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Positive feedbacks between bottom-up and top-down controls promote the formation and toxicity of ecosystem disruptive algal blooms: A modeling study

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ABSTRACT

Harmful algal blooms that disrupt and degrade ecosystems (ecosystem disruptive algal blooms, EDABs) are occurring with greater frequency and severity with eutrophication and other adverse anthropogenic alterations of coastal systems. EDAB events have been hypothesized to be caused by positive feedback interactions involving differential growth of competing algal species, low grazing mortality rates on EDAB species, and resulting decreases in nutrient inputs from grazer-mediated nutrient cycling as the EDAB event progresses. Here we develop a stoichiometric nutrient–phytoplankton–zooplankton (NPZ) model to test a conceptual positive feedback mechanism linked to increased cell toxicity and resultant decreases in grazing mortality rates in EDAB species under nutrient limitation of growth rate. As our model EDAB alga, we chose the slow-growing, toxic dinoflagellate *Karenia brevis*, whose toxin levels have been shown to increase with nutrient (nitrogen) limitation of specific growth rate. This species was competed with two high-nutrient adapted, faster-growing diatoms (*Thalassiosira pseudonana* and *Thalassiosira weissflogii*) using recently published data for relationships among nutrient (ammonium) concentration, carbon normalized ammonium uptake rates, cellular nitrogen:carbon (N:C) ratios, and specific growth rate. The model results support the proposed positive feedback mechanism for EDAB formation and toxicity. In all cases the toxic bloom was preceded by one or more pre-blooms of fast-growing diatoms, which drew dissolved nutrients to low growth rate-limiting levels, and stimulated the population growth of zooplankton grazers. Low specific grazing rates on the toxic, nutrient-limited EDAB species then promoted the population growth of this species, which further decreased grazing rates, grazing-linked nutrient recycling, nutrient concentrations, and algal specific growth rates. The nutrient limitation of growth rate further increased toxin concentrations in the EDAB algae, which further decreased grazing-linked nutrient recycling rates and nutrient concentrations, and caused an even greater nutrient limitation of growth rate and even higher toxin levels in the EDAB algae. This chain of interactions represented a positive feedback that resulted in the formation of a high-biomass toxic bloom, with low, nutrient-limited specific growth rates and associated high cellular C:N and toxin:C ratios. Together the elevated C:N and toxin:C ratios in the EDAB algae resulted in very high bloom toxicity. The positive feedbacks and resulting bloom formation and toxicity were increased by long water residence times, which increased the relative importance of grazing-linked nutrient recycling to the overall supply of limiting nutrient (N).

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1. Introduction

Harmful algal blooms (HABs) have occurred with increasing frequency in recent years with eutrophication and other anthropogenic alterations of coastal ecosystems (Glibert et al., 2005; Heisler et al., 2008). Many of these harmful blooms severely alter or degrade ecosystem function, and have been referred to as ecosystem disruptive algal blooms (EDABs) (Sunda et al., 2006). These blooms are often caused by toxic or unpalatable species that decrease grazing rates by herbivores, and thereby disrupt the transfer of nutrients and energy to higher trophic levels and decrease nutrient cycling. EDAB species include brown tide pelagophytes, and toxic haptophytes, cyanobacteria, and dinoflagellates among others (Sunda et al., 2006).

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Both algal nutrient availability and herbivore grazing are known to influence the dynamics of EDAB events and algal blooms in general (Calbet et al., 2003; Irigoien et al., 2005; Buskey, 2008; Strom et al., 2013). Potential interactions between these two factors have also been noted because nutrient limitation of algal prey can increase the grazing defenses in phytoplankton (Graneli and Flynn, 2006; Sunda et al., 2006) and thereby decrease the population growth and nutrient recycling rates of zooplankton which are linked to grazing rates (Sterner and Elser, 2002; Mitra and Flynn, 2005).

Sunda et al. (2006) presented a conceptual model for the development of EDABs based on positive feedback interactions among algal growth competition for limiting nutrients, low herbivore grazing rates on EDAB species, and grazing-linked nutrient regeneration. This model was based on the hypothesis of fundamental tradeoffs between functional traits that promote high rates of nutrient acquisition, growth and reproduction in an algal species and traits that promote low grazing mortality rates (large cell size, toxicity, and unpalitability) (Yoshida et al., 2003; Litchman et al., 2007; Sunda and Hardison, 2010). Due to these tradeoffs species with high maximum specific growth rates (high-nutrient adapted, r-selected species) were also susceptible to high rates of grazing mortality, while low-nutrient-adapted, *K*-selected species were well defended against grazing, but had much lower maximum growth rates. Thus, each set of species is adapted for optimal net population growth under different sets of nutrient and grazing conditions.

In their conceptual model Sunda et al. (2006) proposed that nutrient inputs first stimulated blooms of fast-growing, readily-grazed, r-selected species (e.g., diatoms) which drove dissolved nutrients to low concentrations and promoted the growth of zooplankton. This set the stage for population growth of *K*-selected EDAB species, which were better adapted for growth at low nutrient levels and were well defended from zooplankton grazing due to their toxicity or low palatability. This combination of traits allowed the EDAB species to proliferate at the expense of the r-selected species, whose growth rate was greatly reduced at low nutrient concentrations and were less well-defended against grazing. Sunda et al. (2006) noted that the increased biomass of the poorly-grazed EDAB species would cause zooplankton grazing rates to decline, which would decrease grazing linked nutrient recycling and further decrease limiting nutrient concentrations, and thereby further promote population growth of the EDAB species at the expense of competing algae. The authors proposed that these effects constituted an inherent positive feedback that should promote the development of harmful blooms (Sunda et al., 2006). They also noted that there would be an additional EDAB-promoting positive feedback in cases where the toxicity and related grazing defenses of the EDAB species increased with nutrient limitation of growth rate, which commonly occurs in toxic algae (Sunda et al., 2006; Graneli and Flynn, 2006).

Previously, Sunda and Shertzer (2012) developed a stoichiometric nutrient–phytoplankton–zooplankton (NPZ) model to test the positive feedback hypothesis of Sunda et al. (2006) for the development of EDAB events where the EDAB species had high levels of the grazing defenses, which did not vary with nutrient limitation of growth rate. In the present model we examined the effect of an additional positive feedback associated with increases cellular toxins with nutrient limitation of growth rate of the EDAB species (Sunda et al., 2006). Such increases in cellular toxicity with nutrient limitation were proposed to substantially promote both the occurrence and toxicity of EDAB events (Mitra and Flynn, 2006; Sunda et al., 2006).

In the present model the slow growing, toxic EDAB species *Karenia brevis* (K.b.) was competed with two faster growing, less well defended diatom species (*Thalassiosira pseudonana* [T.p.] and *T. weissflogii* [T.w.]). The selection of K.b. as our model EDAB species is supported by its nearly annual formation of toxic red tide blooms in the Gulf of Mexico, which adversely affect human health, marine ecosystems, and coastal economies (Flewelling et al., 2005; Kirkpatrick et al., 2006; Watkins et al., 2008). K.b. was also chosen because of the recent publication of quantitative relationships between nutrient (N and phosphorus [P]) limitation of specific growth rate and increased ratios of cellular toxins (brevetoxins) to cell carbon (C) in this species (Hardison et al., 2012, 2013). These relationships provided the data needed to construct an NPZ model that directly examines the effect of increased cellular toxins under N-limitation of growth rate on the occurrence and toxicity of EDAB events.

Another major reason for the choice of the above three algal species in our stoichiometric NPZ model was the recent availability of data describing relationships among nutrient (NH_4^+) concentration, NH_4^+ uptake rates, cellular N:C ratios, and specific growth rate in these and other algal species (Sunda and Hardison, 2007, 2010; Sunda et al., 2009; Hardison et al., 2012). These data were used to compute growth competition for NH_4^+ among the three species. Due to analytical difficulties, no equivalent growth versus nutrient data are available for any other N-substrates or for phosphate, which precluded the inclusion of other N-substrates such as nitrate or dissolved organic N (DON) in our model.

In addition, NH_4^+ was chosen as the limiting nutrient for several other reasons. Nitrogen is the primary limiting nutrient in coastal waters and stratified ocean waters (Ryther and Dunstan, 1971; Sanders et al., 1987; Litchman et al., 2007; Moore et al., 2013) and NH_4^+ is frequently the primary limiting inorganic N species in N-limited marine waters (Harrison et al., 1996). Also NH_4^+ is a major form of regenerated N (Dugdale and Goering, 1967; Verity, 1985), and thus, is an essential component of NPZ models (like this one) that include grazer-mediated N-regeneration.

A central process in our NPZ model is differential grazing by zooplankton, which removes individual algal species at varying rates, and thus, along with differing growth rates, controls the abundance and species composition of the algal community. Algal grazing supports the growth and reproduction rate of the grazing predator and has another critical function: it facilitates the recycling of nutrients through excretion. Therefore, it is essential for resupplying the nutrient (NH_4^+) pool, especially during periods of low external nutrient supply.

In the present model, the toxin content and associated grazing defenses in our model EDAB alga (K.b.) increased with N-limitation of specific growth rate. K.b. produces a suite of structurally related neurotoxins (brevetoxins), which activate voltage-gated sodium channels in cell membranes and thereby disrupt normal activity of excitable membranes needed, for example, for nerve transmission and muscle activity. These channels are widely distributed in biological systems, including unicellular protozoan grazers (Liebeskind et al., 2011). By interfering with these gated sodium channels in protozoans, these toxins may disrupt critical cellular functions such as cell signaling, flagellar motility, prey ingestion, or the coordinated beating of cilia used in motility and feeding. Recently, cellular brevetoxins were shown to deter grazing by zooplankton, specifically copepods (Cohen et al., 2007; Hong et al., 2012; Waggett et al., 2012), suggesting that toxic blooms of K.b. could be promoted by low grazing rates directly linked to cellular brevetoxin concentrations.

The predator in the NPZ model was a generic protozoan grazer, whose grazing rates and metabolic growth efficiency were designed to mimic those of a large ciliate (Hansen et al., 1997). Ciliates are a dominant group of microzooplankton (Pierce and Turner, 1992), and microzooplankton are responsible for most algal grazing in coastal waters (Calbet and Landry, 2004). In addition, ciliates have been identified as dominant grazers during a