layer of *Aureococcus*. Alternatively, *Aureococcus* possesses a suite of unique genes involved in the production of secondary metabolites that may encode for the synthesis and metabolism of toxins or other compounds that deter grazing (Gobler et al., 2011).

Aureococcus blooms have waned in their intensity and frequency in some U.S. estuaries such as the Peconic Estuary, NY. This system has not experienced changes in its chemical or physical characteristics relative to periods when intense blooms first occurred (Nuzzi and Waters, 2004). Grazing communities can evolve resistance to harmful algae over time via natural selection processes (Hairston et al., 2001). The selective adverse impact of blooms on more sensitive grazer species and more sensitive phenotypes within populations (Caron et al., 2004) should eventually lead to the establishment of grazer communities that are better adapted to co-exist with and consume *Aureococcus*. Recent studies have shown that some NY estuaries, which formerly hosted zooplankton communities that consumed *Aureococcus* at low rates during massive blooms, have shifted to communities that actively graze *Aureococcus* at rates similar to those for other algal species (Deonarine et al., 2006). Similarly, although *Aureococcus* inhibits grazing by mollusks (Bricelj and Kuenstner, 1989; Gallagher et al., 1989; Bricelj et al., 2001), the filter feeding slipper limpet, *Crepidula fornicata*, was able to avidly graze multiple toxic strains and natural bloom samples of *Aureococcus* while other mollusks (e.g. *Mercenaria mercenaria*) could not (Harke et al., 2011). This limpet is now present at high abundance in estuaries that no longer experience brown tide blooms (e.g., the Peconic Estuary; Harke et al., 2011). Thus, the recent abatement of brown tides in some Long Island bays may, at least partly, reflect a shift in the grazing communities toward populations more capable of feeding on *Aureococcus*.

The occurrence of brown tides in Long Island bays may be partly related to the dramatic decline in shellfish populations, initially from over-harvesting and later from mortality caused by persistent brown tide blooms (Kraeuter et al., 2008). In support of this hypothesis, mesocosms containing high but environmentally realistic concentrations of hard clams (*M. mercenaria*) maintained non-bloom densities of *Aureococcus* ($\sim 10^3$ ml^{-1}), while identical mesocosms with few or no clams developed dense brown tide blooms ($> 10^5$ ml^{-1}) (Cerrato et al., 2004).

Aureococcus inhibits *M. mercenaria* filtration rates at densities above 4 x 10^4 cells ml^{-1} (Bricelj et al., 2001). Thus, there may be a threshold effect whereby clams consume *Aureococcus* at low cell densities, thereby retarding bloom development, but their grazing is inhibited at high cell concentrations, leading to the formation of dense brown tides (Bricelj et al., 2001).

Viruses likely contribute to the collapse of brown tides. Electron micrographs of blooms in Suffolk County have revealed the presence of intracellular, icosahedral virus-like particles in *Aureococcus* cells (Gastrich et al., 2002) and *Aureococcus*-specific viruses have been isolated from Suffolk County waters (Milligan and Cosper, 1994; Gobler et al., 2007b). Viral densities during *Aureococcus* blooms are generally elevated compared to most estuarine environments (Gobler et al., 2004b) and field studies have observed a high percentage (~40%) of virally infected *Aureococcus* cells at the end of blooms, suggesting that viruses may be an important source of mortality during bloom termination (Gastrich et al., 2004).

Allelopathic interactions with micro- and macroalgae likely effect brown tides. Kang (2016) recently discovered that *Aureococcus* secretes allelochemicals that inhibit the growth of 10 species of phytoplankton and whole phytoplankton communities during blooms and concluded that these chemicals likely allow brown tides to intensify and persist, but not initiate as their effectiveness is dose-dependent. *Aureococcus* may also be effected by allelopathy as the microalga, *Alexandrium fundyense*, excretes allelochemicals that inhibit the growth of *Aureococcus* (Hattenrath-Lehmann and Gobler, 2011) and the green macroalgae, *Ulva*, inhibited the growth of *Aureococcus* in field and lab experiments and *Aureococcus* blooms do not occur in shallow tidal creeks dominated by *Ulva* (Tang and Gobler, 2011). These allelopathic interactions may limit the times and spaces within which brown tides occur.

Aureococcus is the first eukaryotic HAB to have its entire genome sequenced and published (Gobler et al., 2011). The study generated metaproteomes of plankton assemblages present during brown tide blooms and identified six algal species that compete directly with *A. anophagefferens* in its natural environment. Gene sets involved in nutrient acquisition, light harvesting, and potential grazing defense were then compared among the seven species. Consistent with some of the physiological characteristics discussed in this review, a second finding consistent with prior studies was the enrichment in putative genes involved in the production of secondary metabolites that may encode for the synthesis and metabolism of toxins or other compounds that deter grazing (Gobler et al., 2011). Finally, the *Aureococcus* genome

has more genes encoding for selenium-, copper-, and nickel-containing proteins than competing phytoplankton, a new finding for this alga. Recent studies have linked anthropogenic nutrient loading to HABs (Heisler et al., 2008). Given the large number of genes involved in adaptation to low light, assimilation of organic compounds, and synthesis of metalloproteins, anthropogenic activities that increase turbidity and concentrations of dissolved organic matter and trace metals in coastal ecosystems were hypothesized to promote brown tides and perhaps other HABs.

RUST TIDES CAUSED BY THE DINOFLAGELLATE, *COCHLODINIUM POLYKRIKOIDES*
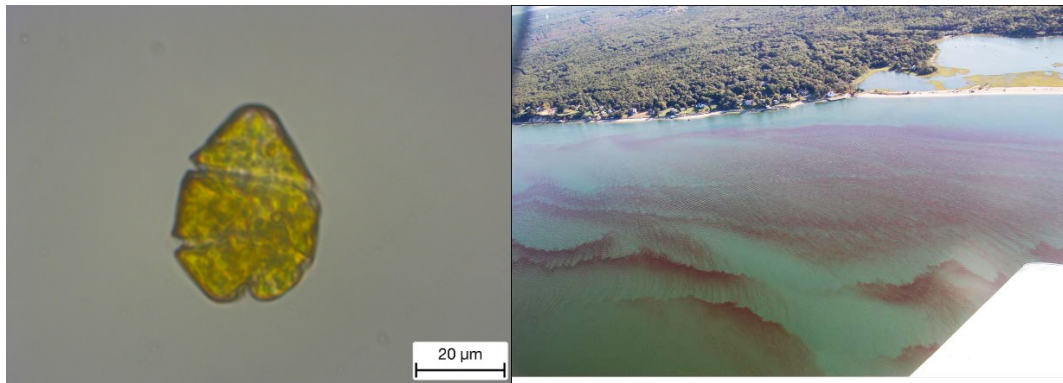
A light microscopic image of a live, single cell of *Cochlodinium polykrikoides* (Photo Credit: Theresa Hattenrath-Lehmann) and an aerial image of a *Cochlodinium polykrikoides* bloom (Photo Credit: Auxiliary Coast Guard).

Dinoflagellates of the genus *Cochlodinium* were first identified in 1895 by Schütt (1895) and have been forming harmful algal blooms in the coastal waters of Southeast Asia and North America for many decades. The past two decades have seen *Cochlodinium* blooms expanded in their geographic distribution across Asia, Europe, and North America but also across Long Island. More than 40 species of *Cochlodinium* have been described, although the HAB-forming species on Long Island is *Cochlodinium polykrikoides* of the North American – Malaysian ribotype (Iwataki et al., 2007; Iwataki et al., 2008). *Cochlodinium* cells are large (~40 µm) and these athecate (lacking a theca) dinoflagellates commonly form chains of 2 to 8 cells during blooms. In Suffolk County, *Cochlodinium* blooms are generally characterized by spatially large (10's to 1000's of meters) and dense (>1,000 cells ml^{-1}) cell aggregates that are heterogeneous in their vertical and horizontal distributions and are dark brown in color leading to the common name 'rust tide'. *Cochlodinium* blooms are ichthyotoxic (fish-killing) and can also kill many other marine organisms, although the compound(s) responsible for these impacts have yet to be

identified and bloom-associated toxins are not known to affect human health. Below is a review of the current state of knowledge regarding distribution, ecophysiology, life history, food web interactions, and mitigation of HABs formed by *Cochlodinium*.

Emergence and Distribution of *Cochlodinium* blooms in Suffolk County

Many investigators have noted the global expansion of HABs during the past several decades with regard to their impacts, duration, intensity, and/or distribution (Anderson, 1989; Hallegraeff, 1993; Smayda, 1997a; Glibert et al., 2005). While *Cochlodinium* blooms had been identified in New Jersey waters in the 1960s (Silva, 1967) and in Narragansett Bay in the 1980s (Tomas and Smayda, 2008), blooms had been unknown to Suffolk County prior to 2002. Since that time they have become annual occurrences. Given the large size of *Cochlodinium* cells and patchy, highly discolored nature of blooms, HABs caused by *Cochlodinium* are fairly easy to identify on the micro- and macroscopic level, particularly in light of the robust monitoring programs for HAB in Suffolk County. Since this organism was present regionally (NJ, RI) prior to 2002, it is hypothesized that the organism was also present in Suffolk County and that environmental factors such as nutrient loading and increased water temperatures set the stage for large scale blooms to occur.

In 2002, Nuzzi described the first ever *Cochlodinium* bloom in Suffolk County, in West Neck Bay on Shelter Island. Ironically, this is the embayment that hosted brown tides more frequently than any other estuary on Long Island during the 1980s and 1990s (Gobler and Sanudo-Wilhelmy, 2001a). While there were no official reports of *Cochlodinium* blooms in 2003, large and wide-spread blooms were observed in 2004 and 2005 (C. Gobler, pers. comm.) and in 2006, the Gobler Lab and SCDHS commenced their collaborative monitoring of these 'rust tides'. Gobler et al. (2008) provided the first comprehensive descriptions of *Cochlodinium* blooms in Suffolk County, describing blooms in the Peconic Estuary and Shinnecock Bay with dense bloom patches that achieved cell densities up to 10^5 mL^{-1} and chlorophyll *a* levels exceeding 100 µg L^{-1} and more moderate background bloom densities of 10^3 - 10^4 cells mL^{-1}. *Cochlodinium* blooms tend to be visible beginning in mid-to-late summer (July or August) and persist through October (Gobler et al., 2008; Gobler et al., 2012; Koch et al., 2014). Within Shinnecock Bay, they are typically found in the northern extent of the eastern basin which is near the Shinnecock Canal. In the Peconic Estuary, they are often densest within the western extent (Flanders Bay,

Great Peconic Bay) but have been found throughout the Peconic Estuary as far east as Accabonac Harbor in East Hampton, and Orient Harbor in Orient. Blooms can be near monospecific in the open waters of these estuaries, but are typically mixed with other dinoflagellates within more eutrophic tributaries (Gobler et al., 2008; Gobler et al., 2012; Koch et al., 2014). While *Cochlodinium* blooms have occurred every year, there have been inter-annual differences in their intensity and distribution which have been challenging to quantify due to the heterogeneous nature of these HABs. In some years, blooms have been first noted in tributaries of the western Peconic Estuary and eastern Shinnecock Bay (Gobler et al., 2008; Gobler et al., 2012). In other years, they have first appeared in East Hampton Town (2014 – 2016). It is possible that cysts play a key role in controlling the year-to-year distribution of these blooms (Hattenrath-Lehmann et al., 2016, see below).

There have been three 'aberrant' *Cochlodinium* blooms in Suffolk County this decade, meaning blooms outside of the Peconic Estuary and eastern Shinnecock Bay. In 2011, the first and only known *Cochlodinium* bloom occurred in Great South Bay (Gobler, 2012). In 2015, *Cochlodinium* blooms occurred in western Shinnecock Bay (Gobler, pers. obs.) and in 2016, an extensive bloom occurred in Port Jefferson Harbor (Hattenrath-Lehmann, pers. obs). In 2012 and 2016, warm summer temperatures were observed in Long Island Sound and there are anecdotal reports of *Cochlodinium* bloom patches by the Connecticut's Department of Energy and Environmental Protection (CTDEEP). This suggests that, perhaps in warmer years, other north shore harbors in Suffolk County may also be vulnerable to *Cochlodinium* blooms.

A final point regarding the distribution of *Cochlodinium* is its propensity to form dense surface aggregations by day (Gobler et al., 2008; Gobler et al., 2012; Koch et al., 2014). Observational and experimental work in the Gobler lab has established that these surface patches break up late in the day and sometimes do not return to surface water until later the next morning. Vertical migration is likely an adaptive strategy to multiple environmental conditions including optimization of light and nutrients, minimization of grazing and possibly shear-stress (Sullivan et al., 2003).

Ecophysiology – Nutrients and physical factors

Cochlodinium is eurythermal and euryhaline, blooming over a broad salinity (20- 30) and temperature (15-28°C) range (Gobler et al., 2008; Gobler et al., 2012, SCDHS, 2004 – 2015;

Griffith and Gobler, 2016). There are indications that blooms are denser, more expansive, and occur earlier during warm summers such as 2012, 2013, and 2016.

Cochlodinium seems to display a flexible nutrient acquisition strategy. Mulholland et al. (2009b) also found that *C. polykrikoides* utilizes a variety of nutrients in the Chesapeake Bay, including dissolved organic N, P, and C, to meet its nutritional requirements. Gobler et al. (2012) found that bloom populations in the Peconic Estuary and Shinnecock Bay displayed the greatest N uptake rates for the compounds in the greatest abundance (nitrate, ammonium, urea, or glutamic acid). Several studies have linked *Cochlodinium* blooms to eutrophication or demonstrated that *Cochlodinium* readily accesses nutrients typically associated with neritic or eutrophied environments. For example, both Imai et al. (2006) and Verity (2010) reported an increase in *C. polykrikoides* as part of a long-term trend of increasing HAB organisms in the Seto Inland Sea and a Georgia (USA) estuary, respectively. Imai et al. (2006) further noted that the low N and P requirements to reach a "significant" bloom could result in coastal blooms of *Cochlodinium* with only minor nutrient enrichment. An isolate of *Cochlodinium* from the Peconic Estuary was shown to grow faster on glutamic acid (~0.5 d^{-1}) as a sole N source than nitrate, ammonium, and urea all of which yielded similar growth rates (~0.4 d^{-1}; Gobler et al., 2012). Several authors (e.g. Viquez and Hargraves, 1995; Lee and Lee, 2006; Anton et al., 2008; Mulholland et al., 2009b; Morse et al., 2011) have also associated increases in *Cochlodinium* with rainfall, attributed to changes in both the physical environment and the influx of discharge-associated nutrients. Consistent with these observations, experimental enrichment with N concentrations has been shown to significantly enhance net growth rates and photosynthesis of *Cochlodinium* during blooms in NY coastal waters (Gobler et al., 2012).

A recent review (Hansen, 2011) places *Cochlodinium*, along with several other red tide organisms, in a group ("Type 1") that generally utilizes mixotrophy to moderately enhance growth at low irradiance levels, while several reviews (Stoecker et al., 2006; Burkholder et al., 2008) suggest phagotrophy and osmotrophy are common in HAB organisms, particularly for those forming this alga. While research on the Japanese-Korean ribotypes of *Cochlodinium* has demonstrated mixotrophic or phagotrophic feeding on multiple types of planktonic prey (Larsen and Sournia, 1991; Jeong et al., 2004; Jeong et al., 2005; Du Yoo et al., 2009), this has not been explored in the US.

In summary, *Cochlodinium* exhibits wide flexibility in its nutrient acquisition strategies. While its nutrient kinetics are generally consistent with pelagic origin, this genus readily utilizes organic nitrogen and phosphorus compounds, and appears to do well in moderately eutrophied environments, particularly when DIN and DIP are low. While its ability to phagotrophically consume prey further extends its nutritional flexibility, the relative importance of all of these nutrient acquisition strategies during bloom events has not been well characterized.

Cyst formation

The life history of *Cochlodinium* has been a subject of recent study, but is still not well characterized. The recurrence of annual blooms following initial colonization of an area by *Cochlodinium* strongly implicates seed populations in the establishment of this organism in new habitats. In 2012, (Tang and Gobler, 2012) discovered the production of resting cysts by *Cochlodinium* in laboratory cultures isolated from Suffolk County. Resting cyst germination occurred up to 3 months after cyst formation and 2–40% of resting cysts were successfully germinated in cultures maintained at 18–21°C. In 2016, Hattenrath-Lehmann et al. (2016) developed a fluorescent in situ hybridization (FISH) assay using oligonucleotide probes specific for the large subunit (LSU) rDNA of *Cochlodinium* to quantify *Cochlodinium* cysts in Suffolk County estuaries. The study found that elevated densities of *Cochlodinium* cysts in Shinnecock Bay (>100 cm^{-3}) during spring of a given year were spatially consistent with regions of dense blooms the prior summer. The identity of cysts in sediments was confirmed via independent amplification of *C. polykrikoides* rDNA. The discovery that *Cochlodinium* can make resting cysts provides a mechanism to account for the recurrence of annual blooms Suffolk County as well as their expansion across estuaries since 2011.

Food web interactions and toxicity

Cochlodinium has been implicated in kills of wild and impounded fish around the globe (Onoue et al., 1985; Yuki and Yoshimatsu, 1989; Guzman et al., 1990; Qi et al., 1993; Kim et al., 1999; Whyte et al., 2001; Garate-Lizarraga et al., 2004). Studies have also demonstrated that this alga causes rapid mortality in copepods (Jiang et al., 2009; Jiang et al., 2010a), other phytoplankton (Tang and Gobler, 2010), shellfish larvae (Ho and Zubkoff, 1979; Tang and Gobler, 2009b), bivalves (Ho and Zubkoff, 1979; Gobler et al., 2008; Tang and Gobler, 2009a;

Li et al., 2012), and cultured fish (Gobler et al., 2008; Dorantes-Aranda et al., 2009; Tang and Gobler, 2009a). Long Island bivalves shown to be sensitive to *Cochlodinium* in a laboratory setting include bay scallops, hard clams, and Eastern oysters (Gobler et al., 2008; Tang and Gobler, 2009a, 2009b; Li et al., 2012). In Suffolk County, pound net fishermen have reported nets filled with dead menhaden during *Cochlodinium* blooms (Ed Warner, John Semlar, Southampton Town Trustees, pers. comm.). At the Stony Brook- Southampton Marine Science Center, hundreds of fish and shellfish kept in flowing seawater tables have perished through the years (2006 – 2016) as *Cochlodinium* bloom water has been inadvertently circulated through holding sea tables. In 2012, a fish kill occurred in Casses Creek along the north shore of Flanders Bay Aquebogue. In 2005, a massive kill of soft shell clams occurred during a dense *Cochlodinium* bloom in Flanders Bay. The East Hampton Town shellfish hatchery and baymen in Southampton Town have reported that bay scallop populations that were dense in early summer have experienced severe mortality during *Cochlodinium* blooms (EHTSH, 2012). In some cases, precise cause and effect of these events require more precise confirmation.

Despite the severe and broad nature of the toxicity of *Cochlodinium,* the precise compounds responsible for this toxicity have yet to be confirmed. Some studies have presented data suggesting the toxic principles are similar to reactive oxygen species (ROS; i.e. superoxide anions, hydrogen peroxide, and many other compounds) and are produced by *C. polykrikoides* cells and may be one of the factors responsible for lipid peroxidation and subsequent fish kills caused by this species (Kim et al., 1999; Tang and Gobler, 2009a, 2009b, 2010). Given the gradual accumulation of polysaccharides in *C. polykrikoides* cultures (Kim et al., 2002) and cells (Gobler et al., 2008), it has been suggested that there may be multiple, biologically active metabolites secreted by *C. polykrikoides* such as cytotoxic agents and mucus substances such as polysaccharides that are responsible for fish kills caused by this species (Kim et al., 2002). Research since that time has suggested that *Cochlodinium* toxins are extracellular, highly reactive, and labile compounds that are produced continuously by actively growing *C. polykrikoides* cultures, similar to, but not necessarily being, ROS (Griffith and Gobler, 2016). Some harmful algae display a wide range of toxicity or noxious effects among culture strains and field populations (Burkholder and Glibert, 2009) and strains of *Cochlodinium* isolated from NY estuaries range from highly lethal to non-toxic among strains of different origins (Y.Z. Tang and C. Gobler, unpubl. data). Regardless, given that the smallest individuals and larvae of fish and

shellfish as well as plankton are most vulnerable to the toxicity of *Cochlodinium*, the ecosystem-level impacts of these blooms almost certainly extend well-beyond obvious fish kills observed during bloom events.

The ability of *Cochlodinium* blooms to suppress survival and grazing by copepods (Shin et al., 2003; Jiang et al., 2009; Jiang et al., 2010a, 2010b), protozoan zooplankton (Jeong et al., 1999), bivalve larvae (Ho and Zubkoff, 1979; Tang and Gobler, 2009b), adult bivalves (Gobler et al., 2008; Tang and Gobler, 2009a), and fish (Gobler et al., 2008; Dorantes-Aranda et al., 2009; Tang and Gobler, 2009a) indicates that *Cochlodinium* blooms are partly promoted by an absence of benthic and pelagic grazing control. Since the ability of *C. polykrikoides* to inhibit many of these grazers is density dependent, with maximal suppression occurring at densities exceeding 10^3 cells ml^{-1} (Gobler et al., 2008; Jiang et al., 2009; Tang and Gobler, 2009a, 2009b; Jiang et al., 2010a, 2010b), it is likely that grazing suppression contributes to bloom maintenance but not necessarily bloom initiation.

In addition to suppressing grazers, *Cochlodinium* blooms and strains isolated from Suffolk County have exhibited strong allelopathic effects on natural communities and ten species of cultured phytoplankton, causing complete lysis of competing algae in 24 h at *C. polykrikoides* cell densities exceeding 10^3 cells ml^{-1} (Tang and Gobler, 2010). The allelopathic effects of *C. polykrikoides* are dependent on the relative and absolute cell abundance of *C. polykrikoides* and target algal species as well as exposure time (Tang and Gobler, 2010). Since *C. polykrikoides* is generally slow-growing (μ = 0.4 d^{-1};Kim et al., 2001; Lee et al., 2001; Gobler et al., 2012) compared to other diatoms and dinoflagellates (Smayda, 1997a), it must rely on other mechanisms, such as allelopathy and grazing inhibition to form mono-specific blooms. Since *C. polykrikoides* causes dramatic mortality in planktonic grazers (as discussed above), allelopathic effects on competitors together with grazing deterrence could promote *C. polykrikoides* blooms through positive feedback (Sunda et al., 2006) whereby higher *C. polykrikoides* cell densities yield fewer competitors and predators which in turn facilitates higher cell densities. Since the allelopathic effects of *C. polykrikoides* on co-occurring algae depend on absolute and relative cell abundances, allelopathy is more likely to contribute toward bloom maintenance when cell densities are high, than bloom initiation when cell densities are low.

BLOOMS THREATENING PUBLIC HEALTH

HABs FORMED BY THE PSP-PRODUCING MARINE DINOFLAGELLATE, *ALEXANDRIUM FUNDYENSE*

A lugol's preserved sample of *Alexandrium fundyense* (Photo credit: Theresa Hattenrath-Lehmann)

Blooms of the dinoflagellate *Alexandrium* are common to coastal regions around the world and are particularly harmful because they produce saxitoxins, the causative agent of the human health syndrome, paralytic shellfish poisoning (PSP; Anderson, 1994; Van Dolah, 2000; Glibert et al., 2005; Anderson et al., 2012a). Saxitoxins are a suite of over 20 neurotoxins, each with its own potency, and placed into three groups: the carbamate toxins (the most potent), decarbamoyl toxins and the N-sulfocarbamoyl toxins (least potent; Oshima, 1995). These toxins block sodium channels, acting on the peripheral nervous system with a rapid onset (~30 minutes) causing numbness and tingling of the mouth and extremities, loss of motor control, and in severe cases can cause death due to respiratory failure in humans who consume saxitoxin-contaminated shellfish (Anderson, 1994; Van Dolah, 2000). Paralytic shellfish poisoning is thought to be the most widespread and severe poisoning syndrome (Anderson et al., 2012a) with occurrences on virtually every continent (except Antarctica) and is thus closely regulated by environmental managers, with the United States Food and Drug Administration (USFDA) imposing a regulatory closure limit of 80µg of saxitoxin eq. $100g^{-1}$ shellfish tissue (USFDA, 2011). Perhaps one of the most dangerous aspects of these *Alexandrium* blooms is that they do not discolor the water like the *Cochlodinium* and brown tide blooms discussed above. Therefore, a clear and seemingly innocuous body of water can host an *Alexandrium* bloom and toxic shellfish without showing signs of what we typically think of as a 'red tide'.

Alexandrium is a globally distributed genus, encompassing more than 30 species, half of which produce toxins (Anderson et al., 2012a). The *Alexandrium tamarense* species complex (Balech, 1985; Lilly et al., 2007), composed of the morphospecies, *A. tamarense*, *A. fundyense* and *A. catenella*, is a widely distributed group with a complicated taxonomic history that has undergone a recent formal revision (John et al., 2014). Members of the *A. tamarense* species complex differ morphologically based on the presence/absence of a ventral pore and their ability to form chains (Balech, 1985). Scholin et al. (1994) described this species complex as five geographically distinct ribotypes based on a fragment of LSU rDNA. This geographic designation was reevaluated by Lilly et al. (2007) and was changed to a group numbering system based on phylogenetically well-supported clades as new rDNA evidence indicated that these ribotypes expanded beyond their originally proposed geographic boundaries. More recently, John et al. (2014) proposed a formal revision of the *Alexandrium tamarense* species complex, designating each group defined by Lilly et al. (2007) into five distinct species (*A. fundyense, A. mediterraneum, A. tamarense, A. pacificum and A. australiense*).

Alexandrium fundyense is the species responsible for toxic PSP blooms along the east and west coasts of the United States. *A. fundyense* is 30-38μm in size (Steidinger, In press), tolerant of a wide range of salinities (15-40; Bill et al., 2016) and is a slow growing dinoflagellate with reported growth rates varying from 0.19 to 1.0 d^{-1} (Anderson et al., 2012a). *Alexandrium fundyense* has specific temperature tolerances with cyst germination stimulated by temperatures above 5°C, vegetative growth occurring from 5-21°C and optimal growth between 12 and 20°C (Watras et al., 1982; Anderson et al., 1983; Anderson, 1998; Etheridge and Roesler, 2005; Hattenrath et al., 2010; Ralston et al., 2014; Bill et al., 2016). Given these optimal temperature ranges, in Long Island, New York we find cells present in the water column between the months of March and June, with peak *Alexandrium* densities occurring in May (Schrey et al., 1984; Hattenrath et al., 2010). While some regions experience two blooms, one in the spring and fall, this is not the case in Long Island, New York, contrary to reports by Schrey et al. (1984), as no fall blooms of PSP-producing *Alexandrium fundyense* have been detected during 10 years of research using species-specific DNA probes in this region (Hattenrath et al., 2010, Hattenrath-Lehmann pers. obs.).

Alexandrium fundyense blooms are especially common along the northeast coast of the United States where they occur as both large-scale coastal events (Anderson, 1997; Anderson et al., 2005a; Anderson et al., 2005b; Townsend et al., 2005) as well as regional events in estuaries and coastal embayments (Anderson and Morel, 1979; Anderson, 1997; Hattenrath et al., 2010). The ability of these *Alexandrium* blooms to persist in these and other regions around the globe is due to their ability to produce overwintering cysts which lay dormant in the sediment until favorable conditions allow them to excyst and persist in their vegetative (actively dividing) form (Anderson et al., 1983; Nehring, 1993). The presence of *A. fundyense* on Long Island was first documented during the early 1980's from both surveys conducted within the water column (vegetative cells; Schrey et al., 1984) and in the sediments (cysts; Anderson et al., 1982). At that time, moderate densities of *A. fundyense* ($<10^3$cells L^{-1}) were found on the north shore of Long Island in Northport Bay and Mattituck Inlet (Schrey et al., 1984), with south shore concentrations as high as 10^5 cells L^{-1}; these blooms, however, were not associated with PSP events (e.g. toxic shellfish or human illness; Anderson et al., 1982; Schrey et al., 1984). The presence and distribution of *Alexandrium* in Suffolk County waters was also investigated by Nuzzi and Waters (1993) who found PSP toxins in shellfish that exceeded (190 µg $100g^{-1}$ shellfish) the federal regulatory level on one occasion (May 1986) in an area of the Peconic estuary that was not open to shellfishing (Reeves Bay). At that time, no determination could be made as to whether the populations were indigenous, or transported from New England waters (Anderson et al., 1982). However, subsequent work has demonstrated that toxin profiles from southern strains (e.g. Connecticut, New York) of *A. fundyense* are vastly different than those from the north (e.g. Canada, Maine, Massachusetts) along the east coast of North America, exhibiting a north to south PSP toxicity gradient with northern strains predominately synthesizing the more potent carbamate toxins and the southern strains containing a higher ratio of the less potent N-sulfocarbamoyl toxins (Maranda et al., 1985; Anderson et al., 1990a; Anderson et al., 1994; Bricelj and Shumway, 1998). Further work, indicates that northern populations are genetically distinct from southern populations (M. Richlen, Woods Hole Oceanographic Institute, pers. comm.), evidencing that recent transport was not likely. While there was a single observed event where shellfish toxicity exceeded the federal closure limit (Nuzzi and Waters, 1993), since 2006 several embayments across Long Island (Fig.4 Table 2) have hosted chronic *A. fundyense* blooms with shellfish bed closures due to the presence of PSP

contaminated shellfish (NYSDEC; Hattenrath et al., 2010; Hattenrath-Lehmann et al., 2013), indicating a regime shift where our coastal waters are now conducive to the growth and proliferation of HABs.

As a result of the 2006 shellfish bed closure in Northport Bay due to PSP contaminated shellfish, the Gobler Lab of Stony Brook University launched a multi-year large scale HAB monitoring effort funded by numerous agencies, including NYSDEC, Long Island Sound Study, NY Sea Grant, NOAA's MERHAB program and Suffolk County Department of Health. While this monitoring program started on a smaller scale, the effort was expanded due to the presence of *Alexandrium* in high abundances in multiple regions demonstrating the need for wider-scale monitoring on Long Island. As of December 2016 this monitoring program has resulted in the collection and subsequent analysis of ~1200 *Alexandrium* samples island-wide (Fig. 5). This large-scale effort has allowed for the identification of *Alexandrium* hotspots across Long Island, specific sites of concern are those indicated in red (>1000 cells L^{-1}) in Figure 5, 19 sites altogether, 6 of which are in the Northport Bay-Huntington Harbor complex. Northport Bay has chronically hosted high densities (>3,000 cells L^{-1}, annual maximum) of *Alexandrium* since the start of the HAB monitoring program, and had the largest recorded bloom anywhere on Long Island, reaching 1 million cells L^{-1} in 2008. This bloom led to the closure of over 7000 acres of shellfish beds with a maximal shellfish toxicity of ~1400µg of STX eq. $100g^{-1}$ shellfish (Table 2), which is 18 times higher than the federal closure limit and over the known lethal dose for human consumption (EFSA, 2009). Since 2006, Northport Bay has been closed seven times due to the presence of PSP contaminated shellfish over the federal closure limit (Fig. 4, Table 2). In addition to Northport Bay, five other sites across Long Island, including Shinnecock Bay, Sag Harbor Cove, Mattituck Inlet, James Creek and Meetinghouse Creek, have been closed due to the presence of PSP in shellfish over the federal closure limit and fall in-line with the sites indicated as *Alexandrium* hotspots (Figs. 4, 5, Table 2). Maximum shellfish toxicities and corresponding maximal observed *Alexandrium* densities during these closures can be found in Table 2. Over the course of this multiyear (2007- 2016) field investigation, *Alexandrium* was found at 65 of 79 (82%) sites sampled across Long Island (n=1198; Fig. 5) and has exceeded densities reported in the 1980's (Schrey et al., 1984). The frequency and expansion of shellfish bed closures in the last ten years taken together with presence of *Alexandrium* at multiple sites across Long Island demonstrates the continued need for monitoring in this region.

In addition to multiple shellfish bed closures, a terrapin mortality event was also documented in association with an *Alexandrium* bloom here on Long Island (Hattenrath-Lehmann et al. submitted). Diamondback terrapins (*Malaclemys terrapin*) are a threatened or endangered species in much of their range along the U.S. Atlantic and Gulf coasts (IUCN, 1996). Over an approximately three-week period from late April to mid-May 2015, hundreds of adult diamondback terrapins were found dead on the shores of Flanders Bay, Long Island, New York, USA. Concurrent with the mortality event, elevated densities of the paralytic shellfish toxin (PST)-producing dinoflagellate, *Alexandrium fundyense* ($>10^4$ cells L^{-1}) and high levels of PST in bivalves (maximal levels= 540μg STX eq. $100g^{-1}$ shellfish tissue) were observed in the Flanders Bay region, resulting in shellfish bed closures in regional tributaries. Gross and histologic postmortem examinations of terrapins revealed no physical trauma to individuals or a common, underlying disease process to explain the deaths. PST compounds ($0.2 – 12.5$ μg STX eq. $100g^{-1}$), however, were present in various *M. terrapin* tissues collected over the duration of the mortality event. In addition, high-throughput sequencing revealed that the ribbed mussel (*Geukensia demissa*, a PST vector) was present in the gastrointestinal tracks of all terrapin samples tested. In the absence of other significant findings from necropsies and pathological analyses, this provides evidence that PST in shellfish was likely high enough to cause or contribute to the mortality in these small (< 2.0 kg) animals (Hattenrath-Lehmann et al. submitted).

Factors promoting *Alexandrium* blooms

Nutrient loading has been cited as a primary factor promoting multiple HABs around the world (Anderson et al., 2002; Glibert et al., 2006; Anderson et al., 2008; Heisler et al., 2008). For *Alexandrium* specifically, culture and field studies demonstrate that the addition of inorganic nitrogen, especially reduced forms such as ammonium, enhances growth and toxicity (Leong et al., 2004; Hattenrath et al., 2010; Xu et al., 2012). A number of studies has also demonstrated that organic forms of nitrogen, such as urea, glutamine (Hattenrath et al., 2010), high molecular weight (HMW) dissolved organic matter (Legrand and Carlsson, 1998; Fagerberg et al., 2009; Loureiro et al., 2009), HMW sewage treatment plant water (Hattenrath-Lehmann et al. unpublished), and humic substances (Carlsson et al., 1998; Gagnon et al., 2005) are capable of enhancing *Alexandrium* densities, indicating that both inorganic and organic forms of nitrogen

(total nitrogen) must be considered in the prevention and mitigation of these blooms. Studies have also demonstrated increases and decreases in toxin content per cell when *Alexandrium* is P- and N-limited, respectively (Anderson et al., 1990a; Anderson et al., 1990b; Guisande et al., 2002; Lippemeier et al., 2003; Poulton et al., 2005; Van de Waal et al., 2014). In the Mediterranean, Penna et al. (2002) found that seasonal blooms of *A. taylori* are promoted by nutrient loading, primarily wastewater sources. Trainer et al. (2003) found an increase in maximal paralytic shellfish toxin concentrations with increasing human population, in Puget Sound, a eutrophied region of Washington State with poor circulation. Here in Long Island, New York, Hattenrath et al. (2010) was able to make a direct connection between the Northport Bay sewage treatment plant and toxic *Alexandrium* blooms. Specifically, this study found, through conducting nutrient amendment experiments with *Alexandrium* bloom water, that nitrogen enrichment significantly increased *Alexandrium* densities and levels of saxitoxin equivalents per cell. Further, the isotopic nitrogen signature of these blooms was consistent with those found in wastewater indicating that anthropogenic nutrient loading was promoting these *Alexandrium* blooms in Northport Bay (Hattenrath et al., 2010). Similarly, a recent study (Stinnette, 2014) demonstrated that *Alexandrium* abundances within Long Island's south shore estuaries were significantly correlated with residence times of- and total nitrogen loads to- Moriches, Shinnecock, and Quantuck Bay. Data recently obtained from the NYSDEC shows drastic changes in nitrogen, both total inorganic nitrogen (TIN) and total organic nitrogen (TON), coming from the effluent (Fig. 6) of the Northport Bay Sewage treatment plant since its upgrade in 2014. Total N and TIN dropped to about 5 kg N day^{-1} and the percentage of TON increased while the percentage of TIN contributing to the total nitrogen of the sewage treatment plant decreased (Fig. 6). While there is a reduction in nitrogen emanating from the sewage treatment plant and PSP closures have not occurred in the last three years in this region as shellfish did not exceed the federal closure limit, the peak densities of the *Alexandrium* blooms similar densities to blooms that resulted in toxic shellfish beds (Fig. 7). These bloom peaks, however, were shorter in duration even though peak bloom densities are similar. It is also possible that given there is less nitrogen coming from the sewage treatment plant that *Alexandrium* has less nitrogen to use to make their nitrogenous rich toxin, saxitoxin, and may explain the lack of closures in the area, however, we do not currently have data to support this hypothesis. Similarly, during the late 1980s in Mumford Cove, CT, the diversion of sewage

discharge reduced macroalga blooms of *Ulva lactuca* (Harlin, 1993) and PSP closures which occurred several times before this diversion never recurred (CTDEEP, 1980 - 2016). While for some regions the connection between *Alexandrium* and nutrients are hard to make, it is clear that in Long Island, New York a direct connection between eutrophication and *Alexandrium* can be made and therefore in our region *Alexandrium* may be the most responsive to reductions in nitrogen.

Temperature is another factor known to affect the growth of *Alexandrium* spp. As mentioned above, vegetative growth occurs from 5-21°C and is optimal between 12 and 20°C (Watras et al., 1982; Anderson et al., 1983; Anderson, 1998; Etheridge and Roesler, 2005; Hattenrath et al., 2010; Ralston et al., 2014; Bill et al., 2016) and has been shown to play a significant role in the onset, maintenance and demise of *Alexandrium* blooms in Long Island, New York (Hattenrath et al., 2010, unpublished). Specifically, in Long Island, New York a warmer earlier spring (March/April) has led to an earlier onset of *Alexandrium* blooms while a warmer May has led to the early termination of *Alexandrium* blooms in Northport Bay as waters exceed the threshold for which vegetative growth occurs (Hattenrath et al., 2010, unpublished). A model created by Gobler et al. (submitted) used temperature-growth relationships from multiple strains of *Alexandrium fundyense* and ~30 years of high resolution sea surface temperature records (1982-2015) to hindcast *Alexandrium* blooms in regions of the Northwest Pacific (Salish Sea) and North Sea. The model output from this study demonstrated that ocean warming in the North Atlantic since 1982 has significantly increased the potential mean growth rate and duration of bloom season for *A. fundyense* while some of these increases were less apparent yet still significant in some regions of the Pacific Northwest (Gobler et al. submitted). Observational studies in the NW Atlantic have described shifts in *A. fundyense* bloom dynamics and duration that are related to inter-annual temperature variability (Hattenrath et al., 2010; Ralston et al., 2014; Ralston et al., 2015). A compilation of *A. fundyense* bloom dynamics from the Bay of Fundy indicated that, consistent with Gobler et al.'s (submitted) findings, the first appearance of *A. fundyense* cells was, on average, three weeks earlier in the first decade of this century compared to the final 12 years of the 20th century and that maximum cell densities during blooms more than doubled over the same time period (Martin et al., 2014). Furthermore, with the exception of 1999, the start of bloom seasons predicted by Gobler et al.'s (submitted) study for the NW Atlantic was significantly correlated ($p<0.01$) with the date at which 40

Alexandrium cells L^{-1} were first observed within the Bay of Fundy from 1988 to 2010 (Martin et al., 2014). In addition, previous regional studies of the Salish Sea have identified recent decadal increases in temperature as a factor enhancing the expansion of PSP in this region (Moore et al., 2008; Moore et al., 2015) and have suggested that window of opportunity for *A. fundyense* blooms could increase by several weeks by the end of this century (Moore et al., 2011; Moore et al., 2015). These studies provide evidence that increases in temperature are creating an opportunity for *Alexandrium* to expand in some regions of the North Pacific and the North Atlantic.

Beyond temperature, pH, both eutrophication- and climate change- induced coastal ocean acidification, the former already creating pH changes in present day coastal oceans not thought to be seen by climate change induced acidification for another 100 years (Cai et al., 2011; Wallace et al., 2014), can affect the growth and toxicity of *Alexandrium* spp. Using acid additions to manipulate pH, Hwang and Lu (2000) found that a culture of *Alexandrium minutum* grew maximally at a pH of 7.5. Recently, Tatters et al. (2013) reported significantly higher growth rates and a two-fold increase in total toxicity in *Alexandrium catenella* (now *A. fundyense*) when exposed to 75 Pa PCO_2 compared to 38 Pa. Hattenrath-Lehmann et al. (2015b) demonstrated that the effects of PCO_2 on the toxicity of *Alexandrium fundyense* cultures are strain-specific, as cellular toxicity was significantly and consistently enhanced (70- 80%) at higher PCO_2 levels in the Northport Bay strain while the Bay of Fundy strain displayed more variability and no consistent pattern of increased toxicity. While the most abundant toxin in the Northport Bay strain was the epimer pair C1,C2, the cellular toxicity was driven mainly by the more potent derivative, GTX1,4, which became a larger percentage of the toxin composition, increased in toxin content, and was the only derivative whose contribution to the total toxicity significantly increased (almost doubled) with increasing PCO_2. Tatters et al. (2013) also demonstrated that concentrations of GTX1,4 doubled in high PCO_2 treatments, suggesting a specific biochemical pathway may be involved in this composition shift for this species. Further, Hattenrath-Lehmann et al. (2015b) demonstrated that *Alexandrium* densities were significantly and consistently enhanced when natural populations were incubated at 150 Pa PCO_2 compared to ~39 Pa. Moreover, during natural *Alexandrium* blooms in Northport Bay, PCO_2 concentrations increased over the course of a bloom to more than 170 Pa and were highest in regions with the greatest *Alexandrium* abundances, suggesting *Alexandrium* may further exacerbate acidification

and/or be especially adapted to these acidified conditions. The co-occurrence of *Alexandrium* blooms and elevated PCO_2 represents a previously unrecognized, compounding environmental threat to coastal ecosystems. The ability of elevated PCO_2 to enhance the growth and toxicity of *Alexandrium* indicates that acidification promoted by eutrophication or climate change can intensify these, and perhaps other, harmful algal blooms.

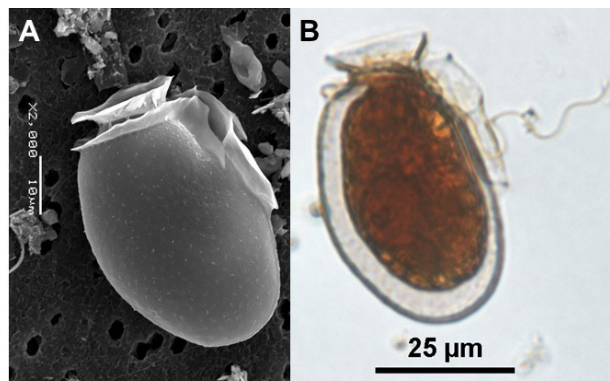**BLOOMS CAUSED BY THE DSP-PRODUCING MARINE DINOFLAGELLATE, *DINOPHYSIS ACUMINATA***

Photo: (A) A scanning electron micrograph (Photo credit: Steve Morton) and (B) a light microscope image of a Lugol's preserved *Dinophysis acuminata* from Long Island, New York (Photo credit: Theresa Hattenrath-Lehmann).

In contrast to paralytic shellfish poisoning (PSP), diarrhetic shellfish poisoning (DSP) is globally less common with recurring cases primarily occurring in Europe, South America and Southeast Asia (Hallegraeff, 1993; Van Dolah, 2000; Reguera et al., 2014). Both, *Prorocentrum lima* and other bloom forming dinoflagellates of the *Dinophysis* genus have been implicated in DSP events and closures around the world (Hallegraeff, 1993; Van Dolah, 2000; Reguera et al., 2012; Reguera et al., 2014). Similar to *Alexandrium*, these dinoflagellates do not need to discolor the water for toxins to be present in shellfish at concentrations harmful to humans. These dinoflagellates synthesize the causative toxins of DSP, namely okadaic acid (OA), and associated congeners, dinophysistoxins (DTXs; Lee et al., 1989). These toxins are protein phosphatase inhibitors which cause the hyperphosphorylation of control proteins in intestinal cells leading to diarrhea, nausea and vomiting (Van Dolah, 2000; Reguera et al., 2014). The onset of symptoms is generally rapid (~3h) as is resolution (2-3 days), however can lead to

complications from severe dehydration and greatly affect those with compromised immune systems (Van Dolah, 2000; USFDA, 2011). Also found associated with DSP toxins are another group of toxins known as pectenotoxins (PTXs), which are potentially hepatotoxic to, and promote tumor formation in, mammals when injected intraperitoneally (Lee et al., 1989; Burgess and Shaw, 2001). The USFDA (2011) regulates DSP toxins in shellfish setting a federal closure limit of 160 ng g^{-1} of shellfish tissue (0.16ppm), which includes total okadaic acid congeners (free toxins + esters). Unlike the European Union, however, the United States does not regulate for PTXs and is therefore not included in combination with DSP toxins in regulatory limits (EFSA, 2008; USFDA, 2011).

Dinophysis is also found worldwide, encompassing more than 100 species (Gómez, 2005), with only 12 of these species capable producing DSP toxins, pectenotoxins (PTXs) or both (Reguera et al., 2012; Reguera et al., 2014). Dinophysis acuminata is the species most often associated with DSP events and forms a "complex" with other morphologically similar species such as Dinophysis ovum and sacculus (Reguera et al., 2012; Reguera et al., 2014). Unlike most phylogenetic studies, species within the Dinophysis acuminata complex cannot be molecularly identified by using the ribosomal LSU genes (Edvardsen et al., 2003). Thus far, the mitochondrial cytochrome oxidase 1 (cox1) gene has been the only molecular marker that distinguishes Dinophysis acuminata from the other morphologically-similar species within the D. acuminata complex (Raho et al., 2008; Raho et al., 2013). This molecular marker, however, hasn't been as successful in terms of species-specificity in other clades or groups (Raho et al., 2013). The Dinophysis acuminata complex is responsible for the majority of DSP closures found in North America (Campbell et al., 2010; Deeds et al., 2010; Trainer et al., 2013; DMF, 2015) with the exception of some events found in Maine and in Canada where Dinophysis norvegica (Jon Deeds pers. communication) was present during these events (Subba Rao et al., 1993; Todd, 1997).

While DSP events are a common occurrence in Europe, South America and Asia, with reports of human illness dating back to the 1960s (Hallegraeff, 1993; Van Dolah, 2000; Reguera et al., 2012; Reguera et al., 2014), historically DSP closures in North America have been a rarity (Quilliam et al., 1991; Subba Rao et al., 1993; Todd, 1997; Tango et al., 2004; Campbell et al., 2010; Deeds et al., 2010; Trainer et al., 2013). In the United States, prior to 2008 there were no

recorded DSP events (Campbell et al., 2010; Deeds et al., 2010; Trainer et al., 2013; DMF, 2015). In recent years, however, the United States has witnessed an expansion of *Dinophysis* blooms causing shellfish to accumulate DSP toxin levels exceeding the USFDA action level on the east (NY, MA; Hattenrath-Lehmann et al., 2013; DMF, 2015), west (WA; Trainer et al., 2013) and gulf coasts (TX; Campbell et al., 2010; Deeds et al., 2010; Swanson et al., 2010). Of note, is the fact that in the United States shellfish accumulate DSP toxins over regulatory thresholds at a range (10^3 -10^5 cells L^{-1}) of *Dinophysis* densities (Campbell et al., 2010; Hattenrath-Lehmann et al., 2013; Trainer et al., 2013) that are often higher than densities (10^2-10^3 cells L^{-1}) in other regions of the world, in part due to differences in cellular toxicity (Reguera et al., 2012; Reguera et al., 2014). Over a decade (1971-1986, n=1300 samples) of monitoring on Long Island (NY, USA) showed that twelve species of *Dinophysis* were present across multiple harbors with *D. acuminata* being the most abundant species (Freudenthal and Jijina, 1988). Cells densities were generally low (<10^3 cells L^{-1}), peaking in the summer months (June-July) and on a single occasion reaching 13,000 *D. acuminata* cells L^{-1} in Cold Spring Harbor. Despite the presence of *Dinophysis* in multiple embayments across Long Island, toxic shellfish were not observed in these regions (Freudenthal and Jijina, 1988) although these authors described cases suggestive of DSP in Long Island's Nassau County as early as 1983. In coastal waters off of Long Island, *D. acuminata* was determined to be a significant percentage of the phytoplankton population in Block Island Sound in June 1971 (Staker et al., 1979) and Staker and Bruno (1978) found significant numbers in southwest Block Island Sound (Napeague Bay, Tobaccolot Bay, Fort Pond Bay) during June, 1976. During a recent investigation of *Alexandrium* in Northport Bay, NY (Hattenrath et al., 2010), elevated *Dinophysis acuminata* densities (10^4 cells L^{-1}) were found in 2008 and thus targeted in the HAB monitoring program in proceeding years.

As a result of the elevated *Dinophysis acuminata* densities found in Northport Bay in 2008 targeted sampling was conducted for this species as part of the Long Island HAB monitoring program (see above *Alexandrium* section). As of December 2016 this monitoring program has resulted in the collection and subsequent analysis of ~1150 *Dinophysis* samples island-wide (Fig. 8). This large-scale effort has allowed for the identification of *Dinophysis* hotspots across Long Island, specific sites of concern are those indicated in red and blue (>10^4 cells L^{-1}) in Figure 8, 9 sites altogether, 2 of which are in the Northport Bay-Huntington Harbor

complex. Northport Bay (max= >1 million cells L^{-1}), Meetinghouse Creek (max= 2 million cells L^{-1}), Cold Spring Harbor (max= 68,000 cells L^{-1}) and Hempstead Harbor (max= 175,000 cells L^{-1}) have hosted the largest and most chronic (Northport Bay and Cold Spring Harbor) *Dinophysis* blooms since the start of the HAB monitoring program. During the 2011 *Dinophysis* bloom in Northport Bay, which peaked at >1 million cells L^{-1}, DSP toxins (max toxicity= 1245 ng g^{-1}) were detected in shellfish for the first time in Long Island, New York (Hattenrath-Lehmann et al., 2013). Five samples (from four sites) exceeded the USFDA action level and while four of these samples were in areas already closed to shellfish due to coliform contamination, one of these samples was collected in an area open to shellfish harvest (Table 3). Another *Dinophysis* hotspot region, Cold Spring Harbor, also had toxic shellfish that were slightly over the USFDA action level but were found in regions already closed to shellfishing (Table 3). Maximum shellfish toxicities and corresponding maximal observed *Dinophysis* densities during these blooms can be found in Table 3. The DSP toxins, okadaic acid, dinophysistoxin 1 and their esters, in addition to pectenotoxins have been found in shellfish and *Dinophysis* concentrates. Over the course of this multiyear (2008- 2016) investigation, *Dinophysis* was found at 58 of 59 (98%) sites sampled across Long Island (n=1143; Fig. 8) and have exceeded densities reported 30 years ago (Freudenthal and Jijina, 1988). The presence of *Dinophysis* and DSP toxins over the federal closure limit in multiple sites across Long Island demonstrates the continued need for the monitoring of this HAB in NY waters.

In Long Island, New York, *Dinophysis acuminata* cells were 47.29 ± 2.92 μm long by 29.28 ± 1.86 μm wide and somewhat oval-shaped with a convex dorsal margin (Hattenrath-Lehmann et al., 2013). For some of the major monitoring sites (Northport Bay and Meetinghouse Creek), *D. acuminata* was present in the water column at salinities and temperatures of 8.2 to 26.7 and 8.7 to 26.7°C, respectively (Hattenrath-Lehmann et al., 2015a). At these same sites, peak *Dinophysis* densities typically occurred between April and June, at temperatures of 13-24°C (Hattenrath-Lehmann et al., 2015a) with peak shellfish toxicity occurring in June through July (Table 3; Hattenrath-Lehmann pers. obs.). Maximal growth rates of cultures isolated from Long Island, New York reached 0.5 d^{-1} (Hattenrath-Lehmann pers. obs.) and fall within range (0.06 to 0.95 d^{-1}) of those reported for *Dinophysis* spp. cultured thus far (Park et al., 2006; Kim et al., 2008; Kamiyama and Suzuki, 2009; Riisgaard and Hansen, 2009; Tong et al., 2010; Nagai et al., 2011; Nielsen et al., 2012, 2013).

One of the main objectives of the MERHAB funded portion of the monitoring program, was to identify ideal technologies for monitoring DSP toxins. Therefore, passive solid-phase adsorption toxin tracking (SPATT) samplers using two different types of resins, HP20 and XAD-2, were deployed (Fig. 9). Overall, the HP20 resin on average adsorbed 3.1 times more total DSP toxins and 2.7 times more total PTXs than the XAD-2 resin; suggesting that HP20 resin is the better candidate for continued DSP monitoring efforts. Overall toxin concentrations adsorbed by the HP20 resin paralleled *Dinophysis* densities, particulate toxins and toxins in blue mussels and was able to detect toxins in the water column prior to detection by shellfish that were specifically hung for monitoring purposes (Hattenrath-Lehmann et al. submitted). The use of SPATT samplers containing HP20 resin is therefore a promising HAB monitoring technology that is easy, safe and forgoes the use of live animals (mussels) that are time consuming in their weekly maintenance. In addition, the use of the Abraxis protein phosphate inhibition (PP2A) assay for measuring total DSP toxins in New York shellfish was investigated and compared to analysis using liquid chromatography- mass spectrometry (LCMS). The Abraxis kit was validated using certified reference materials purchased from the National Research Council of Canada, with all reference materials including a non-toxic mussel homogenate, a naturally contaminated mussel sample and a methanolic calibration standard, yielding recoveries between 100-113% (Hattenrath-Lehmann et al. submitted). Shellfish extracts, from various species, measured via PP2A (Gobler Lab) and LCMS (Morton Lab) were not significantly (p=0.2) different from each other and were also highly correlated (R^2= 0.97; p<0.001). Overall, the Abraxis PP2A assay was found to be an excellent candidate for integration into monitoring programs due to their correlation with LCMS data, ease of use, and rapid results (within a day). While ELISA assays have a similar potential as PP2A's we found that for Long Island the toxin composition in shellfish was such that the assay would grossly underestimate toxins in shellfish given that cross reactivity with one of the dominant congeners (DTX1, >30% of total toxicity) was only 50%. This highlights the importance of prescreening shellfish using LCMS to assess congener composition for newly studied areas to determine the most efficient and appropriate analytical method to use (Hattenrath-Lehmann et al. submitted).

Factors promoting *Dinophysis* blooms

Unlike other HABs, *Dinophysis* is an obligate mixotroph, a trait that has until the recent discovery of its food source, *Mesodinium rubrum*, thwarted the ability to study this organism in culture (Park et al., 2006). Culturing *Dinophysis* requires a three-step process in which *Dinophysis* sequesters and utilizes plastids (kleptoplastids) from *M. rubrum* (Minnhagen and Janson, 2006; Wisecaver and Hackett, 2010; Minnhagen et al., 2011) which they, in turn, steal from their cryptophyte prey (Hansen et al., 2012). Given this recent discovery most of the knowledge gained on this organism prior to these past 10 years has been gleaned from field investigations that are unable to separate the multiple physical, chemical and biological factors that may influence *Dinophysis* growth and survival. Thus far, *Dinophysis* culture research has focused on: differences in toxicity and toxin profiles between species and geographic isolates (Hackett et al., 2009; Kamiyama and Suzuki, 2009; Fux et al., 2011); toxin production and/or excretion (Nagai et al., 2011; Tong et al., 2011; Smith et al., 2012; Nielsen et al., 2013); the effects of prey concentration (*Mesodinium rubrum*) on growth (Kim et al., 2008; Kamiyama and Suzuki, 2009) and toxin production (Nielsen et al., 2012); the effects of temperature on *Dinophysis* growth (Gobler et al. submitted) and toxicity (Kamiyama et al., 2010; Basti et al., 2015); the effects of light intensity on the growth and/or toxicity of *Dinophysis* (Kim et al., 2008; Tong et al., 2011; Nielsen et al., 2012, 2013) and most recently the effects of nutrients on *Dinophysis* growth (Hattenrath-Lehmann and Gobler, 2015) and toxicity (Nagai et al., 2011; Tong et al., 2015). Despite these recent advances, there are still many areas of *Dinophysis* ecology that have yet to be explored.

To date, there have been few studies on the effects of nutrients on *Dinophysis* growth. While a series of field studies have found no relationship between *Dinophysis* densities and nutrient concentrations (Delmas et al., 1992; Giacobbe et al., 1995; Koukaras and Nikolaidis, 2004), another field-based study used ^{15}N-labeled nitrogen to demonstrate that *Dinophysis* dominated-communities (91% of total biomass as C) had a high affinity for ammonium (Seeyave et al., 2009). Similarly, a recent ecosystem-based study in NY found that *D. acuminata* abundances were positively dependent on multiple environmental parameters including ammonium (Hattenrath-Lehmann et al., 2015a). This same study conducted nutrient amendment experiments using water from NY estuaries (Hattenrath-Lehmann et al., 2015a) and demonstrated that *Dinophysis* growth can be promoted by the addition of both inorganic (nitrate, ammonium) and organic N (glutamine, HMW STP). However, it was unclear whether these

effects were direct (via nutrient uptake by *Dinophysis*) or indirect (via nutrient stimulation of prey) as these experiments were conducted using the whole phytoplankton community. Another study (Hattenrath-Lehmann and Gobler, 2015) using a culture of a Long Island, New York *Dinophysis* isolate demonstrated using multiple experimental approaches that both inorganic and organic nutrients significantly enhanced the growth of *D. acuminata*. More specifically, Hattenrath-Lehmann and Gobler (2015) demonstrated that these nutrient effects were direct as enhancements were seen in cultures of *D. acuminata* where no *M. rubrum* was added. *Dinophysis* was found to rapidly assimilate ^{15}N-labeled ammonium and urea, and to a far lesser extent nitrate. Cultures grown with and without prey generally grew faster with ammonium, glutamine, or organic matter from sewage effluent added than respective controls, while nitrate grown cultures yielded significantly more rapid growth only when fed copious amounts of *Mesodinium* (Hattenrath-Lehmann and Gobler, 2015). The only other culture studies investigating the effects of nutrients on *Dinophysis* growth concluded that both organic matter originating from sonicated *M. rubrum* cultures (Nagai et al., 2011) and nitrate (Tong et al., 2015) had no effect on the growth of *D. acuminata* cultures. While Tong et al. (2015) only tested the effect of nitrate on *Dinophysis* cultures their findings were similar to those of Hattenrath-Lehmann and Gobler (2015) that demonstrated nitrate only enhanced *Dinophysis* densities when prey was available (indirect effect). These studies demonstrate that N can directly promote the growth of *Dinophysis* and supports the hypothesis that accelerated N loading in coastal ecosystems can promote *Dinophysis* blooms.

Temperature is another factor that can significantly affect *Dinophysis* growth. Recent culture studies (*D. acuminata* and *D. caudata*) have demonstrated that *Dinophysis* growth increases with temperature with upper thresholds likely being species or strain specific (Kamiyama et al., 2010; Tong et al., 2010; Tong et al., 2011; Basti et al., 2015, Gobler et al. submitted). Using multiple regression models, Hattenrath-Lehmann et al. (2015a) found that *D. acuminata* abundances were positively and strongly dependent on temperature, consistent with the distinct seasonality of these New York blooms (Hattenrath-Lehmann et al., 2013) as well as other blooms around the globe (Hoshiai et al., 2003; Reguera et al., 2012). A model created by Gobler et al. (submitted) used temperature-growth relationships from a culture of *Dinophysis acuminata* and ~30 years of high resolution sea surface temperature records (1982-2015) to hindcast *Dinophysis* blooms in regions of the Northwest Pacific and North Sea. This model was

consistent with in situ observations demonstrating that regions of rapid warming are also regions were *Dinophysis* blooms are occurring earlier in the year (Edwards and Richardson, 2004; Edwards et al., 2006; Whyte et al., 2014). While some *Dinophysis* blooms in other regions of the world were related to various hydrodynamic processes such as cell accumulations from wind and stratification or advection of offshore populations into embayments (Subba Rao et al., 1993; Lindahl et al., 2007; Campbell et al., 2010; Swanson et al., 2010; Sjöqvist and Lindholm, 2011), this is not the case in NY as estuaries here are relatively well mixed and have been highly correlated with other physical factors such as temperature (Hattenrath-Lehmann et al., 2013; Hattenrath-Lehmann et al., 2015a). The above studies demonstrate that *Dinophysis* blooms are temperature dependent and may increase in intensity or shift the window in which they typically occur (window of opportunity) with future climatic warming.

FRESHWATER CYANOBACTERIAL BLOOMS

The most common genera of cyanobacteria found in Suffolk County and a bloom of *Microcystis* within Lake Agawam, Southampton, NY (photo courtesy of M. Harke).

Cyanobacteria, also known as blue-green algae, are a common family of photosynthetic prokaryotic organisms that can dominate phytoplankton communities in aquatic systems (Paerl, 1988). They can be found in marine, brackish, and freshwater systems, some of which are used as drinking sources (Falconer, 1989; Carmichael, 1994; Chorus and Bartram, 1999). In eutrophic freshwaters, cyanobacteria can grow at dense concentrations, often forming blooms, the decomposition of which can result in hypoxia or anoxia, leading to fish mortality, and a breakdown of the food chain within the system (Carmichael, 1994; Chorus and Bartram, 1999). In addition, at least twelve species of blue-green algae including those in the genera *Anabaena*,

Aphanizomenon, *Nodularia*, *Oscillatoria*, and *Microcystis*, can produce strong neurotoxins and gastrointestinal toxins (Carmichael, 1994; Chorus and Bartram, 1999). While toxic freshwater blue-green harmful algal blooms have been reported in the scientific literature for more than 130 years (Francis, 1878), in recent decades, the incidence and intensity of these blooms, as well as economic loss associated with these events has increased (Chorus and Bartram, 1999; Carmichael et al., 2001; Carmichael, 2008; Paerl and Huisman, 2008).

Blue-green algal toxins can cause animal and human poisonings or health risks (Codd et al., 2005). While many blue-green algal toxins have recognized immediate toxic effects, the impact and long term effects of many of these compounds is unknown (Tonk, 2007). Globally and in Suffolk County, the hepatotoxin or liver toxin, microcystin, is the most prevalent cyanobacterial toxin followed by neurotoxins such as the neurotoxin, anatoxin-a. There are multitudes of examples of animal sicknesses and deaths associated with chronic, or even sporadic, consumption of water contaminated with cyanotoxins (O'Neil et al., 2012; Backer et al., 2013). Cyanotoxin exposure has been linked to mild and potentially fatal medical conditions in humans including gastrointestinal cancers (i.e. liver, colorectal; Chorus and Bartram, 1999). Cox et al., (2003; 2005) recently described the presence of the neurotoxic compound, BMAA in most blue-green algae. It has been hypothesized that BMAA may be a possible cause of the amyotrophic lateral sclerosis parkinsonism–dementia complex (ALS-PDC; Cox et al., 2003; Cox et al., 2005; Cox et al., 2009).

One very serious danger of blue-green algal toxins is associated with the tendency of the blue-green algae to float to the surface of lakes during the day due to their possession of photosynthetic gas vesicles (Chorus and Bartram, 1999). When persistent south winds develop off the Atlantic Ocean during the day in summer, this tends to blow surface accumulating blue-green algae along the northern extent of lakes, forming a shoreline scum. For example, samples that were collected to contrast the whole lake and shoreline scum levels of microcystin have shown microcystin levels can be 50-times higher in the scum than within Suffolk County Lakes, at times reaching 15,000 µg L^{-1} (Gobler et al., 2007a), a level 750-times the World Health Organization recreational guideline for this toxin and 2,000-times the drinking water standard (Chorus and Bartram, 1999). Dogs may be most at risk from blue-green algal toxins that are concentrated at the shoreline. Backer et al. (2013) compiled data through the US Centers for

Disease Control and Prevention and reported on more than 400 cases of intoxication of dogs with blue-green algae toxins, in many cases leading to death. According to the NYSDOH, in 2012, a dog died upon drinking water or consuming shoreline scum from Georgica Pond in East Hampton and according to Suffolk County Department of Health Services, in 2015, two dogs were sickened drinking from Fort Pond in East Hampton that has experienced blue-green algal blooms. In 2016, a dog became seriously ill following the consumption of water from Mecox Bay with a blue-green algal bloom (William Pell, Southampton Town Trustee, pers. comm.). In some cases, animal illnesses have been associated with exposure to blooms, but not necessarily exposure to toxins.

Blue-green algal blooms in Suffolk County

While blue-green algae was likely present in Suffolk County since it was geologically formed, the first reported blue-green algae bloom was in Lake Agawam, Southampton, by the Gobler Laboratory in 2003. At that time, the toxic cyanobacteria, the potentially toxic blue-green algae, *Microcystis* and *Anabaena* were identified in lake waters the toxins microcystin and anatoxin-a were found at levels of 2.5 and 1.0 µg L^{-1}, respectively. These levels of microcystin surpassed the World Health Organization (WHO) safe drinking water guidelines of 1 µg L^{-1} but were below the recreational water guideline of 20 µg L^{-1} (Chorus and Bartram, 1999). Since 2004, dense, toxic blue-green algal blooms have been recorded from May and through November each year in Lake Agawam.

The discovery of blue-green algae in Lake Agawam prompted a Suffolk County-wide initiative supported by the Suffolk County Department of Health Services that surveyed 20 lakes across the County from 2004 through 2007. All twenty lakes sampled contained potentially toxic blue-green algae (typically *Microcystis* sp.) and detectable levels of the hepatotoxin made by blue-green algae, microcystin (Fig. 10). Fifteen of the lakes had levels of microcystin exceeding levels permissible for drinking water according to the World Health Organization (WHO). Fortunately, Suffolk County residents use these systems for recreation, not potable water, and levels of toxin in 15 of the 20 lakes sampled were below levels considered to be a low recreational risk by the WHO. However, the remaining five lakes (Lake Agawam, Old Town Pond, Mill Pond (Water Mill), East Mill Pond (Forge River) and Lake Ronkonkoma) all posed

moderate-to-high risks to human health for recreation at various times during the study. Each of these systems hosted elevated densities of multiple species of toxic cyanobacteria (up to 10^6 cells ml^{-1}) and high levels of microcystin (up to 15 mg L^{-1}) and, in some cases, anatoxin-a (up to 1 µg L^{-1}). All of these systems except Lake Ronkonkoma are hypereutrophic, with high levels of nitrogen and phosphorus, suggesting a relationship between nutrient loading and bloom occurrence. Incubation experiments with natural communities in Mill Pond and Lake Agawam demonstrated that nitrogen and phosphorus loading can enhance the biomass and toxicity of these two systems.

Through 2010, the Gobler Lab continued to document blue-green algal blooms in Mill Pond and Lake Agawam with support from Southampton Town and/or the Lake Agawam Conservation Association. Some important observations during this time included hypoxic events and fish kills following the collapse of blooms. In September 2006, there was a kill of thousands of white perch across Lake Agawam when nighttime oxygen levels dropped to < 1mg L^{-1} and in September 2008, thousands of fish and eels perished in Mill Pond following the collapse of a blue green bloom that may have been intensified by disturbance of bottom sediments by a water circulator (Gobler, 2010).

Analyses by Dr. Greg Boyer from SUNY College of Environmental Science and Forestry in 2004 demonstrated that microcystin is also accumulating in both the fillet and viscera of carp and bass in Lake Agawam at levels exceeding the World Health Organization's allowable limits for fish (Chorus and Bartram, 1999). The presence of these toxins prompted the Southampton Town Trustees to first close Lake Agawam to recreation and fishing in 2004. Similar advisories have been implemented by Suffolk County's Department of Health Services for most of the summer since 2013 when they began their County-wide program of monitoring lakes.

In 2013, New York State Department of Environmental Conservation (NYSDEC) began a program to monitor hundreds of lakes and ponds in all 62 counties of the state and to create public warnings and lists specific water bodies on its statewide website when they experience a blue green algae bloom at an intensity exceeding its threshold of 25 µg of blue green algal pigment L^{-1}. During 2014, 2015, and 2016, blue-green algal blooms were reported in Suffolk County more frequently and in more water bodies than any other County in NYS (Fig. 11 & 12). In Suffolk County, Lake Agawam was listed by NYSDEC more frequently than any other water

body in NYS. In total, 31 water bodies across Suffolk County have experienced blue-green algae blooms at a level exceeding the NYSDEC threshold of 25 µg of blue green algal pigment L^{-1}, leading to the listing of these water bodies on the NYSDEC website (Table 4). The World Health Organization (WHO) considers levels greater than 1 µg L^{-1} as a health risk for drinking water and 20µg L^{-1} a moderate recreational risk (Chorus and Bartram, 1999). Of the 31 water bodies in Suffolk County that have experienced blue-green algal blooms, 23 have had microcystin levels exceeding 1 µg L^{-1} and eight have had more than 20 µg L^{-1}, Lake Agawam, Wickapogue Pond, Mill Pond, Maratooka Lake, Roth Pond, Lake Ronkonkoma, the Spring Lake Golf Course, and Georgica Pond (Table 4).

The role of nutrients in promoting blue-green algal blooms

Blue-green algal blooms are promoted by excessive nutrient loading (Chorus and Bartram, 1999; Paerl et al., 2001; Gobler et al., 2016). Traditionally, it has been thought that algal growth is controlled by phosphorus in freshwater bodies based on the premise that nitrogen-fixing blue-green algae balance ecosystem N deficiencies (Schindler, 1974; Schindler et al., 2008; Scott and McCarthy, 2010; Schindler, 2012). As the total P concentration in many freshwater bodies has increased, a shift has been reported in phytoplankton assemblages toward blue-green algae dominance (Smith, 1983; Trimbee and Prepas, 1987; Watson et al., 1997). Over the past several decades, many lakes have been driven increasingly out of stoichiometric balance due to disproportionate anthropogenic inputs of N and P (Conley et al., 2009; Glibert et al., 2011; USEPA, 2015). Consequently, the literature is rich with examples of the importance of P (Schindler, 1977; Wetzel, 2001; Sterner, 2008; Schindler et al., 2016, and references therein), and N (e.g. Gobler et al., 2007a; Davis et al., 2010; Beversdorf et al., 2013; Beversdorf et al., 2015) in controlling cyanobacteria blooms as well as with examples of N and P co-limitation (Elser et al., 1990; Elser et al., 2007; Lewis and Wurtsbaugh, 2008; Xu et al., 2010; Chaffin et al., 2013; Chaffin et al., 2014; Davis et al., 2015). Concurrently, thought has evolved to recognition of the importance of controlling nitrogen to restrict phytoplankton assemblage structure and productivity (Conley et al., 2009; Glibert et al., 2011; USEPA, 2015; Paerl et al., 2016).

There are several lines of evidence that demonstrate that nitrogen controls the growth and toxicity of blue-green algae within some Suffolk County water bodies. First, blue-green algae

blooms in many Suffolk County lakes are largely comprised of *Microcystis*, a blue-green alga that cannot extract gaseous forms of nitrogen and thus must rely on in-lake sources of nitrogen (Gobler et al., 2016). Gobler et al. (2007a) reported on the ability of nitrogen, but not phosphorus, to promote both the growth and toxicity of blue-green algal blooms in Lake Agawam and Mill Pond. Davis et al. (2009) also showed that nitrogen, but not phosphorus, promoted the growth of *Microcystis* in Lake Agawam and Lake Ronkonkoma when temperatures were elevated. Davis et al. (2010) and Gobler et al. (2007a) both showed that the ratio of inorganic nitrogen to inorganic phosphorus varied from high to low levels from spring to summer and that during summer, levels were far below 16, the ratio that provides the proper balance of nitrogen and phosphorus in the system, suggesting Lake Agawam was deficient in nitrogen at that time. Davis et al. (2010) also showed that nitrogen almost always promoted the growth of total, toxic, and non-toxic forms of *Microcystis.* In the summer of 2014 and 2015, the Gobler laboratory returned to Lake Agawam to again access the relative importance of nitrogen and phosphorus in controlling the growth of blue-green algae and found that, regardless of whether populations were incubated at ambient or elevated temperatures, nitrogen selectively promoted the growth of blue-green algae.

Careful research during the past decade has demonstrated that the blue-green algal blooms in some Suffolk County water bodies are being promoted by excessive nitrogen loading and, to a lesser extent, phosphorus loading (Gobler et al., 2007a, unpublished; Davis et al., 2010). High levels of nitrogen promote both the growth and toxicity of blue-green algae in Lake Agawam (Gobler et al., 2007a, unpublished; Davis et al., 2010). Prior experimental work has shown that reducing nitrogen loads into Lake Agawam could reduce the intensity of blue-green algal blooms there (Harke et al., 2008).

Physical factors: Temperature, salinity, residence time, climate change

Blue-green algae blooms are known to be warm temperature phenomena. Consistent with global trends within temperate latitudes (Chorus and Bartram, 1999), in Suffolk County, they typically commence in May or June once water temperatures exceed 20°C and typically end in the fall, as water temperatures drop below 15°C, although blooms have been observed as late as December in some cases (e.g. Mill Pond, Water Mill, 2016). Blooms are most common within lakes and ponds although blooms have been observed in Georgica Pond, East Hampton,

and Mecox Bay, Southampton, which are temporarily open-closed estuaries that can experience salinities as low as 5 and have experienced blue-green algae blooms at salinities < 15 PSU, a finding consistent with the known salinity tolerance of some blue-green algae (Chorus and Bartram, 1999). Blooms are also most common in stagnant water bodies that permit for the accumulation of nutrients and biomass (Chorus and Bartram, 1999). While many lakes and ponds in Suffolk County have outlets to estuaries of other lakes, most bloom-prone systems have very limited flushing. It is notable that in Georgica Pond, East Hampton, blue-green algae blooms that persisted for several weeks ended in 2014 and 2015 within hours of the water body being opened to the Atlantic Ocean (C. Gobler, unpublished).

The sum of research conducted regarding the evolutionary history, ecophysiology, and *in situ* dynamics of cyanobacteria suggests that they will thrive under the conditions predicted for global climate change (Paul, 2008; Paerl and Huisman, 2009). Phytoplankton communities are influenced by increases in temperature as algal growth rates are strongly, but differentially, temperature dependent (Eppley, 1972; Goldman and Carpenter, 1974; Raven and Geider, 1988). As temperatures approach and exceed 20ºC, the growth rates of freshwater eukaryotic phytoplankton generally stabilize or decrease while growth rates of many cyanobacteria increase, providing a competitive advantage (Canale and Vogel, 1974; Peperzak, 2003; Paerl and Huisman, 2009). Beyond the direct effects on cyanobacterial growth rates, rising temperatures will change many of the physical characteristics of aquatic environments in ways that may be favorable for cyanobacteria. For instance, higher temperatures will decrease surface water viscosity and increase nutrient diffusion towards the cell surface, an important process when competition for nutrients between species occurs (Vogel, 1996; Peperzak, 2003). Secondly, since many cyanobacteria can regulate buoyancy to offset their sedimentation, a decrease in viscosity will preferentially promote the sinking of larger, non-motile phytoplankton with weak buoyancy regulation mechanisms (e.g. diatoms) giving cyanobacteria a further advantage in these systems (Paerl and Huisman, 2009; Wagner and Adrian, 2009). Thirdly, insular heating will increase the frequency, strength, and duration of stratification. This process will generally reduce the availability of nutrients in surface waters favoring cyanobacteria that regulate buoyancy to obtain nutrients from deeper water, or that are diazotrophic. Consistent with the sum of these observations, cyanobacteria tend to dominate phytoplankton assemblages in eutrophic, freshwater environments during the warmest periods of the year, particularly in

temperate ecosystems (Paerl, 1988; Paerl et al., 2001; Paerl and Huisman, 2008; Paul, 2008; Liu et al., 2011). For all of these reasons, it has generally been concluded that cyanobacterial blooms may increase in distribution, duration and intensity, as global temperatures rise (Paul, 2008; Paerl and Huisman, 2009).

Zooplankton grazing

While zooplankton are expected to control phytoplankton populations, many species of blue-green are not readily grazed by mesozooplankton (Porter and Orcutt, 1980; Lampert, 1981; Fulton and Paerl, 1987; Sellner et al., 1994; Gobler et al., 2007a). This reduced grazing can, in turn, allow these HABs to occur (Sunda et al., 2006; Smayda, 2008). The grazing ability of large cladocerans, such as *Daphnia*, are more inhibited by the presence of cyanobacteria than the grazing ability of small-bodied cladocerans (Gliwicz, 1977), rotifers (Orcutt and Pace, 1984; Fulton and Paerl, 1987), and copepods (Richman and Dodson, 1983). Disruption of grazing is likely due to 1) colony size and/or morphology of cyanobacteria (i.e. large filamentous or colonial forms are not well-grazed); 2) the absence of nutritional compounds (essential fatty acids) in cyanobacteria that zooplankton need to survive, and 3) compounds synthesized by cyanobacteria that may serve as grazing deterrents (as reviewed by Wilson et al., 2006). For example, the ability of *Microcystis* to synthesize multiple secondary metabolites that may inhibit zooplankton grazing and/or may cause zooplankton mortality (Arnold, 1971; DeMott et al., 1991; Rohrlack et al., 1999).

HIGH BIOMASS MICROALGAL BLOOMS
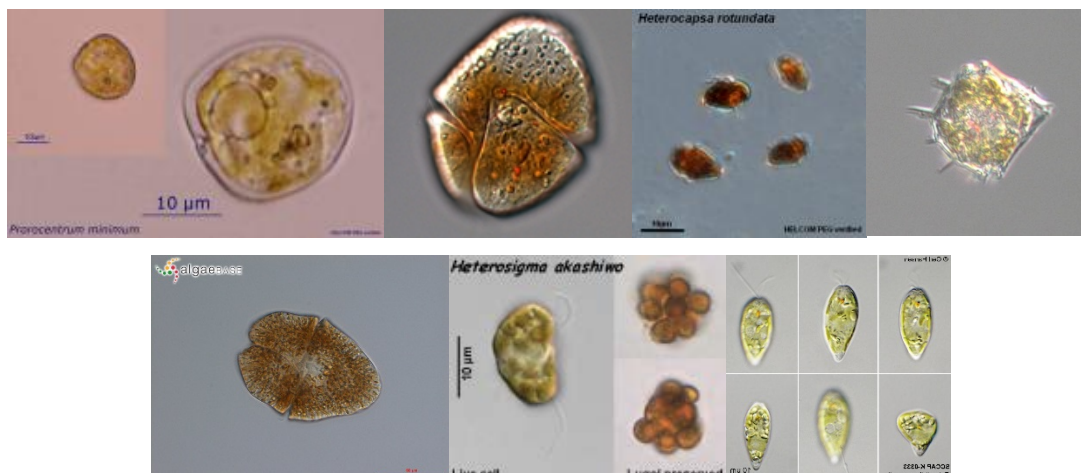
Images of algal species that form high biomass blooms in the Long Island, New York region. From top left to bottom right: the dinoflagellates, *Prorocentrum minimum* (source: Nordic microalgae), *Gymnodinium instriatum* (source: algal resources collection), *Heterocapsa rotundata* (source: Nordic microalgae), *Peridinium quinquecorne* (source: Nordic microalgae), *Akashiwo sanguinea* (source: algaebase), the raphidophyte, *Heterosigma akashiwo* (source: WoRMS), and the Euglenophyte group, *Eutreptiella* spp. (source: Nordic microalgae).

There are numerous species of microalgae indigenous to Long Island, New York, waters that bloom annually, are considered ecologically destructive, and commonly form high biomass (causes discoloration of water) algal blooms. These include the dinoflagellates, *Prorocentrum minimum*, *Gymnodinium instriatum*, *Heterocapsa rotundata*, *Peridinium quinquecorne*, *Akashiwo sanguinea*, the raphidophyte, *Heterosigma akashiwo*, and the Euglenophyte group, *Eutreptiella* spp. To our knowledge, *Gymnodinium* spp., *Peridinium quinquecorne* and *Heterocapsa rotundata* do not produce toxins but can contribute to biological oxygen demand at night and upon decay of high biomass blooms and have been associated with fish kills (Heil et al., 2001; Wang et al., 2005; FFWCC, 2010; Schuster, 2015; Alkawri et al., 2016). *Prorocentrum minimum*, also known as mahogany tide, has been associated with fish kills in the Chesapeake Bay estuary at concentrations $>10^4$ cells mL^{-1} with the Maryland Department of Natural Resources defining $>3,000$ cells mL^{-1} as a "threshold above which living resources" are impacted (Tango et al., 2005). While these blooms are typically considered ecologically destructive due to the association with hypoxic events and fish kills, there is also recent evidence for potential toxin production by some, but not all, strains of *P. minimum*, specifically neurotoxins (Vlamis et al., 2015), however other researchers have not yet been able to confirm or deny this claim. *Heterosigma akashiwo*, an ichthyotoxic raphidophyte, is known to cause death of fish from asphyxiation via an undefined mechanism (Cochlan et al., 2013) and has been responsible for fish kills around the world (Chang et al., 1990; Rensel, 2007; Kempton et al., 2008). The red tide dinoflagellate, *Akashiwo sanguinea*, has been associated with mortality and multiple forms of marine like. In 2007, a seabird mortality event occurred in Monterey Bay, California, and was attributed specifically to organic matter created by *Akashiwo sanguinea* containing 'surfactant-like proteins' that coated the feathers of these seabirds (Jessup et al., 2009). In addition, a number of invertebrate and fish mortalities have been associated with dense blooms of *Akashiwo sanguinea* (Cardwell et al., 1979; Harper and Guillen, 1989; Shumway, 1990). In addition, some species (Kim et al., 1999) from the genus *Eutreptiella* have been shown

to produce reactive oxygen species (ROS) and significant fish kills from this genus have been reported in Banderas Bay, Mexico (Cortes-Lara et al., 2010). In addition, other species such as *Euglena sanguinea* can produce toxins known as euglenophycin (Zimba et al., 2010). Given this information, there is clearly a potential for these harmful alga to elicit similar effects in our local waters.

While all the above species bloom in the New York region there have only been a few reports of invertebrate and fish mortalities associated with these events. In June of 1990, *Eutreptiella* sp. was responsible for an extensive bloom that discolored the water (green) in a north shore Nassau County harbor and caused some post-set oyster deaths as reported by a local commercial oyster company (Anderson et al., 2000). Again in the summer of 1990, there were a number of complaints of discolored water in Cold Spring Harbor, Hempstead Harbor and Manhasset Bay with samples indicating the causative species as *Prorocentrum* sp, *Eutreptiella* sp., *Skeletonema*, *Rhizosolenia*, *Chaetoceros* and *Dinophysis.* During this time there was a fish kill involving juvenile flounder in Cold Spring Harbor (July), and dead blue mussels (*Mytilus edulis*) and crabs (*Cancer irroratus*) were reported in Manhasset Bay (Anderson et al., 2000). In addition, there have been a number of fish kills in the Peconic River during the past two decades, notably September of 1999 (estimated at 1-3 million fish), August of 2000 (~750,000 fish), May of 2008 (100,000s) and May of 2009 (100,000s), with low dissolved oxygen (DO) identified as the main contributing factor (Tomarken et al., 2016), however it should be noted that detailed phytoplankton analysis was not conducted. In the spring of 2015 (Tomarken et al., 2016), a series of fish kills occurred in the Peconic River that were coincident with low DO and a number of these HAB species, including *Prorocentrum minimum* (~25,000 cells mL^{-1}), *Gymnodinium instriatum* (~800 cells mL^{-1}), and *Heterocapsa rotundata* (~10,000 cells mL^{-1}). During the spring of 2016, blooms of *Prorocentrum minimum* occurred in Great South Bay, Moriches Bay, Quantuck Bay, Shinnecock Bay, the Peconic River, Flanders Bay, and Georgica Pond and yielded to an extensive amount of foam across Great South Bay. Outside of minor fish kills in the Peconic River at the time (100s of fish), there were no major impacts on these estuarine ecosystems.

MACROALGAL BLOOMS

Images of *Ulva* spp., *Enteromorpha* spp., and *Cladophora* spp. (source: algaebase).

Macroalgae or seaweeds are key components of coastal ecosystems, serving as an important part of food webs and even habitat for some organisms (Valiela, 2006). Like phytoplankton, however, their abundances and species composition can be altered in problematic ways in the face of excessive nutrient loading (Valiela et al., 1997; Conley et al., 2009). The overgrowth of macroalgae is a common symptom of eutrophication as many species are capable of rapid growth in the presence of high nutrient concentrations and have a high assimilative capacity for nutrients (Valiela et al., 1997; Neori et al., 2004). Despite the great diversity of macroalgae, eutrophic conditions often lead to dominance by opportunistic Ulvalean species and species within the genus *Gracilaria* (Valiela et al., 1997). In some cases, blooms of Ulvalean macroalgae can be considered harmful, as they can outcompete and replace seagrass beds or cover other critical benthic habitats (Valiela et al., 1997; Howarth et al., 2000; Lapointe et al., 2004; Lapointe et al., 2005; Valiela, 2006; Thornber et al., 2008). To date, it has been generally concluded that macroalgae dominate estuaries have high nutrient loads and short residence times due to their ability to attach to the benthos and form floating dense mats (Valiela et al., 1997). In contrast, within eutrophic systems with longer residence times, phytoplankton grow and accumulate dense populations in water columns with minimal tidal flushing, leading to a high degree of pelagic light attenuation, an aphotic benthos, and the loss of benthic macroalgal communities (Valiela et al., 1997; MacIntyre et al., 2004).

Macroalgal blooms can be harmful to marine life. The overgrowth of macroalgae can cover critical benthic habitats and promote diel hypoxia/anoxia in estuaries (Valiela et al., 1997; Valiela and Cole, 2002; Liu et al., 2009) and *Ulva* has been shown to cause mortality in multiple calcifying animals including bivalves, barnacles, and larval crabs (Magre, 1974; Johnson and Welsh, 1985; Nelson et al., 2003). Since these calcifying animals are also sensitive to high levels of CO_2 (Ries et al., 2009; Findlay et al., 2010; Talmage and Gobler, 2010; Long et al., 2013), the stimulation of harmful macroalgae such as *Ulva* under elevated pCO_2 levels may

represent a previously unrecognized, compounding environmental threat to some forms of marine life (Young and Gobler, 2016). Sudden beaching of huge seaweed masses can smother the coastline and form rotting piles on the shore. The number of reports of these events in previously unaffected areas has increased worldwide in recent years (Smetacek and Zingone, 2013). These 'seaweed tides' can harm tourism-based economies, smother aquaculture operations or disrupt fisheries (Smetacek and Zingone, 2013).

Macroalgae in Suffolk County

The overgrowth and beaching of macroalgae, also known as green tides in the scientific literature (Smetacek and Zingone, 2013) are common occurrences in Nassau County, specifically within the Western Bays on the south shore and along ocean beaches of Long Beach and Lido Beach. The overgrowth of macroalgae is somehwhat less common in Suffolk County. Macroalgae have been observed to wash-up on some oceans beaches, for example in Quogue, and on bay beaches such as Amityville Beach and Tanner Park beach in Great South Bay. Nuzzi and Waters (2004) reported on the overgrowth of *Cladophora* in Great South Bay during which the water column of Great South Bay was unusually clear in the summer of 1999. This occurred again in 2011 when an unusually dense set of *Mulinia* dwarf surf clams kept the water column clear for much of the summer allowing *Ulva* and *Cladophora* to bloom across the Bay (Gobler, 2012). In 2015, a bloom of *Ulva* and other macroalgae occurred across central and eastern Great South Bay while there was a dense set of blue mussels that may have been related in part to the presence of the New Inlet. In each of these cases, the macroalgae populations collapsed in the late summer and a dense brown tide occurred in the fall, likely due in part to the enrichment of water column DON levels due to the degradation of the macroalgae (Nuzzi and Waters, 2004; Gobler, 2012). In 2011, the only known occurrence of a *Cochlodinium* bloom in Great South Bay followed the collapse of the macroalgae and preceded the fall brown tide (Gobler, 2012). One lesson from these sporadic macroalgae blooms in Great South Bay during the past twenty years is that an event that clears the water column such as an aberrant set of bivalves allows macroalgae to be the dominate autotroph in place of phytoplankton during summer, but that this trend is not sustainable and eventually yields a HAB event in the fall.

The other notable occurrences of macroalgae blooms in Suffolk County are in freshwater and brackish water bodies. While these have been casually observed in most cases, in 2014 and

2015, the Gobler lab documented a dense and widespread bloom of *Cladophora* in Georgica Pond, East Hampton. During these blooms, fish kills, eel kills, and bird kills were observed along with nocturnal anoxia. As this bloom collapsed in each summer, it was immediately succeeded by a dense blue-green algae bloom suggesting that, like Great South Bay, the remineralization of nutrients from within the macroalgae supported the bloom of blue-green algae.

Macroalgae that prevent HABs

While dying macroalgae release nutrients that may promote HABs, recent studies have demonstrated that some macroalgae can have strong allelopathic impacts on phytoplankton in general, and multiple HAB species, in particular. Multiple species of the green macroalgal genus, *Ulva*, including *U. fasciata, U. pertusa,* and *U. linza* have displayed the ability to cause rapid lysis of *Prorocentrum micans, Prorocentrum donghaiense, Heterosigma akashiwo* (Jin and Dong, 2003; Nan et al., 2004; Jin et al., 2005; Wang et al., 2007a; Wang et al., 2007b; Nan et al., 2008) and to reduce the growth of *Alexandrium tamarense* (Nan et al., 2004). The red coralline macroalga, *Corallina pilulifera*, has displayed algicidal activity against several harmful algae including *C. polykrikoides, Gymnodinium mikimotoi* (=*Karenia mikimotoi), G. sanguineum* (=*Akashiwo sanguinea), A. tamarense and H. akashiwo* (Jeong et al., 2000; Wang et al., 2007a). Other macroalgae which have been shown to inhibit the growth of HAB species include *Ecklonia kurome*, *Gracilaria lemaneiformis* and *Sargassum thunbergii* collectively displaying the ability to inhibit the growth of *A. tamarense, H. akashiwo, P. donghaiense, Amphidinium carterae and Scrippsiella trochoidea* (Nagayama et al., 2003; Liu et al., 2006; Wang et al., 2007a; Wang et al., 2007b; Lu et al., 2008). In many cases, impacts on HABs were observed from both co-culturing and from administering macroalgae extracts (Jeong et al., 2000; Jin et al., 2005; Wang et al., 2007a). More detailed studies have revealed that the isolation and offering of polyunsaturated fatty acids from *U. fasciata* and *U. pertusa* have effects on HABs similar to those observed for extracts and whole macroalgae, suggesting these compounds are active agents against the HAB species (Alamsjah et al., 2005; Alamsjah et al., 2008).

In Suffolk County, the Gobler lab has executed a series of culture and field experiments that have demonstrated that at least two species of macroalgae indigenous to Suffolk County can inhibit and prevent the occurrence of HABs: extracts of *Ulva rigida* / *Ulva lactuca* and *Porphyra*

purpurea. *Ulva* and *Porphyra* collected from Shinnecock Bay used at environmentally realistic levels (mg L^{-1}) were capable of lysing or strongly inhibiting the growth of seven different HAB species including several indigenous to Long Island such as *Aureococcus anophagefferens*, *Cochlodinium polykrikoides*, *Prorocentrum minimum* and *Pseudo-nitzschia multiseries* (Tang and Gobler, 2011; Tang et al., 2015). The dramatic allelopathic effects of extracts of dried and powdered macroalgae with and without post-extraction heat treatment demonstrated that the macroalgae contain heat-stable allelochemicals that play a major role in the observed allelopathic effects (Tang and Gobler, 2011; Tang et al., 2015). The addition of live *Ulva* thalli in bottle and mesocosm experiments conducted in the field during blooms of *A. anophagefferens* ('brown tide'; > 10^5 cells mL^{-1}) consistently yielded a significant (p<0.05) and often large (>50%) reduction in cell densities in ~48 h (Tang and Gobler, 2011). These findings combined with well-known nutrient removal capacity of seaweeds collectively suggest that use of macroalgae may be a promising mitigation strategy for HABs in coastal ecosystems.

AN OVERVIEW OF HAB MITIGATION AND PREVENTION STRATEGIES

As discussed in every section that appears above, watershed management of nitrogen loading will be the most effective, long-term solution for mitigating HABs in Suffolk County. Given that wastewater is the prime source of nitrogen to most Suffolk County water bodies, replacing old and aging septic systems that release most of their nitrogen to groundwater with innovative and alternative systems that remove a large fraction of nitrogen from wastewater streams before it enters groundwater is likely the most important step to reduce nitrogen loads and HABs in Suffolk County. It is expected that certain HABs will be more responsive to reduction in nitrogen than others based on the HAB-nitrogen linkages discussed in the above individual HAB sections. For example, given the strong responses of *Alexandrium* and *Dinophysis* to various sources of nitrogen these HABs may be the most responsive in terms of reducing nitrogen loads to mitigate these blooms. For example, PSP events that occurred annually in Northport Bay have ended since the upgrade of the sewage treatment plant (surface water discharger) in Northport Bay that was a primary source of nitrogen to these blooms. As discussed earlier, inorganic nutrient loading serves as the source of organic nutrients that fuel brown tides (LaRoche et al., 1997; Gobler and Sanudo-Wilhelmy, 2001a; Gobler et al., 2004b)

and, therefore, nitrogen reduction efforts would also aid in the mitigation of this HAB. Macroalgae blooms are known to be a direct consequence of nutrient overloading and thus, nitrogen mitigation should lessen the intensity and frequency of these events. *Cochlodinium* is known to be nutritionally flexible and can form blooms in open water environments with low nitrogen levels and thus may be less affected by reductions in nitrogen (Gobler et al., 2012). Importantly, however, even this dinoflagellate has shown more rapid growth when given higher nitrogen levels. Overall, long-term reductions in N loading might also influence the structure of phytoplankton and grazer communities in a favorable manner. Lower concentrations of N and P relative to Si could foster a nutrient regime which was more favorable for diatoms which are often readily grazed and support efficient food webs (Turner et al., 1998; Irigoien et al., 2002).

Within freshwater systems, nutrient reduction is also likely to be the most successful, long-term approach to reducing the incidence and intensity of harmful cyanobacterial blooms (Conley et al., 2009; Smith and Schindler, 2009). While phosphorus is the most common limiting element to blue-green algae, emerging evidence suggests that reduction of both nitrogen and phosphorus may be important for combating all types of blue-green algal blooms and their toxins (USEPA, 2015; Gobler et al., 2016), particularly in some Suffolk County water bodies (as described above; Gobler et al., 2007; Davis et al., 2009, 2010). As discussed in the brown tide section, blooms of *Aureococcus* have shown symptoms of phosphorus limitation and hence efforts to reduce phosphorus loads to SSER estuaries may minimize the intensity of brown tides in these systems.

Mitigation of HABs via biological controls: increasing water filtration capacity

Control of brown tide events by filter-feeding bivalves may be possible. The experimental results of Schaffner (1999) and Cerrato et al. (2004) indicated that elevated densities of *Mercenaria mercenaria* can effectively control *Aureococcus*. The reseeding of estuarine systems to the high densities found during the height of the hard clam fishery in Great South Bay would be logistically difficult and an expensive undertaking. However, such an effort on a smaller scale in concert with the establishment of "no take" marine preserve areas could eventually lead to the natural reestablishment of the former fishery, and concurrent benthic control of pelagic algal populations. Marine preserves consistently foster enhanced biomass and

diversity both within the preserve region and within neighboring regions (Halpern and Warner, 2002). The ability of hard clam larvae to settle and establish populations in regions surrounding the preserve area suggest that settled hard clams, both within the preserve and within adjacent areas, might eventually serve as a natural biocontrol in estuaries which are prone to brown tides. The ability to keep harmful algae in check via biological control can likely extend to other HABs as well. While such an effort was undertaken in Great South Bay by The Nature Conservancy with modest success and in Shinnecock Bay by Stony Brook's Shinnecock Bay Restoration Project which has led to newly recruited juvenile hard clams increases in densities.

Mitigation of HABs via physical flushing

The increased tidal exchange between estuaries which host brown tides and the coastal ocean may also mitigate the occurrence of blooms. Gobler et al (2005) suggested that a barrier-island breach could mitigate brown tides and the formation of the new inlet in Great South Bay has affirmed this hypothesis. Since being formed by Hurricane Sandy on October 29, 2012, the new inlet has radically transformed water quality in eastern Great South Bay, Narrow Bay, and western Moriches Bay, making these regions cooler in summer, warmer in winter, saltier, with increased water clarity, lower chlorophyll levels, lower nutrient levels, and lower densities of *Aureococcus* cells when blooms have occurred in Great South Bay (Gobler et al., in prep; SCDHS, 2012-2015). While all of Great South Bay has experienced more ocean flushing and higher salinity since the new inlet has formed, this has not discouraged brown tides as they have occurred annually in Great South Bay since 2012, although some of the events have been isolated to the fall only (2014 and 2016; Gobler et al., in prep; SCDHS, 2012-2015). It should be noted that some temporarily closed estuaries in the Town of Southampton are purposely dredged open on a regular basis by local governments in an effort to mitigate water levels and water quality (Gobler et al, 2005). Town of Southampton officials have publically discussed the possibility of constructing devices that would enhance physical flushing of brown tide prone estuaries (J. Schneiderman, pers. comm). Artificial mixing (Huisman et al., 2004; Visser et al., 2016) and flushing (Verspagen et al., 2006; Mitrovic et al., 2011) of lakes, may also suppress cyanobacterial populations. Notably, the water mixers, SolarBees, were used to mix Mill Pond in Water Mill and were shown to alter the composition of blue-green algal blooms, but were also

implicated in the occurrence of a massive fish kill in 2008 when Pond water levels dropped and devices mixed sediments and sediment nutrients into the water column (Gobler, 2010).

Targeted dredging to mitigate marine and freshwater HABs

Removal of bottom sediments by dredging is a mechanism for reducing the release of nutrients from the sediments and in some estuaries, sediments can be a major source of nutrients (Peterson, 1981). For example, the Peconic Estuary Program comprehensive management plan concluded that sediment fluxes were the largest source of nitrogen to the estuary (PEP, 2002) Similarly, studies of lakes in Suffolk County have concluded that sediments were the largest source of phosphorus (Harke et al., 2008; Gobler, 2010). Dredging sediments could also lead to the removal of cysts of HABs and increased flushing to aid in nutrient reduction.

Macroalgae aquaculture and harvest to mitigate nutrient loading and HABs

Beyond the experimental evidence which has already been amassed from around the globe demonstrating that many macroalgae can inhibit the growth of HABs (see Macroalgae that prevent HABs), there are many aspects of macroalgae that make them an attractive option for directly and indirectly mitigating HABs. Firstly, and perhaps most importantly, macroalgae are a natural component of coastal waters and thus their use as a control strategy would not involve the introduction of a non-native biotic or abiotic entity to estuarine ecosystems. Next, there is already a great precedent for the purposeful use and deployment of macroalgae in coastal waters as a nutrient mitigation strategy (Neori et al., 2004; Yang et al., 2006; Chopin et al., 2008; Barrington et al., 2009). In many cases, macroalgae are deployed simply to restrict nutrient concentrations and fluxes to coastal waters (Neori et al., 2004; Yang et al., 2006; Zhou et al., 2006). In other scenarios, macroalgae have become principal components of integrated multi-trophic aquaculture (IMTA) systems whereby macroalgae are cultivated with aquacultured fish and/or shellfish with the macroalgae often being harvested for commercial use and subsequent profit (Chopin et al., 2001; Schuenhoff et al., 2003; Fei, 2004; Kraemer et al., 2004; Neori et al., 2004; Chopin et al., 2008; Barrington et al., 2009). While such IMTA systems are not common to the US, it could have the combined benefits of reducing nutrients, lessening the occurrence of HABs, and being a prevention control and mitigation strategy which would render a profit, rather

than represent a cost. For example, since *Porphyra* spp. are the most commercially important macroalgae in the World and, since one species of this genus has already been shown to restrict the growth of HAB species (Tang et al., 2015), the cultivation of this seaweed could be of both economic and ecological benefit to coastal ecosystems. Moreover, there is a wealth of experience from Asia to draw on regarding the deployment of large regions of aquacultured macroalgae (Pereira and Yarish, 2008; Pereira and Yarish, 2009). As such, macroalgae could become an important part of future aquaculture programs which may expand in Suffolk County.

The direct harvest of natural stands of macroalgae could be a means for reducing nutrient loads and thus also mitigating HABs in Suffolk County. For example, research by the Gobler lab has determined that two species of macroalgae, *Ulva* and *Gracilaria* grow quickly and rapidly in Shinnecock Bay during the May through November and show great promise for nutrient removal. Macroalgae cover at least 10% of Shinnecock Bay and harvesting this weekly would generate up to 3,000 lbs of N per week. In addition to removing an environmental threat, this material from this industry-free estuary would generate a clean, non-polluting, low-cost, sustainable, renewable, product such as organic fertilizer.

During the past several years, Georgica Pond, East Hampton, NY, USA, has experienced a series of significant water quality impairments and human health threats including toxic cyanobacterial blooms, anoxia, kills of aquatic and terrestrial wildlife, and macroalgae blooms. In 2015, the precise nitrogen and phosphorus loading rates to Georgica Pond were quantified. At the same time, the hypothesis was put forth that the purposeful removal of macroalgae and aquatic plants from Georgica Pond could help improve water quality conditions. In 2016, an Aquatic Weed Harvester was deployed in Georgica Pond to remove the accumulation of macroalgae and aquatic plants across the entire system as a means of combating the delivery of nitrogen and phosphorus to the Pond and thus combating the late summer occurrence of toxic blue-green algae blooms. Harvesting began in June and persisted through early September with peak removal occurring during the months of July and August. A total of 55,740 lbs of macroalgae and aquatic plants was harvested through the season. This harvest represented only a small fraction of the annual nitrogen and phosphorus load to Georgica Pond (1% and 2%, respectively). Importantly, however, all of this harvest was concentrated between June 23 and September 8 of 2016. Prorating the harvest to these summer months, when freshwater delivery of nitrogen and phosphorus is minimal, this harvest represented up to 10% of the July-August

nitrogen load and 20% of the July-August phosphorus load. While the nitrogen reduction is still far below the 80% reduction suggested to meet target values, the 20% reduction in phosphorus was a significant portion of the 50% reduction suggested to meet US EPA target values for this element. Concurrent with the removal of the macroalgae and aquatic plants, environmental conditions in Georgica Pond during 2016 were significantly improved compared to prior years. Blue-green algae levels were an order of magnitude lower than the two prior years and these algae never bloomed to the exclusion of other algae. While Georgica Pond had the highest levels of blue-green algae in Suffolk County in August of 2014 and 2015, it had some of the lowest levels in 2016. In addition, unlike 2013 – 2015 when anoxia and mortality of wildlife occurred in Georgica Pond, in 2016 dissolved oxygen levels stayed above 4 mg L^{-1} throughout the summer and mortality of fish or other wildlife were not observed. While many inter-annual differences could have contributed to the improved environmental conditions in Georgica Pond in 2016, the significant reduction in nitrogen and phosphorus loading via the harvest of macroalgae and aquatic plants during the months when blue-green algae form blooms were likely to have played a key role in these improved conditions.

The sum of evidence suggests that while the dense overgrowth of macroalgae can be an ecosystem threat and a HAB, this is uncommon in Suffolk County. Instead, macroalgae may serve as an important ecosystem management strategy being grown in a mariculture setting while concurrently leading to the extraction of nutrients and direct mitigation of HABs.

Chemical mitigation of HABs

Given the highly destructive nature of ichthyotoxic *Cochlodinium* blooms, there has been significant interest in, and progress towards, developing techniques for the prevention, control, and, mitigation (PCM) of these events. In Korea, the spraying of fluidized clays has been employed by aquaculture fish farmers to prevent the rapid mortality of valuable, caged fish (Lee et al., 2008). Lee et al. (2008) specifically reported on rapid removal of nearly all (95%) *Cochlodinium* cells during blooms using a mixture of sophorolipid and yellow clay. Subsequent work by Song et al. (2010) demonstrated that dredged sediments of mixed compositions (slaked lime, quicklime, aluminum sludge bentonite, and zeolite) effectively removed lower *Cochlodinium* cell densities within an experimental setting, but less effectively removed higher

cell densities. The Gobler Lab demonstrated that wild populations and cultures of *Cochlodinium* could be removed from the water column at levels >90% with bentonite clays as well as polyaluminum chloride (Gobler, 2012). Beyond density-dependent removal rates, a second concern with regard to clay applications is the ability of *Cochlodinium* to reform blooms once removed by clay. For example, Lee et al. (2009) demonstrated that a fraction of *Cochlodinium* cells survive precipitation and sinking caused by yellow loess clays and may reform substantial cell densities (>2,500 cells ml^{-1}) within weeks of removal. *Cochlodinium*, however, is likely the best test subject for this type of mitigation strategy given its highly patchy and visible nature, thus using clays on other algal blooms common to Long Island would likely be ineffective. Studies exploring the effectiveness of clay as a mitigation strategy against *Cochlodinium* blooms have not been explored at length beyond those in Korea.

Some lakes have been treated with clays, aluminum, or 'Phoslock®' to bind phosphate and coagulate with cyanobacterial cells and causing their sedimentation (Robb et al., 2003; Lürling and van Oosterhout, 2013). The Town of Southampton tried Phoslock to mitigate blue-green algal blooms in Mill Pond, Water Mill, in 2013, but the blooms that year were as intense or more intense than prior years. This can lead to unbalanced N:P ratios as Phoslock can cause phosphorus limitation and therefore may promote more toxic cyanobacterial blooms. Other chemicals such as copper have been used as algicides but are broadly toxic to a wide range of organisms and can be persistent within sediment (Griffiths and Saker, 2003).   However, barley straw may be a promising mitigation strategy as it has been found to inhibit the growth of *Anabaena, Microcystis* and *Aphanizomenon* (Rajabi et al., 2010).

Hydrogen peroxide (H_2O_2) is a naturally occurring compound in aquatic ecosystems being generated by all photosynthetic organisms and via the photochemical reactions with dissolved organic matter (O'Sullivan et al., 2005). While all living organisms are sensitive to hydrogen peroxide, cyanobacteria are more sensitive than other, eukaryotic phytoplankton (Drabkova et al., 2007). Research in the Netherlands has demonstrated that the application of 2 – 5 mg L^{-1} of hydrogen peroxide can mitigate blooms of blue-green algae in an ecosystem setting while having no notable effect on the food web and allowing other phytoplankton to thrive (Matthijs et al., 2012; Matthijs et al., 2016). Another study in the Netherlands (Burson et al., 2014), the first application in a marine system, demonstrated that the application of 50 mg L^{-1} of hydrogen peroxide to an *Alexandrium ostenfeldii* bloom decreased cell densities >98% after 48

hours of application, and 96 hours after application toxin levels fell below regulatory limits. While total phytoplankton biomass decreased significantly, the community composition shifted from one dominated by *Alexandrium* to one dominated by chlorophytes, euglenophytes and *Anabaena* spp.; zooplankton decreased, and larger macroinvertebrates and fish showed little effect (Burson et al., 2014). While hydrogen peroxide may be a useful mitigation strategy for marine HABs, further and targeted testing would need to be conducted before in situ applications could be performed.
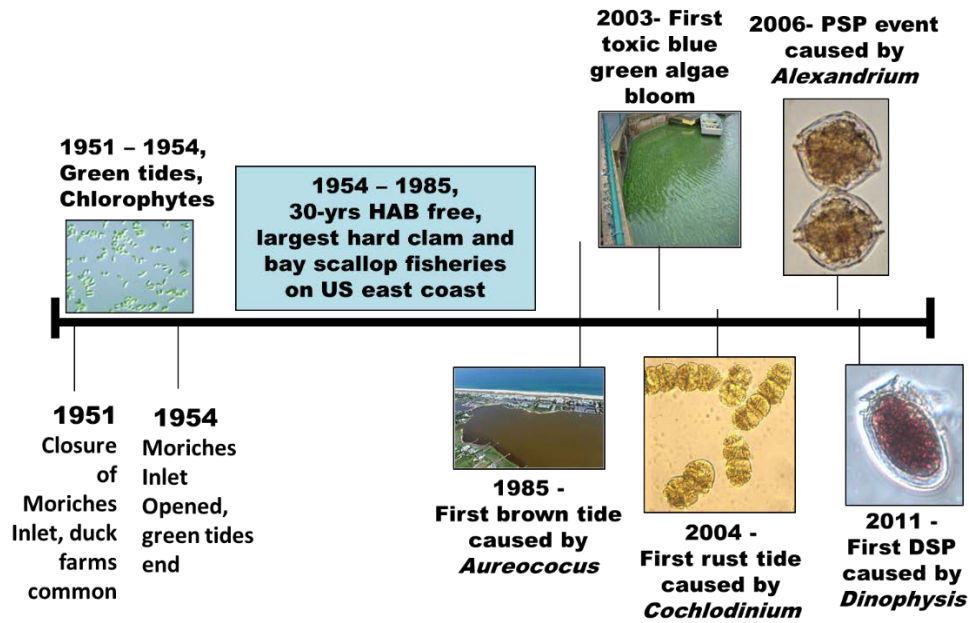
History of HABs on Long Island

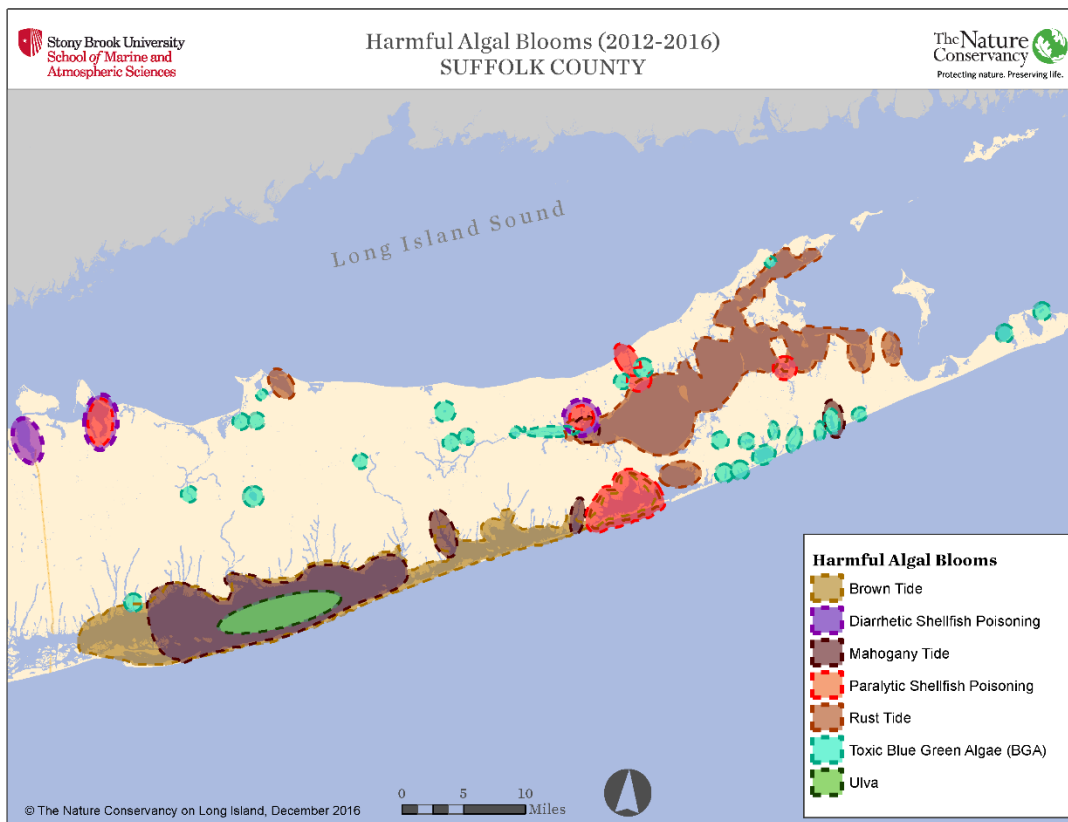**Figure 1.** A history of harmful algal blooms across Long Island, New York.

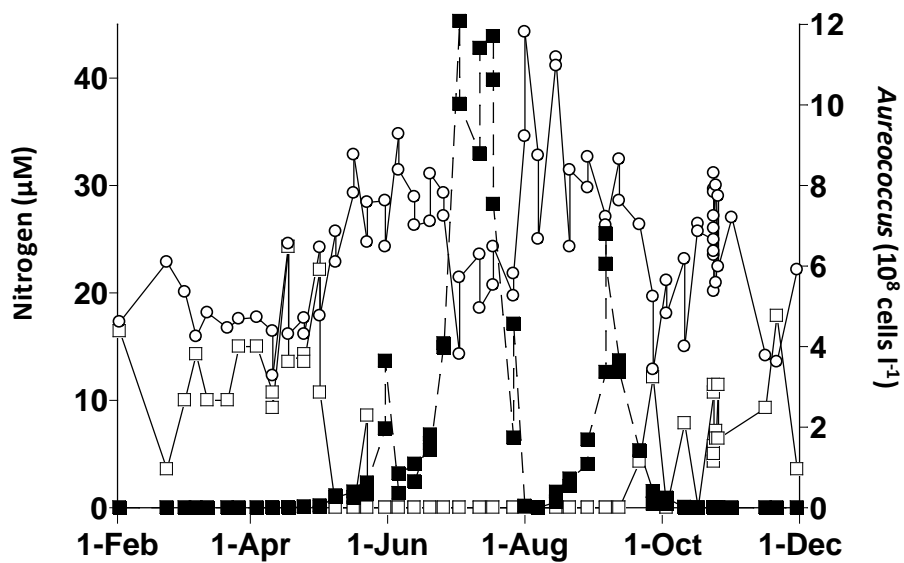**Figure 2.** Location of harmful algal blooms across Suffolk County from 2012-2016.

Figure 3. Concentrations of nitrate plus nitrite (x10 □), dissolved organic nitrogen (○), and *A. anophagefferens* cells (■) in surface waters of Flanders Bay, Long Island, NY, USA, 1995. Figure from Sunda et al. 2006, based on data supplied by the Suffolk County Department of Health Services.
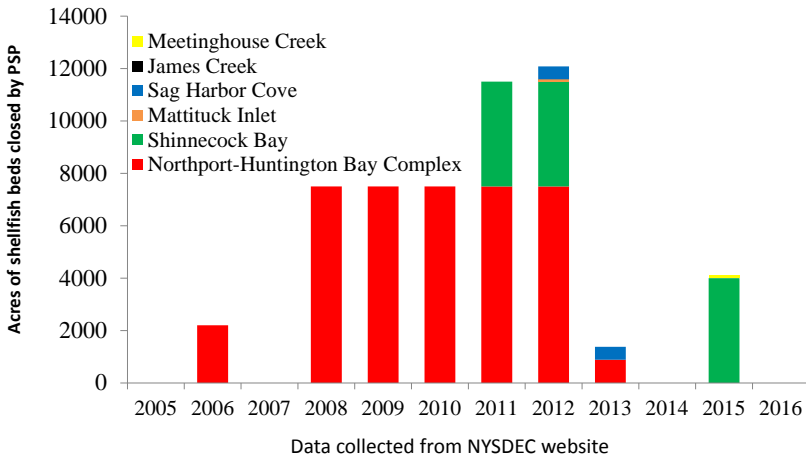
Figure 4. The expansion of PSP- induced shellfish bed closures (in acres) on Long Island, NY from 2005 to 2016. Data collected for NYSDEC website.

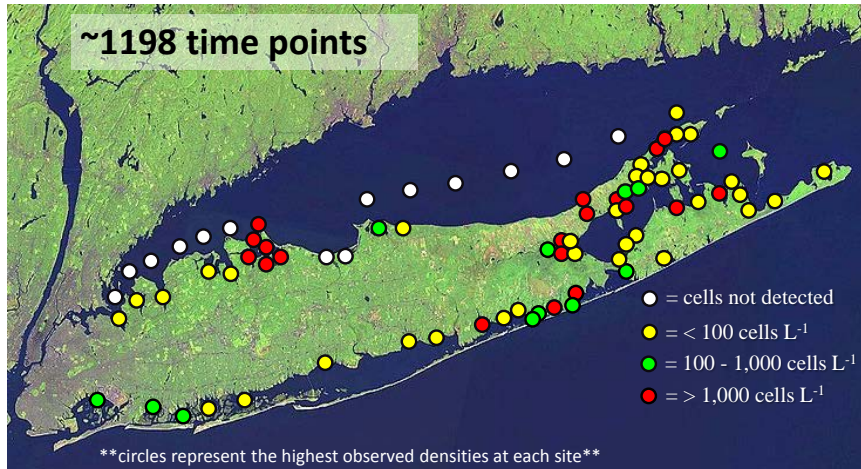## Presence of PSP-producing *Alexandrium* in LI: 2007-2016

Figure 5. The distribution of PSP-producing *Alexandrium fundyense* in Long Island embayments. Circles indicate the highest observed densities of *Alexandrium* (cells L^{-1}) found at each site during 2007-2016 (n=1198).

Changes in Northport Bay STP effluent
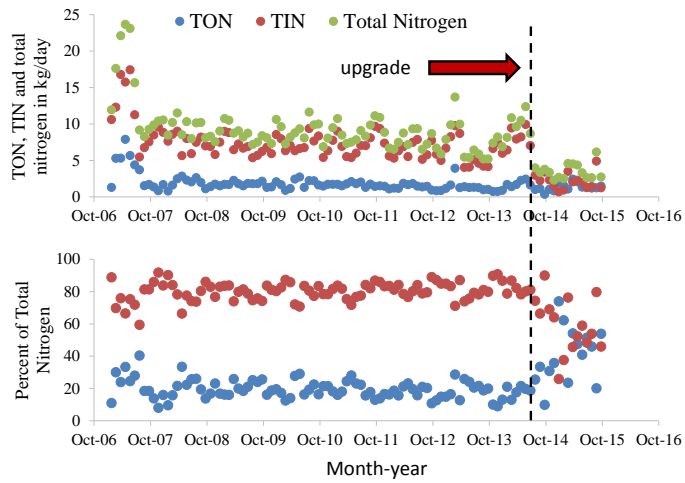
Figure 6. Changes in Northport Bay Sewage Treatment Plant over from 2006-2016. TON=total organic nitrogen, TIN= total inorganic nitrogen.
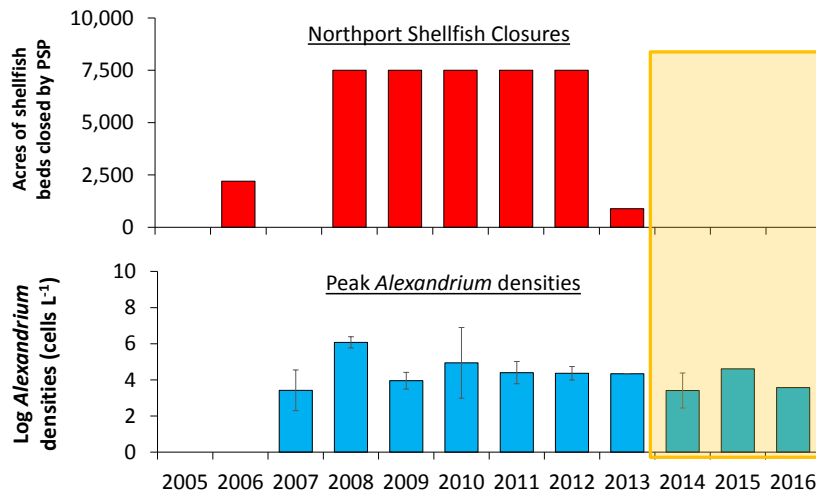
Figure 7. Northport Bay Acreage of Shellfish Bed Closures and Peak *Alexandrium* (cells L^{-1}) densities from 2007-2016. Yellow box emphasizes the years post-sewage treatment plant upgrade when there were no closures in this system. Note: There was no *Alexandrium* monitoring program prior to 2007.

Presence of DSP-producing *Dinophysis* in LIS: 2011-2016
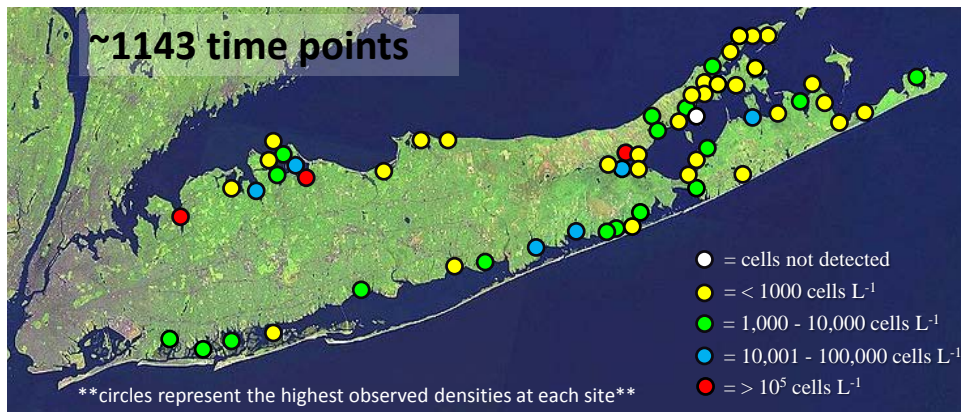
Figure 8. The distribution of DSP-producing *Dinophysis acuminata* in Long Island embayments. Circles indicate the highest observed densities of *Dinophysis* (cells L^{-1}) found at each site during 2008-2016 (n=1143).
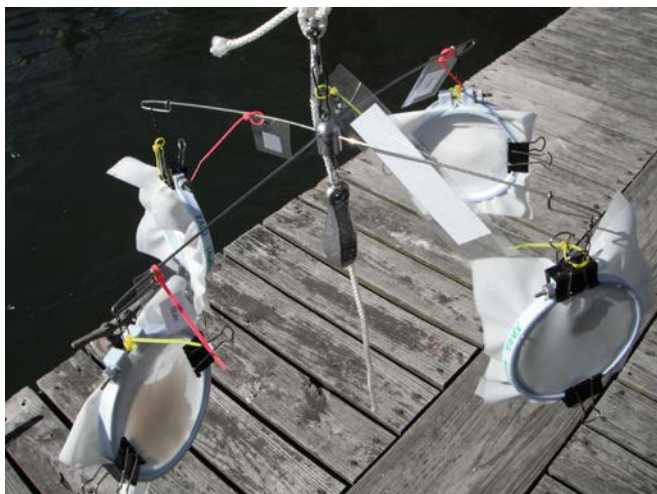
Figure 9. SPATT (solid-phase adsorption toxin tracking) samplers deployed in Northport Harbor at site 2 (Britannia) and site 8 (Woodbine) during spring 2012.

Figure 10. Mean levels of the cyanotoxin, microcystin, in 20 Suffolk County lakes, 2004 – 2007.

Figure 11. Number of lakes listed for blue green algae by county in NYSDEC in 2015. 62 Counties in NYS; 45 counties with blue green algal blooms

Figure 12. Top 10 lakes in NYS, blue green algae listings, NYSDEC 2015.

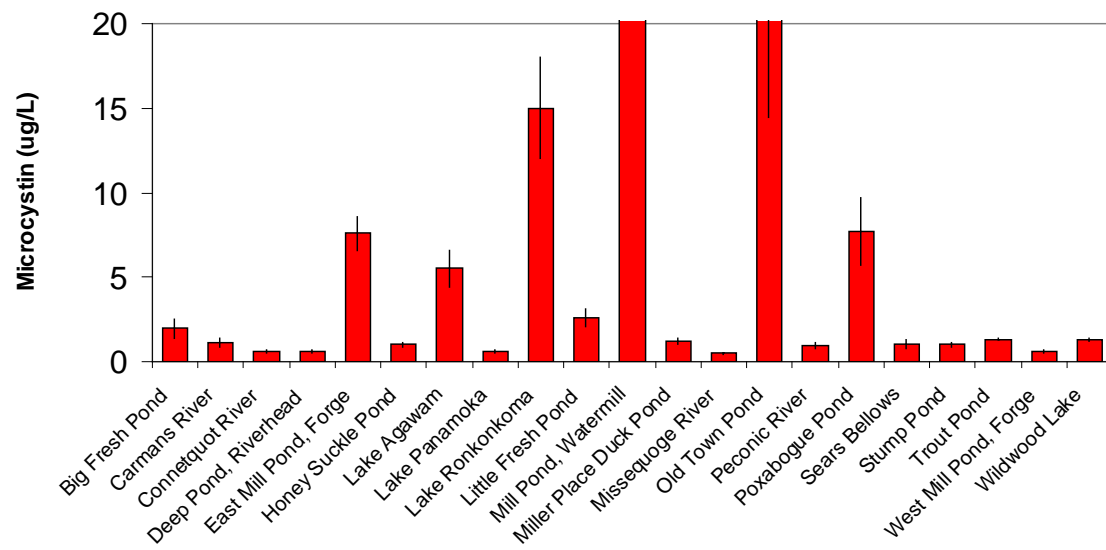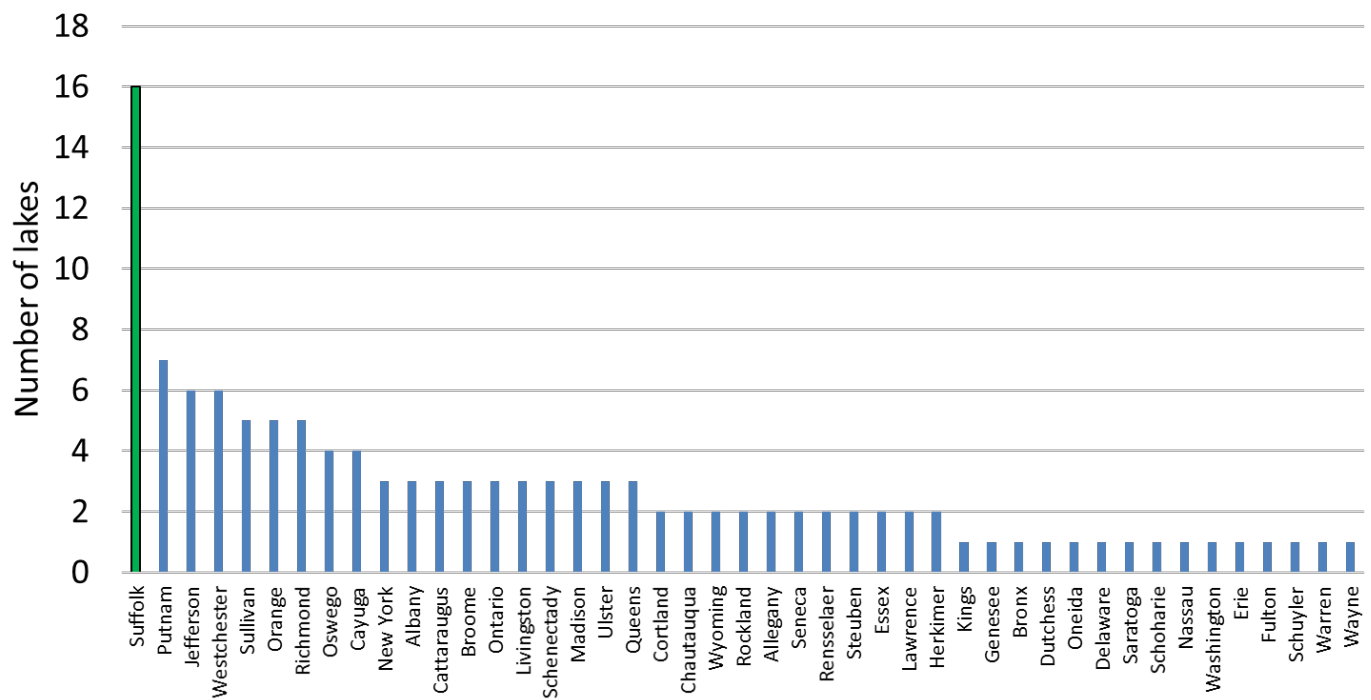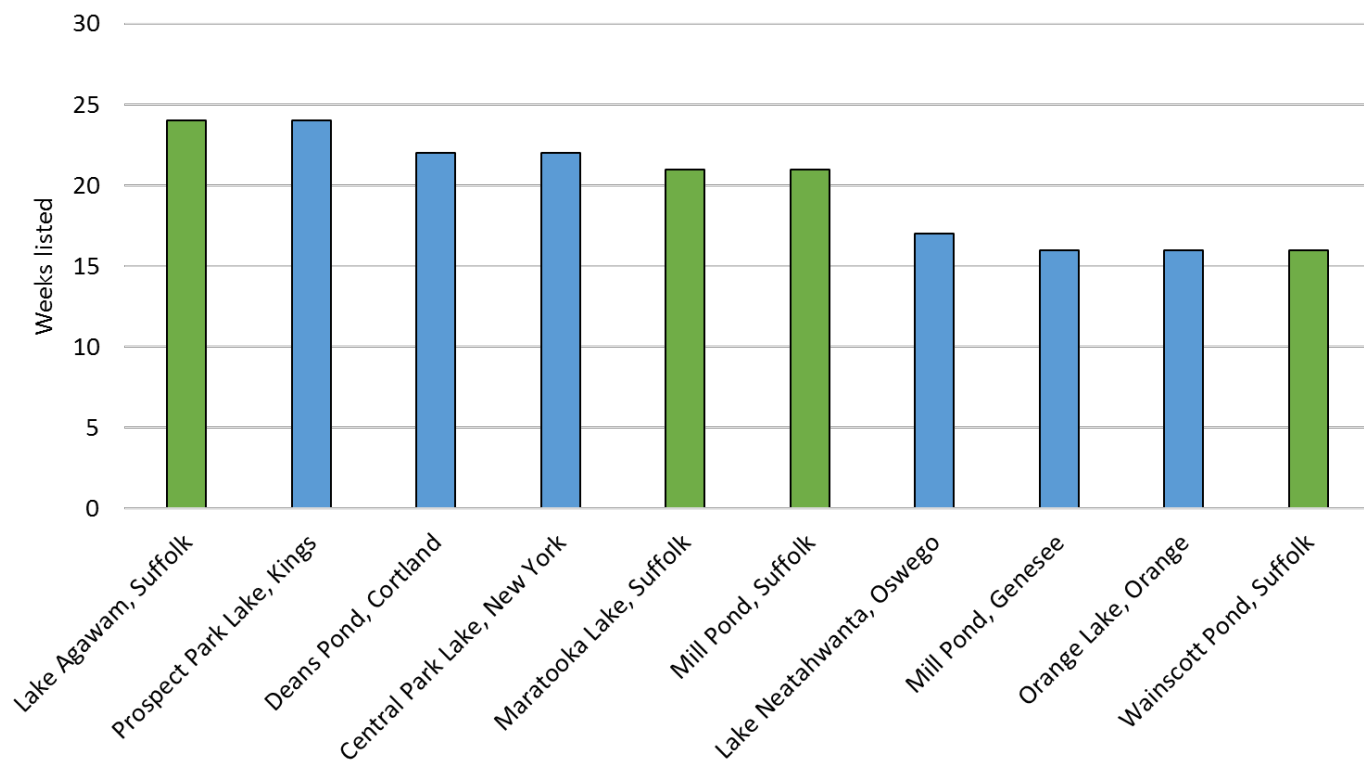| Common name	Genus species	Class of Algae	Toxin	Name of human health syndrome	Symptoms/Impact	Cell size/ general morphology	Bloom appearance	Bloom Season	Recent location of blooms in Suffolk County
Red tide	*Alexandrium fundyense*	Dinoflagellate	saxitoxin	Paralytic Shellfish Poisoning	Potent neurotoxins act on sodium channels causing numbness and tingling sensation in mouth and extremities; rapid onset (30 min); can cause death due to respiratory failure	25-40 microns; single cells or in chains	Despite its name it is usually not visible	Spring; March-June	PSP closures have occurred within Northport Bay, Huntington Bay, Shinnecock Bay, Meetinghouse Creek, Terry Creek, Sag Harbor Cove, Mattituck Inlet, James Creek
Red tide	*Dinophysis acuminata*	Dinoflagellate	okadaic acid and dinophysistoxins	Diarrhetic Shellfish Poisoning	Causes diarrhea, nausea, vomitting, dehydration; rapid onset (~3h); rapid resolution (2-3 days)	30-50 microns; single cells	Despite its name it is usually not visible	Spring/Summer; April-July	Dense blooms and shellfish with DSP toxins observed in: Northport Bay, Meetinghouse Creek, Cold Spring Harbor, Hempstead Harbor
Brown Tide	*Aureococcus anophagefferens*	Pelagophyte	Unconfirmed; related to extracellular polysaccharide	not toxic to humans	Responsible for the destruction of seagrass beds and decline in scallop and hard clam populations	2-3 microns; single cells	Forms uniformly chocolate brown water (not patchy)	Spring/Summer/Fall; May-July; Sept-Dec	Great South Bay, Moriches Bay, Quantuck Bay, Shinnecock Bay (Peconic Estuary 1985-1995)
Rust Tide	*Cochlodinium polykrikoides*	Dinoflagellate	Unconfirmed; similar to reactive oxygen species (ROS)	not toxic to humans	Ichthyotoxic (kills fish); known to kill impounded fish, as well as bivalves, and copepods	40 microns; chains of up to 8 cells	Forms large, rust-colored, streaky patches	Summer/Fall; July-October	Shinnecock Bay, Peconic Estuary; has occurred in Great South Bay, Port Jefferson Harbor

Cyanobacterial / Blue-green algal blooms	*Microcystis* spp.; *Anabaena* spp.; *Aphanizomenon* spp.	Cyanobacteria	Microcystins, Anatoxin-a	Neutrotoxicity, gastrointestinal toxins	Microcystin is a hepatotoxin that can affect the liver of humans and animals; blooms can cause fish and/or animal kills; can cause gastrointestinal cancers	individual cells are 3-6 microns but form large colonies (*Microcystis*) or chains (*Anabaena*)	Green surface slicks	Spring/Summer/Fall; May - November	More than twenty freshwater lakes including Lake Ronkonkoma, Lake Agawam, Mill Pond, Georgica Pond, Maratooka Lake
Green tides / Macroalgal blooms / Sea lettuce	*Ulva* spp., *Enteromorpha* spp., *Cladophora* spp.	Chlorophyta	Hydrogen sulfide; contributes to hypoxia	not toxic to humans	Overgrowth can cover benthic habitats and promote hypoxia/anoxia; beaching and decay of macroalgae can cause sulfidic, unpleasant smell	Centimeters, up to a foot in length	Large growths of green "lettuce-like" algae on bay bottoms or washed onto coastlines	Summer months	Blooms have occurred in Great South Bay, Shinnecock Bay; limited beachings on Southampton ocean beaches

Table 1. Summary of harmful algal blooms found on Long Island.

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Table 2. Year, location, maximum shellfish toxicity and maximum *Alexandrium* densities across Long Island. [a] from Nuzzi and Waters 1993.

Year	Location	Maximum shellfish toxicity (µg STX eq/100g shellfish tissue)	Maximum *Alexandrium* densities (cells L-1)	Closure implemented?	Acres
1986	Reeves Bay	190	14,000 (20-May)[a]	no	n/a
2006	Northport-Huntington Bay	137	n/a	yes	2200
2008	Northport-Huntington Bay	1432	1,199,566 (16-May)	yes	7500
2009	Northport-Huntington Bay	92	9,030 (27-May)	yes	7500
2010	Northport-Huntington Bay	430	88,466 (14-May)	yes	7500
2011	Northport-Huntington Bay	207	25,298 (9-May)	yes	7500
2011	Shinnecock Bay	54	49,042 (27-Apr)	yes	4000
2012	Northport-Huntington Bay	840	23,233 (7-May)	yes	7500
2012	Shinnecock Bay	56	331 (20-Apr)	yes	4000
2012	Mattituck Inlet	<36	2,471 (25-May)	yes	92
2012	Sag Harbor Cove	<37	3,495 (17-Apr)	yes	490
2012	Meetinghouse Creek	380	17,206 (11-Apr)	no	
2013	Northport-Huntington Bay	600	21,875 (8-May)	yes	887
2013	Sag Harbor Cove	62	250 (1-May)	yes	490
2015	Shinnecock Bay	180	209 (18-May)	yes	4000
2015	James Creek	350	13,930 (15-May)	yes	31
2015	Meetinghouse Creek	540	46,690 (29-April)	yes	100

Table 3. DSP toxins in New York (Long Island) shellfish measured via multiple extraction (EU vs Abraxis, NOAA) and analysis methods (LCMS, PP2A). Sites as in Hattenrath-Lehmann et al. 2013 and Hatenrath-Lehmann et al. submitted.

Bay	Site	Date	Species	Total DSP ng/g			
				PP2A		LCMS	
				EU extraction	Abraxis extraction	EU extraction	NOAA extraction
Northport Bay	S3	6/28/2010	*Mytilus edulis*				52
	S4	6/28/2010	*Mytilus edulis*				115
	S7	6/20/2011	*Mytilus edulis*				143
	S3	**6/28/2011**	***Mytilus edulis***				**1245**
	S5	**7/6/2011**	***Mytilus edulis***				**165**
	S6	7/6/2011	*Mytilus edulis*				37
	S1	**7/7/2011**	***Mya arenaria***				**957**
	S2	**7/7/2011**	***Mya arenaria***				**1089**
	S2	**7/7/2011**	***Geukensia demissa***				**1137**
	S1	7/2/2014	*Geukensia demissa*	<63		19	
	S1	7/2/2014	*Mya arenaria*	<63	<63	22	
Cold Spring Harbor	**CSH1**	**7/10/2013**	***Mytilus edulis***			**168**	
	CSH1	7/10/2013	*Crassostrea virginica*			25	
	CSH1	7/10/2013	*Mya arenaria*			78	
	CSH1	**7/7/2014**	***Geukensia demissa***	**202**	**176**	**149**	
	CSH1	7/14/2014	*Geukensia demissa*	73	69	39	
	CSH1	7/21/2014	*Mytilus edulis*	96	90	73	
	CSH1	7/21/2014	*Geukensia demissa*	<63		23	
	CSH2	7/7/2014	*Mytilus edulis*	81			
	CSH2	7/14/2014	*Mytilus edulis*	89			

Table 4. Highest and mean levels of phycocyanin (blue green algal pigment) in µg L^{-1} and microcystin (µg L^{-1}).

Name of Lake	Highest Bluegreen	Date of Highest Bluegreen	Highest Microcystin	Date of Highest Microcystin	Mean Bluegreen	Mean Microcystin
Spring Lake Golf Course	26,754	6/30/2016	141.27	6/30/2016	13,442	71.60
Lake Agawam	16,470	8/30/2016	1,836.84	8/30/2016	638	72.61
Roth Pond	1,057	8/25/2016	97.58	8/5/2013	342	18.09
Maratooka Lake	858	6/27/2016	242.53	6/22/2015	102	25.79
Setauket Mill Pond	715	6/23/2016	0.38	6/23/2016	322	0.20
Big Reed Pond	593	4/19/2015	11.95	8/7/2014	202	1.84
Meadow Brook Pointe- Mill Pond Golf Course	570	9/20/2016	0.32	9/20/2016	570	0.32
Lake Ronkonkoma - Brookhaven Town Beach	560	6/15/2016	60.52	6/23/2014	34	5.68
Wainscott Pond	500	8/31/2016	7.66	10/23/2015	168	0.79
Old Town Pond	472	7/7/2016	6.68	7/7/2016	80	0.84
Georgica Pond	445	8/25/2015	19.47	9/8/2016	62	0.98
Mill Pond	428	10/26/2016	377.46	7/15/2014	99	15.33
Forge Road	406	7/6/2016	9.52	7/6/2016	205	9.52
Lake Capri	325	9/4/2014	4.47	9/4/2014	325	4.47
Sagaponack Pond	295	8/17/2016	0.46	8/19/2015	60	0.17
Willowbrook Lake	256	8/29/2016	0.71	8/29/2016	209	0.67
Peconic River	254	8/12/2016	0.18	8/12/2016	87	0.18
Wickapauge Pond	240	6/25/2014	553.35	7/9/2014	30	57.17
Lake McKay	201	8/12/2015	5.12	8/12/2015	186	3.45
Niger Pond, Otis Pike Preserve		8/17/2016	0.05	9/6/2016		0.03

	199				148	
Kellis Pond	151	8/13/2015	3.9	8/19/2015	44	0.60
Stony Brook Duck Pond	136	7/18/2016	2.69	7/18/2016	111	1.38
Upper Mills Pond	114	7/6/2016	1.55	7/6/2016	114	1.55
Fort Pond East Hampton	92	7/29/2015	4.03	7/29/2015	18	0.54
East Marion Lake	56	8/8/2016	1.16	8/8/2016	56	1.16
Mecox Bay	48	8/18/2016	0.24	8/31/2016	21	0.18
Hook Pond	44	8/10/2016	3.29	9/8/2016	14	0.87
Lake Ronkonkoma - Islip Town Beach	43	8/27/2015	3.74	6/8/2016	19	0.45
Laurel Lake	39	4/29/2016	3.15	4/29/2016	5	3.15
Blydenburg Lake	38	9/13/2016	0.08	9/13/2016	38	0.08
Fort Pond Montauk	34	9/21/2015	3.65	9/26/2016	12	1.24
Fort Pond Boat Ramp	27	9/21/2015	0.25	9/21/2015	27	0.25
Poxabogue Pond	23	7/16/2015			23	0
Coopers Neck Pond	21	7/16/2015			21	0
Stony Brook Mill Pond	20	8/5/2015			15	0
Old Field Mill Pond	17	8/11/2015			11	0
Little Fresh Pond	16	9/9/2015			6	0
Cedar Point, Southold	15	8/16/2016			15	0
Loves Pond, Sunken Meadow Park	12	5/10/2016			12	0
Fresh Pond	11	6/27/2016			6	0
Stump Pond		8/3/2015				0

	11				8	
Deep Pond	10	8/31/2015			4	0
Channel Pond	7	7/16/2015			7	0
Great Pond	6	8/24/2015			3	0
Preston Pond, Otis Pike Preserve	5	8/17/2016			5	0
West Mill Pond	4	8/14/2015			4	0
Big Fresh Pond	3	9/15/2014			1	0
Stony Brook Harbor	3	8/17/2015			1	0
Wildwood Lake	3	8/31/2015			1	0
Southards Pond	3	8/20/2015			3	0
Bellow Pond	3	9/3/2014			1	0
Lake Panamoka	2	9/22/2016			2	0
Sherwood Lake, Islip	2	7/1/2016			2	0
Lake Arrowhead	2	6/22/2015			1	0
Kaler's Pond	2	8/14/2015			2	0
West Lake	2	8/14/2015			2	0
Swan Lake	2	8/14/2015			2	0
Argyle Lake	1	8/20/2015			1	0
Great Patchogue Lake	1	8/14/2015			1	0
Hashamomuck Pond	1	8/10/2015			1	0
Shelter Island	0	8/11/2014			0	0
Oakdale		8/31/2016				0

	-				-	
Wolf Pit Pond	-	6/13/2016			-	0

LIST OF ACRONYMS

CCMP- The Provasoli-Guillard National Center for the Culture of Marine Phytoplankton

DIN- dissolved inorganic nitrogen

DOC- dissolved organic carbon

DOM- dissolved organic matter

DON- dissolved organic nitrogen

DOP- dissolved organic phosphorus

DSP- diarrhetic shellfish poisoning

EPS- extracellular polysaccharide

HAB- harmful algal bloom

HABHRCA- Harmful algal bloom and hypoxia research and control act

LIS- Long Island Sound

N- Nitrogen

NSSP- National Shellfish Sanitation Program

NYSDEC- New York State Department of Environmental Conservation

P- Phosphorus

PSP- paralytic shellfish poisoning

ROS- reactive oxygen species

SCDHS- Suffolk County Department of Health Services

TIN- total inorganic nitrogen

TON- total organic nitrogen

GLOSSARY

Autotroph- any organism capable of self-nourishment by using inorganic materials as a source of nutrition. For example, plants and phytoplankton

Axenic- indication that a culture is free of all other living organisms other than the cultured species itself.

Eutrophication- the over enrichment of a water body with nutrients

Heterotroph- an organism that cannot fix carbon from inorganic sources (like an autotroph can) of nutrients but must rely on organic forms of carbon for growth. For example, humans.

Mixotroph- an organism that can have multiple modes of nutrition, for example a mix of autotrophy and heterotrophy.

Photosynthesis- the process by which organisms (plants, phytoplankton) use sunlight to synthesize food from carbon dioxide and water.

Ocean acidification- the decrease of pH (hydrogen ions) in the Earth's oceans caused by the uptake of carbon dioxide from the Earth's atmosphere.

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