Vol. 493: 71–81, 2013
doi: 10.3354/meps10515

MARINE ECOLOGY PROGRESS SERIES
Mar Ecol Prog Ser

Published November 20

Simulating cell death in the termination of *Karenia brevis* blooms: implications for predicting aerosol toxicity vectors to humans

J. M. Lenes*, J. J. Walsh, B. P. Darrow

College of Marine Science, University of South Florida, St. Petersburg, Florida 33701, USA

ABSTRACT: To predict both waterborne and aerosolized toxin vectors associated with harmful algal blooms (HABs) of *Karenia* spp. in European, Asian, and North American waters, loss processes associated with distinct stages of bloom development, maintenance, and termination must be defined in relation to their toxins. In the case of *Karenia brevis*, exposure to brevetoxins (PbTx) during maintenance phase is detrimental to marine life. In addition, release of PbTx-2, 3 during cell death leads to respiratory difficulties in mammals. Human asthma attacks and chronic obstructive pulmonary disease occur once HAB toxins are aerosolized and transported to the coast. Here, we tested the hypothesis that heterotrophic bacterioplankton are a major source of mortality for *Karenia* HABs. A non-linear lysis term for simulation of *K. brevis* HAB termination on the West Florida Shelf was introduced, with the assumption that particle encounters of planktonic microalgae and bacteria can be described as the square of the phytoplankton biomass. This formulation also accounts for nutrient-limitation of *K. brevis* as a precondition for susceptibility to bacterial and viral attack, and potentially programmed cell death. Two model simulations were run of linear and non-linear lysis cases. Model output was compared against observed weekly maximum *K. brevis* concentrations, with statistical metrics calculated over 3 HAB phases during 2001. Introduction of the non-linear lysis term increased the modeling efficiency by 0.68 due to improved reproduction of the bloom termination.

KEY WORDS: Harmful algal blooms · Brevetoxins · *Karenia brevis* · Lysis · Bacteria · Asthma

INTRODUCTION

Enigmatic losses of phytoplankton in the sea are dependent on many factors (Walsh 1983), but the greatest among them relate to both the palatability of different species and cell lysis. While some marine microalgae have evolved physically protective structures such as the spines on diatoms, e.g. *Chaetoceros*, others employ chemical defense mechanisms (Turner & Tester 1989). As an example of a phytoplankton species capable of chemical defense mechanisms (Vargo 2009), the toxic dinoflagellate *Karenia brevis* is the dominant harmful algal bloom (HAB) species in the Gulf of Mexico (GOM). It is an opportunistic organism capable of utilizing multiple nutrient sources throughout its life history (Vargo et al. 2008).

As part of its survival strategies, *Karenia brevis* produces a suite of 9 polyether ladder, lipid-soluble compounds (Baden & Mende 1982, Baden & Tomas 1988) known as brevetoxins (PbTx1-9). They have been linked to fish kills, as well as to marine mammal and avian mortalities (Landsberg 2002, Flewelling et al. 2005). It would appear that release of these toxic compounds by *K. brevis* at varying ratios over the course of the red tide bloom kills fish upon passage of this neurotoxin across their gills, followed by rapid fish decomposition at summer temperatures of surface

*Email: lenes@usf.edu

GOM waters (Stevenson & Childers 2004). In addition, dominant PbTx-2 and PbTx-3 brevetoxins accumulate in the water column at HAB demise (Pierce et al. 2008, Tester et al. 2008). Once these dissolved poisons are air-borne as part of organic matrices of sea salt aerosol exchanges across the sea–air interface (Fleming et al. 2007), they can be the cause of asthma attacks, combined in some cases with chronic obstructive pulmonary disease (COPD) in the human population in coastal regions. There is a latency period of ~5 d before onset of some HAB asthmatic attacks impacts both residents and tourists (Kirkpatrick et al. 2011).

Inhalation of PbTx-3 by laboratory rats, for example, leads to reduced formation of antibodies by an impaired immune system (Benson et al. 2005). Additional mammal analogs subjected to PbTx-3: guinea pigs (Wells et al. 1984), asthmatic sheep (Singer et al. 1998), and manatees (Bossart et al. 1998) also exhibit increased airway resistance, bronchospasms, and pulmonary hemorrhages. Similar immunosuppression in humans by at least PbTx-3, if not by PbTx-2 and PbTx-1, would subject human asthmatics to other respiratory diseases such as fatal pneumonia (Kirkpatrick et al. 2006). Thus, prudent public health management practices require knowledge of both: (1) the time-dependent, wind-forced onshore transports of brevetoxins and (2) the spatio-temporal fields in which these toxic human respiratory triggers prevail.

Experimental studies have suggested that grazing rates on *Karenia brevis* are minimal (Tester et al. 2000), despite evidence that field populations of the copepods *Paracalanus quasimodo*, *Temora turbinata*, and *Centropages velificatus* eat *K. brevis* when other prey were unavailable (Turner & Tester 1997, Walsh et al. 2003). But, if only small amounts of ingested toxins of particulate *K. brevis* origin are transferred up the marine food chain, how do their HABs terminate?

The same question pertains to HABs of another related ichthyotoxic dinoflagellate *Karenia mikimotoi* = *Gyrodinium aureolum* = *Gymnodinium nagasakiense* (Gentien 1998), which now plagues the English Channel (Vanhoutte-Brunier et al. 2008). This HAB produces aerosolized gymnocins (Satake et al. 2002), which have also induced asthma attacks. The prevalence of asthmatic individuals residing on the Isle of Wight in the English Channel increased from 2.3% in 1964 to 14.9% in 1993 and 40.3% during 1999 (Graham et al. 1967, Tariq et al. 1998, Kurukulaaratchy et al. 2003), coincident with the emergence of previously cryptic populations of *K. mikimotoi* in adjacent waters during 1967 (Boalch 1987). A similar seasonal sequence of nitrogen-fixer precursors (Devassy et al. 1978, Anoop et al. 2007), succession of *K. mikimotoi* HABs (Godhe et al. 2001), toxin releases, and fish kills (Madhu et al. 2011), with concurrent asthma episodes (Paramesh 2002, Iyer et al. 2008, D'Silva et al. 2012), now all co-occur along and adjacent to the West Indian Shelf (WIS).

Several biological and physical mechanisms have been implicated in the termination of *Karenia brevis* and *K. mikimotoi* blooms. On the West Florida Shelf (WFS), horizontal advection off the shelf is a major loss process (Walsh et al. 2009, Lenes et al. 2012, Weisberg et al. in press). While such physical export provides a solution to a local problem, the biotic consequences are frequently still detrimental to the downstream human communities and fisheries. For example, the 2007 diazotroph precursor *Trichodesmium erythraeum* and *K. brevis* blooms began formation on the middle/outer WFS by spring of that year. The phytoplankton populations were then advected by the Loop Current past the Florida Keys into the contiguous Florida Current, eventually making landfall ~120 d later near downstream Jacksonville Beach on 28 September (Walsh et al. 2009). Such a spatial translation velocity of ~8 km d^{-1} between the 2 sets of phytoplankton observations during 2007 was consistent with measured speeds of these western boundary currents, affecting their physical transports during seral succession of entrained WFS diazotrophs and dinoflagellates.

Like previous years of WFS HAB exports, increases in both respiratory distresses and fish kills were subsequently reported north of Miami during 2007 (Walsh et al. 2009). Export to the east coast of Florida coincided with an order of magnitude decrease of the *Karenia brevis* biomass left behind on the WFS. Thus, while horizontal dispersion may lead to a significant loss of red tide biomass to a local area, it does not necessarily remove or transform the associated toxicity from larger, adjoining regions. Horizontal advection simply shifts the location of impact, so that apparent local dilution is not the solution to teleconnected marine pollution.

The same downstream transmission of western European aerosolized dinoflagellate poisons resulted in greater causal *Karenia mikimotoi* HAB asthma triggers. Increments of childhood asthma responses to increased HABs also occurred, with 5-fold larger asthma prevalence between 1981 and 2002 in southern Norway (Nystad et al. 1997, Carlsen et al. 2006), as well as 16-fold greater frequency between 1972 and 2003 off eastern Ireland (Manning et al. 2007). Such temporal changes were affected by seasonal reversals of current transports from HAB epicenters of coastal waters off England and France to those down-

stream off Ireland and Norway (Holt & Proctor 2008, Vanhoutte-Brunier et al. 2008, Farrell et al. 2012).

A second physical and more direct mechanism of bloom termination may be high levels of turbulence. *Karenia brevis*, *K. mikimotoi*, and other dinoflagellate cells exhibit low tolerance to high shear forces (Berdalet 1992, Liu et al. 2002, Gentien et al. 2007). Periods of elevated wind-driven mixing and wave formation can cause cells to rupture in surface waters, thus releasing their internal toxins to the water column. Similarly, blooms that are advected onshore to the beach can become entrained in the surf zone. The associated turbulence not only causes high rates of cell lysis, releasing PbTx to the water column, but the wave energy can provide a mechanism for aerosolization of these toxins (Blanchard 1975, Terrill et al. 2001). This allows the toxins an exit to the overlying marine boundary layer. Finally, autotoxicity may be a factor in HAB demises within the English Channel (Gentien et al. 2007), and presumably elsewhere.

Now, in consideration of non-grazing biological modes of cell loss, recent studies have shown that programmed cell death (PCD) plays an important role in the life cycle of both eukaryotic and prokaryotic organisms (Bidle & Falkowski 2004). Such processes can be induced by environmental stressors related to cell age, nutrient deprivation, high light, or oxidative stress (Bidle & Falkowski 2004). PCD in phytoplankton has been linked with increased caspase-like activity, i.e. cysteine-aspartic proteases involved in PCD and necrosis. In cultures, a decrease in *Karenia brevis* cellular concentration was correlated to caspase 3-like protein activity (Bouchard & Purdie 2010). Additionally, the cellular changes associated with cell death expose the phytoplankton population to increased lysis rates as a consequence of attacks from both viruses (Paul et al. 2002) and algicidal bacteria (Roth et al. 2008a,b).

Different types of lytic ammonifying bacterioplankton have been measured in *Karenia brevis* blooms (Jones et al. 2011). Both *Cytophaga* (Doucette et al. 1999) and Flavobacteriaceae (Roth et al. 2008a), which induce cell death in *K. brevis*, have been isolated from the GOM. Earlier observations of ~3.0 × 10^9 cells l^{-1} of bacteriopankton were found on the WFS along the 20-m isobath (Pomeroy et al. 1995) during the negligible red tide of June 1993 (Walsh & Steidinger 2001). Furthermore, an initial experimental stock of 1 × $l0^7$cells l^{-1} of *K. brevis* was reduced to a population level of only <1 × $l0^1$cells l^{-1} within 5 d, after exposure to similar laboratory bacterial concentrations of >$l0^9$ cells l^{-1} (Roth et al. 2007).

Here, we introduced a simple non-linear lysis term for simulation of annual *Karenia brevis* HAB terminations on the WFS, as an isomorph of similar red tide demises of congener species within the English Channel and on the WIS. We assumed that apparent cellular lysis of toxic dinoflagellate field populations is largely the result of nearshore HAB encounters with *in situ* bacterial predators, e.g. particle-attaching, faster-growing *Pseudomonas* spp. of the plankton community of coastal waters (Crump & Baross 1996, Doucette et al. 1999, Jones et al. 2011). The free-living bacterial species of offshore waters have order of magnitude slower rates of substrate assimilation than nearshore populations, reflecting either their size (Hodson et al. 1981) or nutrient availability.

We thus tested the hypothesis that these versatile heterotrophs are major mortality sources of *Karenia* spp. blooms. We further assumed that such particle encounters (Jackson 1990) of planktonic microalgae and bacteria can be simply described as the square of the phytoplankton biomass, like earlier model formulations of: (1) diatom aggregation of faster sinking flocs at spring bloom termination (Alldredge & Gotschalk 1989, Walsh & Dieterle 1994); (2) direct attack on both *K. mikimotoi* and *Prorocentrum dentatum*, as well as on ciliate predators *Flavella* spp., by other hemolytic dinoflagellates *Heterocapsa cicularisquama* via cell contact (Kamiyama & Arima 1997, Uchida et al. 1999, Miyazaki et al. 2005, Yamasaki et al. 2011); and (3) autotoxicity of *K. mikimotoi* (Gentien et al. 2007, Vanhoutte-Brunier et al. 2008).

In addition, we consider nutrient-limitation of *Karenia brevis* and heterotrophic bacterioplankton, which are no longer able to maintain their populations by simply assimilating dissolved inorganic and organic nutrients, as a precondition for susceptibility of *K. brevis* to bacterial attack. We found that the model's simulated bacterioplankton cannot maintain their population levels without utilization of live competing phytoplankton as an additional source of nutrition. This is in contrast to hydrocarbon sources that contain negligible amounts of phosphorus and nitrogen compounds. Furthermore, both the phytoplankton and bacterioplankton are initial competitors for inorganic nutrient supplies, until the predatory heterotrophs prevail against the autotrophs. Although this new numerical lysis formulation describes HAB demise as a result of algicidal bacterioplankton, it indirectly represents a combination of biological cell lytic pathways such as viral lysis and PCDs since these additional processes are similarly induced by environmental stress.

We added this lysis term to a prior 1-dimensional (1D) simulation analysis of the population dynamics of *Karenia brevis* HABs on the WFS during 2001 (Lenes et al. 2012). Model fidelity was evaluated with weekly observations of *K. brevis* on the nearshore WFS during 2001. Accordingly, 3-dimensional (3D) advective loss processes of the WFS (Weisberg et al. in press) are not considered in the present study. This new biotic loss term was formulated as a non-linear function of *K. brevis* biomass and nutrient stress, in competition with the other phytoplankton and bacterioplankton of the model, all subject to additional linear grazing losses in a 1D vertical model of near shore WFS waters (see Fig. 1).

METHODS

Model setup

In this numerical study, we used a revised non-linear version of the previous linear lysis term of the Harmful Algal Bloom Simulations (HABSIM), initially described in Lenes et al. (2012). The model still consisted of 24 state variables: temperature, salinity, the vertical eddy coefficient (K_z) for vertical mixing, spectral light, colored dissolved organic matter (CDOM), dissolved organic carbon (DOC), dissolved organic nitrogen (DON), dissolved organic phosphorus (DOP), dissolved inorganic carbon (DIC), nitrate + nitrite (NO_3), ammonium (NH_4), phosphate (PO_4), silicate (SiO_4), iron (Fe), 4 functional groups of phytoplankton (diatoms, microflagellates, *Trichodesmium erythraeum*, and *Karenia brevis*), zooplankton fecal pellets, ammonifying and nitrifying heterotrophic bacterioplankton, detritus, larval and adult fish, and sediment microbiota. The model had a 1 m vertical resolution over the water column above the 20 m isobath of the WFS at 27.17° N, 82.90° W, southwest of Sarasota, FL (Fig. 1), with 1 sediment layer of 1 cm thickness, and a 30 s time step.

The simulation runs began on 1 January 2001, with a 3 mo spin-up, such that results of the prior baseline case and the new non-linear lysis component of bacterial–phytoplankton interactions are shown during HAB onset, maintenance, and termination phases of April to December 2001 (see Figs. 2 to 4). To better quantify the simulated description of the HAB termination phase during 2001, we changed the linear lysis term of the baseline case (Lenes et al. 2012) to a more complex formulation of Eqs. (1) to (3). Calculation of the new realized *Karenia brevis* lysis term, l_{kb}, was now described by:

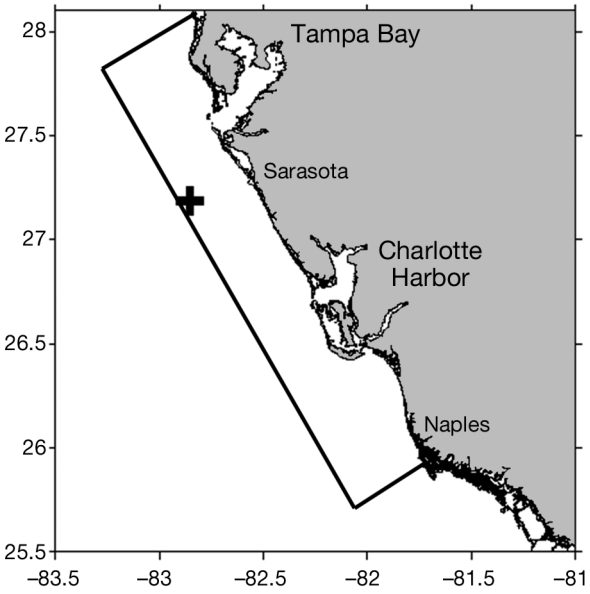

Fig. 1. Location of the 1-dimensional model (+) southwest of Sarasota above the 20-m isobath (27.17° N, 82.90° W), in relation to Tampa Bay and Charlotte Harbor, Florida, USA. The bounded area represents the validation region over which the maximum weekly *Karenia brevis* concentrations were obtained

$$l_{kb} = l_{rm} \times \aleph \quad (1)$$

$$l_{rm} = l_{max} \times (P_{kb}^2 / 90\,000) \quad (2)$$

$$\aleph = 1 - \min(Fe_{lim}, P_{lim}, N_{lim})^2 \quad (3)$$

where l_{max} was the maximum lysis rate and l_{rm} the realized maximum lysis rate.

The parameter 90 000 of Eq. (2) was the square of the *Karenia brevis* biomass (P_{kb}) (300 mmol C m^{-3}) at which the maximum lysis rate occurred in the model. The nutrient limitation adjustment, $\aleph$, was then based on the minima of the *K. brevis* iron, phosphorus, and nitrogen limitation terms (Fe_{lim}, P_{lim}, N_{lim}) at each time step of the model (Lenes et al. 2005, 2008, 2012). Finally, the value of 0.6 d^{-1} for l_{max} obtained from experimental lytic declines of *K. brevis*, subjected to bacterial attack of heterotrophs isolated from the WFS (Roth et al. 2007). An additional numerical stability constraint was imposed that the refuge population of *K. brevis* was 0.1 mmol C m^{-3}, below which amount of biomass no lytic losses prevailed.

Model validation

Here, we utilized monthly nutrient data (see Fig. 2) of 3 cross-shelf sections off Tampa Bay, Sarasota, and Charlotte Harbor during cruises in 2001 of the

NOAA/EPA ECOHAB:Florida and ONR HyCODE programs (Vargo et al. 2008, Walsh & Kirkpatrick 2008). We summed these measurements to compute total dissolved nitrogen (TDN = NO_3 + NH_4 + DON) and total dissolved phosphorus (TDP = PO_4 + DOP).

For additional verification of the model's HAB predictive efficiency, the weekly maximum cell counts of *Karenia brevis* (see Fig. 3) were obtained from the Florida HAB historical database, maintained by the Florida Wildlife Commission's Fish and Wildlife Research Institute (FWC FWRI), at both the surface (<3 m depths) and bottom (<5 m above sediments) within 35 km of the west Florida coast between Cedar Key and Naples (Fig. 1). We used this nearshore information as an estimate of an alongshore cumulative WFS catchment basin of onshore transport of phytoplankton populations during net biological growth and loss processes (Walsh et al. 2003).

Such an approach allowed us to compare the simulated biochemical bloom dynamics by mimicking a drifting bloom independent of the horizontal physical processes and sampling bias (Young & Christman 2006, Heil & Steidinger 2009). The maximal *Karenia brevis* cell counts, i.e. the observed net population successes of their HABs against all gains and losses, were then converted to carbon units of mmol C m^{-3} for comparison of these additional validation data with the model output, using conversion factors of 5.0×10^{-6} µg chlorophyll (chl) $cell^{-1}$ and a C:chl ratio of 30 (Lenes et al. 2012).

Finally, the sparsest data set was a few observations of total bacterioplankton biomass within nearshore WFS waters (see Fig. 4) during June 1993 (Pomeroy et al. 1995) and September 2001 (Jones et al. 2011). These points were presented to represent a range of values measured during bloom and non-bloom scenarios, not as explicit validation. Accordingly, based on the richest data set of weekly cell counts of *Karenia brevis* (see Fig. 3), 6 different statistical criteria (see Table 1) were evaluated for the 2 simulation cases of linear and non-linear lysis terms to assess the predictive efficiency of the revised HABSIM model. The fidelity of model output was again gauged (Lenes et al. 2012) with estimates of: average error (AE); average absolute error (AAE); root mean squared error (RMSE); general standard deviation (RMSE/P); modeling efficiency (MEF); and the coefficient of determination, i.e. the square of the correlation coefficient (r^2).

The statistical goodness of fit criteria were calculated over 3 *Karenia brevis* HAB phases of 2 different lysis model cases during 2001. The statistics for Cases 1 and 2 of the linear and nonlinear lytic terms were first calculated over the full temporal period of model simulations of HAB onset, maintenance, and demise phases from April to December 2001. Then, the statistics for other Cases 1a and 2a were instead computed through the HAB maintenance phase, up to 5 October 2001, only.

RESULTS

Evidence of the improved model performance was first demonstrated by the simulated total dissolved nutrient stocks (Fig. 2). In the baseline case, both the model's surface TDN and TDP underestimated the observed concentrations during HAB termination (Fig. 2). In the new lysis case, the simulated TDN closely matched observations of HAB onset in July and August 2001, HAB maintenance during September and October 2001, and HAB demise in November and December 2001, where these periods were defined by: (1) exponential growth, (2) maintenance of the vertically integrated biomass, and (3) rapid decline in abundance (Vargo 2009, Lenes et al. 2012).

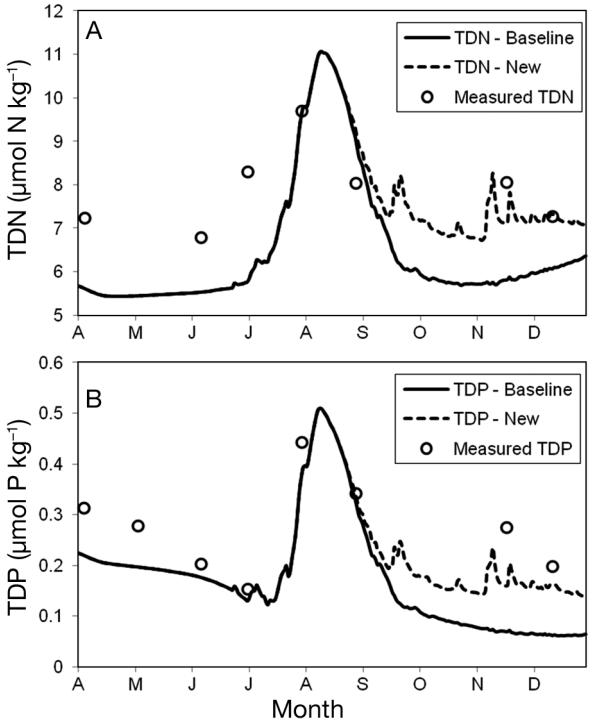

Fig. 2. Simulated daily surface concentrations (µmol kg^{-1}) for Case 1 (baseline; solid line) and Case 2 (new lysis; dashed line) of (A) total dissolved nitrogen (TDN = NO_3 + NH_4 + DON) and (B) total dissolved phosphorus (TDP = PO_4 DOP) in relation to the observed TDN and TDP (O). DON: dissolved organic nitrogen; DOP: dissolved organic phosphorus

The model also yielded larger TDP stocks, approximating those observed (Fig. 2B). These results suggested that the model was now accurately reproducing cell death, since *Karenia brevis* represented the largest source of potential dissolved nutrients.

In order to quantify model improvement, we then compared the simulated surface concentrations of *Karenia brevis* in the baseline and new cases of the model with the weekly carbon biomass equivalents of the FWC FWRI cell counts (Fig. 3A). Little change in either case of the model's HAB biomass was found during initiation of the bloom in July and August 2001 (Fig. 3A). However, a significant divergence of results began during the maintenance phase, with a 30% difference in predicted HAB biomass between the 2 cases by the third week of September 2001. This divergence of the model's surface HAB concentrations reached a maximum of 42% by November 2001 (Fig. 3A). In comparison, the near-bottom biomasses of the model did not vary significantly between the 2 cases (Fig. 3B), since the *K. brevis* population changes at depth were still mainly dictated by settling velocities, swimming speeds, and wind-induced vertical mixing (Lenes et al. 2012).

The improved fidelity of the non-linear lysis case of the model (Fig. 3) reflected both greater lytic losses of *Karenia brevis* and increased success of their bacterial competitors (Fig. 4). Note that the greater simulated biomass of mature populations of these microbial predators now extended over longer time periods during the termination phase of the *K. brevis* HABs (Fig. 4). Yet, without another non-linear definition of predator–prey interactions, in turn, of bacterioplankton and ciliates of this present version of HABSIM, the predicted heterotroph biomass in September 2001 overestimated the bacterial stocks observed at that time on the WFS (Jones et al. 2011).

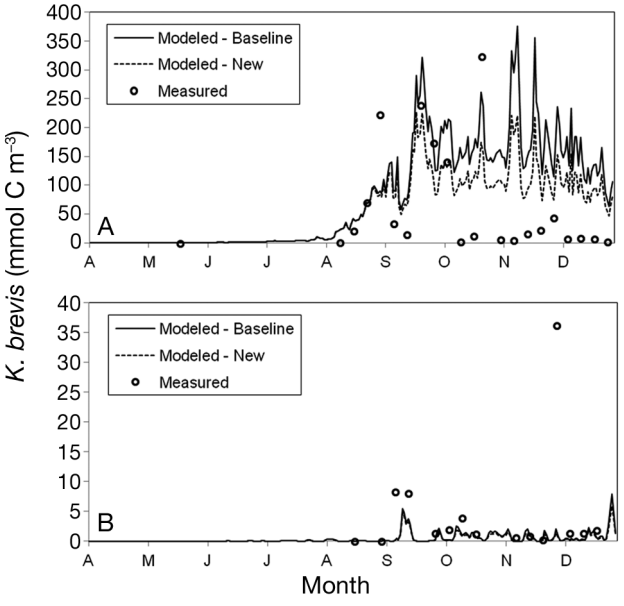

Fig. 3. Simulated daily *Karenia brevis* concentrations (mmol C m^{-3}) for Case 1 (baseline; solid line) and Case 2 (new lysis; dashed line) cases at the (A) surface and (B) near-bottom layer in relation to the observed weekly maximum *K. brevis* concentrations (O)

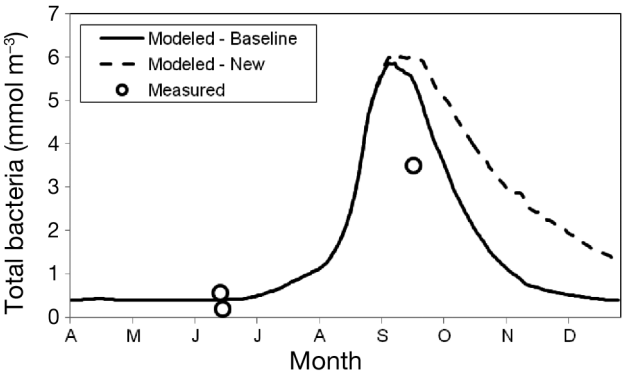

Fig. 4. Simulated daily surface bacterial biovolume (mmol C m^{-3}) for Case 1 (baseline; solid line) and Case 2 (new lysis; dashed line), in comparison with observations during June 1993 in the absence of a large *Karenia brevis* harmful algal bloom (HAB) and September 2001 within a large *K. brevis* HAB

Statistical analysis of the fidelities of the prior baseline (Case 1) and new lysis (Case 2) cases of the model (Table 1) indicated that HABSIM had already successfully reproduced the initiation and early maintenance phases of the 2001 *Karenia brevis* HAB on the WFS, with a MEF of 0.75 between 1 April and 5 October 2001 (Case 1a; Table 1). Now, over all HAB phases of Cases 1 and 2 (Table 1) between 1 April

Table 1. Validation metrics applied to the *Karenia brevis* harmful algal bloom (HAB) phases of multiple lysis model cases during 2001. Statistics for Cases 1 and 2 of the linear and nonlinear lytic terms are calculated over the full temporal domain of simulated HAB onset, maintenance, and demise phases during April to December 2001. Statistics for Cases 1a and 2a are computed through the maintenance phase up to 5 October 2001, only. Metrics were: average error (AE); average absolute error (AAE); root mean squared error (RMSE); general standard deviation (RMSE/P); modeling efficiency (MEF); and the coefficient of determination (r^2)

Statistic	Case 1	Case 1a	Case 2	Case 2a
AE	44.25	5.48	19.23	–8.52
AAE	57.95	24.12	44.48	25.72
RMSE	92.53	42.66	67.47	43.90
RMSE/P	2.38	0.73	1.74	0.75
MEF	–0.46	0.75	0.22	0.73
r^2	0.28	0.75	0.33	0.77

and 31 December 2001, introduction of the non-linear lysis term increased the MEF from −0.46 to +0.22, or an increment of 0.68. This is shown in Fig. 3A, where the dashed line of the new model simulation comes closer to the observations.

The continued failure of the new model to fully reproduce the termination of the 2001 HAB is attributed to 2 major factors. First, poorly parameterized linear *Karenia brevis* grazing losses are now under redefinition as non-linear interactions of explicit state variables of additional ciliate, larvacean, and copepod omnivores, harvested in turn by simulated top predator scyphomedusae, chaetoganths, and oil spills. Secondly, horizontal advection was absent in the 1D simulation, once thermal stratification of the WFS water column broke down in the fall (Weisberg et al. in press).

DISCUSSION

Compilation of the life history of *Karenia brevis* on the WFS found that these shade-adapted HABs initialized near bottom, between the 20 to 40 m isobaths, in response to low light tolerances (Steidinger et al. 1998, Walsh et al. 2009, Weisberg et al. 2009). At this distance from the coast, potential nutrient sources for the HAB initiation phase included: 'new' nitrogen from the diazotrophic *Trichodesmium* spp. (Lenes et al. 2001, Walsh & Steidinger 2001, Mulholland et al. 2004, Lenes & Heil 2010); benthic remineralization of prior spring diatom blooms (Darrow et al. 2003); and influxes from northern GOM rivers (Stumpf et al. 2008). Once *K. brevis* concentrations exceeded 1.0 to 2.5 × 10^5 cells l^{-1}, or 1.0 to 2.5 µg chl l^{-1}, their associated intracellular and waterborne brevetoxin stocks passed mortality thresholds for fish and other marine life (Landsberg 2002).

During this maintenance phase of *Karenia* HABs on the WFS, ichthyotoxic PbTx-1 dominated (Pierce et al. 2008), providing a positive feedback to the *Karenia brevis* populations, since dead fish decay at a rate of as much as 50% per day in the warm summer GOM waters (Stevenson & Childers 2004). Indeed, recent incubation experiments found that *K. brevis* exhibited a realized growth rate of ~1.0 d^{-1}, when fed dissolved nutrients from decaying fish (Killberg-Thoreson et al. in press), compared to the more moderate rate of 0.2 to 0.3 d^{-1} generally observed during blooms (Walsh et al. 2006).

In addition, *Karenia brevis* concentrations of 1.0 to 2.5 × 10^5 cells l^{-1} have been identified as the transition point at which their HABs become monospecific with regard to other competing phytoplankton species (Landsberg 2002). Their initial success could have been due to the allelopathy, while *K. brevis* outcompeted their faster-growing autotrophic counterparts, as well as the bacterioplankton, for available nutrient resources. These initial offshore HABs were subsequently transported toward the coast within the bottom Ekman layer (Weisberg et al. 2009, in press). *K. brevis* HABs then accumulated near the surface, once their stocks increased from both growth and physical aggregation processes, thus reducing photoinhibition through self shading (Walsh et al. 2009). With transition to their coastal maintenance phase, associated toxicity led to fish kills that provided additional recycled nutrients to fuel maximal fall *K. brevis* concentrations of >5.0 × 10^7 cells l^{-1} (Walsh et al. 2006, 2009).

During termination phases of these *Karenia brevis* HABs, a shift in the dominant waterborne toxin forms from PbTx-1 to PbTx-3 was observed on the WFS (Fleming et al. 2011), once cells of *K. brevis* ruptured through bacterial-induced lysis (Roth et al. 2007) and sloppy copepod grazing (Tester et al. 2000). Such observed temporal shifts of brevetoxin dominance are not only an index of HAB status, but also have implications for human health, since PbTx-3 more strongly impacts respiratory functions of analogous mammals (Wells et al. 1984, Bossart et al. 1998, Singer et al. 1998, Benson et al. 2005). Thus, quantification of cell lytic loss processes presented here to more accurately model bloom termination can lead to future simulations of the major toxin pathways as well. This next step would have major public health implications since, depending upon the strength and direction of onshore winds, aerosolized sources of marine HAB toxins can act as asthma triggers of adverse pulmonary episodes.

Although this new numerical lysis formulation of the model described HAB demise on the WFS, it should also represent future applications to additional coastal isomorphs of the English Channel, the WIS, and the western Mediterranean Sea. For example, during July through September 2004, fish kills of Indian oil sardine *Sardinella longiceps* and respiratory distresses of humans, with asthma attacks of residents in the capital city, Thiruvanathapuram (Trivandrum) of Kerala State, India (Iyer et al. 2008), were observed along the southern end of the Malabar coastline of the WIS. These events were then described as follows:

> This bloom resulted in large-scale fish mortality and hospitalization of 200 people especially children who suffered from nausea and breathlessness caused by … *Karenia brevis* (D'Silva et al. 2012, p. 1245).

Subsequently, massive fish kills and ~57 µg chl l^{-1} of *Karenia mikimotoi* were also observed ~220 km farther north in the same coastal Kerala State, near Kochi (Cochin), during October 2009 (Madhu et al. 2011). Previously, another red tide of ~1 × 10^8 cells l^{-1}, or >100 µg chl l^{-1}, of *Gymnodinium nagasakiense* = *K. mikimotoi* occurred off Magaluru (Mangalore) of the adjacent Karnataka State during September 1989 (Karunasagar & Karunasagar 1992). Then, massive diagnostic fish kills of snapper *Lutjanus* spp. and contamination of mussels *Perna viridis* were also found (Karunasagar & Karunasagar 1992). Clearly, future numerical biophysical models, which predict HAB onset, transport, maintenance, and lytic demise, with release of wind-borne onshore asthma triggers of brevetoxin and gymnocin aerosols, would benefit coastal human residents and tourists in Kerala and Karnataka States.

In another isomorph of the western Mediterranean Sea, recent emergence of the previously cryptic tychopelagic dinoflagellate *Ostreopsis ovata* also led to hospitalization of >100 beach visitors along the coast of Genoa, Italy, with respiratory problems, on 18 July 2005 (Zingone et al. 2006). Dispersed by coastal currents before aerosol formation, the lipophylic palytoxins of *Ostreopsis* spp. are more potent than saxitoxins and brevetoxins (Riobo et al. 2004). These regional *Ostreopsis* toxins have, thus far, been responsible for sea-spray initiations of breathing difficulties of adjacent humans from Bari, Italy to Marseilles, France, as well as farther downstream to Barcelona and Majorca, Spain (Vila et al. 2001, Brescianini et al. 2006, Tichadou et al. 2010). Accordingly, these additional aerosolized toxins clearly warrant predictive models of adjacent public health consequences of marine algal cell lysis in many global regions (Rhodes 2011, Parsons et al. 2012).

CONCLUSIONS

Over the last decade, significant progress has been made in testing various hypotheses that drive bloom formation of the toxic dinoflagellate *Karenia brevis* (Vargo 2009). Yet, it is clear that we need a better understanding of the biological loss processes that lead to bloom termination. To predict both waterborne and aerosolized toxin vectors associated with HABs of *Karenia* spp. in European, Asian, and North American waters, loss processes associated with distinct stages of bloom development, maintenance, and termination must be defined in relation to their suite of toxins. In this study, we tested the hypothesis that heterotrophic bacterioplankton are a major source of mortality for *Karenia* HABs by formulating a non-linear lysis term for simulation of *K. brevis* HAB termination on the WFS. The lysis term assumed that particle encounters of planktonic microalgae and bacteria can be described as the square of the phytoplankton biomass, similar to other particle encounter studies. The formulation also accounted for nutrient-limitation of *K. brevis* as a precondition for susceptibility to bacterial attack. This indirectly accounted for viral lysis and PCD due to similar environmental stressors, creating a bulk biological lysis term. The introduction of the non-linear lysis term increased the modeling efficiency by 0.68 due to improved resolution of the bloom termination, setting the stage for future simulations of waterborne and aerosolized toxicity. In addition, future field studies of *Karenia* spp. bloom dynamics should include quantification of bacterial types and biomass to further refine both loss terms and microbial nutrient cycling.

Acknowledgements. This research was sponsored by Grant SA 12-10/GoMRI-007 to J.J.W. and J.M.L. as part of the BP/GRI's C-IMAGE program. Additional support was from: NOAA NA06NOS4780246 to J.J.W. as part of the ECOHAB:Karenia program; ONR N00014-10-1-0794 to J.J.W.; NNX09AT48G to J.M.L. from the National Aeronautics and Space Administration Applied Sciences; and grant #07170 to J.M.L. from the Florida Fish and Wildlife Commission Red Tide Control and Mitigation program. This is ECOHAB contribution #760 and CPR contribution #29. We also thank FWC-FWRI for the *Karenia brevis* data and the 3 anonymous reviews for very helpful comments.

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Editorial responsibility: Steven Lohrenz, New Bedford, Massachusetts, USA

Submitted: December 27, 2012; Accepted: August 14, 2013
Proofs received from author(s): November 11, 2013