Brevetoxin-associated mass mortality event of bottlenose dolphins and manatees along the east coast of Florida, USA

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ABSTRACT: A mass mortality of bottlenose dolphins \textit{Tursiops truncatus} and Florida manatees \textit{Trichechus manatus latirostris} co-occurred with a severe bloom of the toxic algal species \textit{Karenia brevis} along the eastern coast of Florida, USA, between October 2007 and January 2008. Brevetoxin (PbTx), a potent neurotoxin produced by this marine alga, was detected in 69 and 92\% of the tested carcasses of manatees and dolphins, respectively, at concentrations similar to those reported for earlier mortality events along the west coast of Florida. Brevetoxin was also detected in fetal and neonate dolphins, providing evidence of maternal transfer of the toxin in wild populations. This study is the first to document a brevetoxin-associated marine mammal mortality event along the Atlantic coast of Florida. It also demonstrates that, despite the rarity of \textit{K. brevis} blooms in this region, significant negative impacts to marine mammals inhabiting this region can occur.

KEY WORDS: Brevetoxin · Harmful algal bloom · Dolphin · Manatee · Mortality · HAB · Indian River Lagoon

INTRODUCTION

Free-ranging marine mammals in coastal waters of the United States face significant threats to individual health and population status. Some marine mammal species are apex predators and so also serve as sentinel species, so that knowledge of observed changes to marine mammal health can also indicate the state of our oceans’ health (Bonde et al. 2004, Wells et al. 2004, Bossart 2006). In addition to common anthropogenic health stressors such as contaminants, ship strike and fishery interaction (O’Shea et al. 1984, Laist et al. 2001, Read et al. 2006, Balmer et al. 2011), natural processes such as harmful algal blooms (HABs) also pose a serious threat to marine mammal conservation, and exposure to severe HABs frequently leads to mass mortality events (Landsberg et al. 2005, Fire & Van Dolah 2012). Prominent among HABs that cause marine mammal die-offs are blooms of the toxic dinoflagellate \textit{Karenia brevis}, which occur almost annually in Florida coastal waters and can last for several months (Steidinger et al. 1998, Heil & Steidinger 2009). \textit{K. brevis} produces a suite of potent neurotoxins known as brevetoxins, or PbTx (Steidinger et al. 1998). These toxins accumulate in the planktonic and benthic food webs, concentrating in prey organisms such as finfish or on sea-grasses, which then act as vectors of the toxins to dolphins and manatees that feed upon them, resulting in dietary exposure and brevetoxicosis (Fle-

Although K. brevis is endemic to nearly all Florida coastal waters, brevetoxin-producing blooms are rare along the Atlantic coast and associated mass mortality of bottlenose dolphins or manatees has not been reported in this region (Tester & Steidinger 1997, Heil & Steidinger 2009). There are several populations of bottlenose dolphins that inhabit the Atlantic coast of Florida, with a resident Indian River Lagoon (IRL) estuarine system stock as well as 3 oceanic stocks (NOAA 2013). Manatees also inhabit much of the Atlantic coast, and are also found within the IRL (USFWS 2009). The present study describes the first reported mass mortality event of bottlenose dolphin or manatees along the east coast of Florida associated with brevetoxin exposure. Here we present data collected during this 2007 multi-species die-off occurring along Florida’s Atlantic coastal and inshore waters and its co-occurrence with a severe toxic bloom of K. brevis. We present evidence of brevetoxin exposure as the presumed cause of death in both dolphins and manatees and discuss the relevance of this event to similar mass mortality events occurring previously in Florida coastal waters.

**MATERIALS AND METHODS**

**Dolphin sample collection**

All dolphins recovered during this mortality event had stranded or were sampled between 12 and 27 December 2007. All dolphins sampled in the present study (n = 14; 12 carcasses, 2 live animals) were recovered from Atlantic oceanside beaches between New Smyrna Beach and Palm Bay, Florida, USA (Volusia and Brevard counties, 29.013008° to 28.025153° N) except for Hubbs-0783-Tt, which was recovered in the Banana River area, and Hubbs-0778-Tt and Hubbs-0779-Tt, which were live animals entrapped in a small shallow area of the Mosquito Lagoon encircled by spoil islands and sandbars. These 2 dolphins, which were exhibiting no signs of brevetoxin exposure or other health effects, were relocated by boat to deeper water in the lagoon. Standard blood collection (Geraci & Lounsbury 2005) was made prior to release for hematologic evaluation and brevetoxin analysis due to the dolphins’ proximity to the die-off area. Dolphin carcasses were either necropsied on site or transported to the laboratory (Hubbs-SeaWorld Research Institute, Orlando, Florida, USA) for comprehensive necropsy according to established protocols (Geraci & Lounsbury 2005). During necropsy and live sampling, gross pathological findings and morphometric data were recorded, and biological samples were collected for analysis. Dolphins were categorized by sex and age classes using standardized measurements of total length (Geraci & Lounsbury 2005) and age class grouping methods outlined in Wells et al. (1987); ≥246 cm for adult males, ≥231 cm for adult females, 161–245 cm for juvenile males, 161–230 cm for juvenile females, and ≤160 cm for calves. Dolphin tissues (300–500 g per sample) and fluids (2–10 ml per sample) were collected for brevetoxin testing and included samples of stomach contents, feces, liver, lung, blood, muscle, spleen, kidney, blubber, eye and urine. Samples for brevetoxin analysis were collected into polypropylene sample tubes or plastic bags and were stored at −20°C until toxin extraction.

**Manatee sample collection**

All manatees recovered during this mortality event were reported between 4 October 2007 and 12 January 2008. Manatee carcasses were either necropsied on site or transported by trailer to the Florida Fish and Wildlife Conservation Commission’s (FWC) Marine Mammal Pathobiology Laboratory (MMPL) in Saint Petersburg, Florida, USA, for comprehensive necropsy and sample collection. At the MMPL, carcasses were refrigerated overnight until necropsy the following morning. All manatee carcasses in the present study (n = 33) were recovered between Ormond Beach and Fort Pierce (Volusia, Brevard, Indian River and St. Lucie counties, 29.293400° to 27.472102° N) within the Indian River Lagoon (IRL; inclusive of Indian River, Banana River and Mosquito Lagoon). During necropsy, gross pathological findings, gross observations of gastrointestinal contents and morphometric data were recorded, and samples of stomach contents, ingested tunicates, feces, liver, lung, kidney, urine, milk and muscle were collected (n = 30 individuals) for analysis. Manatees were categorized by age class based on methods outlined in
O’Shea & Langtimm (1995); >265 cm for adults, 236–265 cm for sub-adults, and <236 cm for calves. Samples for brevetoxin analysis were collected into polypropylene sample tubes or plastic bags and were stored at −20°C until toxin extraction.

**Phytoplankton collection and analysis**

Phytoplankton samples were collected as part of separate ongoing statewide monitoring and event response efforts routinely conducted by FWC. These phytoplankton data (n = 718 samples; 6 September 2007 to 22 January 2008) were queried from the FWC Harmful Algal Bloom Monitoring Database to obtain concentrations of *Karenia brevis* in seawater samples, reported as cells of *K. brevis* per liter of seawater (cells l⁻¹).

**Toxin extraction**

Urine and serum samples were centrifuged at 3000 × g for 10 min and the supernatants collected prior to analysis. All other samples were homogenized in 80% methanol (4 ml g⁻¹), heated in a hot water bath at 60°C for 20 min, and centrifuged at 3000 × g for 10 min. The supernatant was retained, and the pellet was extracted a second time in the same manner. The supernatants were pooled per tissue sample and brought to a final volume of 10 ml with 80% methanol. The extract was partitioned once with 100% hexane (1:1, v:v), and the methanol fraction was retained. In 2 manatees, samples of seagrass and associated tunicates were opportunistically sub-sampled from stomach contents and extracted using the above methods. Extracts for all samples were stored in glass vials at −20°C until analysis.

**ELISA analysis**

Extracts were analyzed primarily by enzyme-linked immunosorbent assay (ELISA) methods, according to procedures described in Naar et al. (2002). The ELISA uses cross-reactivity of brevetoxin to anti-brevetoxin antibodies to determine brevetoxin-like activity in a sample. Quantitation is determined via competition between brevetoxin in the sample and brevetoxin bound to a 96-well plate for binding to anti-brevetoxin antibodies. Antibodies that bind to the plate are then visualized by recognition by a secondary antibody linked to an enzyme that catalyzes a colorimetric reaction. Concentrations are reported as PbTx-3 equivalents and reflect the overall concentration of brevetoxin and brevetoxin-like compounds present in the sample. Recovery of PbTx-3 standard reference material (AgResearch, Hamilton, New Zealand) added to negative control samples (brevetoxin-negative dolphin and manatee tissues; liver, muscle, brain, lung) was between 94 and 108% using this method. The detection limits for this assay in the present study were approximately 1 ng ml⁻¹ for urine and serum samples and 5 ng g⁻¹ for all other samples.

**LC-MS analysis**

Selected assay-positive extracts were cleaned by C18 solid-phase extraction cartridges (Bond Elut, 500 mg; Agilent) and analyzed by liquid chromatography-mass spectrometry (LC-MS) for parent PbTx toxins and metabolites. Sample preparation and analyses were carried out according to methods described in Fire et al. (2011). Liquid chromatographic separations were performed on a Luna C8(2) 150 × 2 mm column using an Agilent Technologies Model 1100 LC system. Mobile phase consisted of water (A) and acetonitrile (B), with 0.1% formic acid additive with a gradient elution. The mobile phase flow rate was 0.2 ml min⁻¹. The eluate from the LC was analyzed by an AB Sciex 4000 QTRAP hybrid triple quadrupole/linear ion trap mass spectrometer equipped with a TurboVTM interface (Foster City, CA, USA). Analysis of brevetoxin congeners and metabolites by mass spectrometry was achieved by multiple reaction monitoring. Samples were screened for PbTx-3 (dihydrobrevetoxin-B), PbTx-7 (dihydrobrevetoxin-A) and PbTx-9 (tetrahydrobrevetoxin-B), hydrolysis products of PbTx-3 and PbTx-7, and cysteine adducts of PbTx-A (brevetoxin-A) and PbTx-B (brevetoxin-B) and their sulfoxides. The detection limits were 0.2, 2.6, 1.0, and 4.3 ng per gram sample for PbTx-3, PbTx-7, S-desoxybrevetoxin-B2, and brevetoxin-B2, respectively, for toxin standards prepared in methanol.

**RESULTS**

**Association of strandings with *K. brevis* bloom**

The 2007 Florida east coast bloom of *Karenia brevis* was first detected in the northern portion of the study area (Nassau to Flagler counties, Fig. 1), with high cell concentrations exceeding 1 000 000 cells l⁻¹ first re-
Fig. 1. (a) Study area of Nassau County to St. Lucie County, Florida, USA, with locations of bottlenose dolphin *Tursiops truncatus* and manatee *Trichechus manatus latirostris* strandings and phytoplankton sampling efforts during (b) September 2007, (c) October 2007, (d) November 2007, (e) December 2007, and (f) January 2008.
ported 26 September (Fig. 2). By 16 October, *K. brevis* cell concentrations had reached a maximum of 6030 000 cells l$^{-1}$. The appearance of high cell counts progressed southward along the coast, while decreasing steadily in concentration (eventually reaching ~10 000 cells l$^{-1}$) until mid-November. On 20 November *K. brevis* cell concentrations again dramatically increased to more than 1 000 000 cells l$^{-1}$, with cell counts reaching a peak of 5 693 000 cells l$^{-1}$ on 1 December. By this time the resurgent bloom had spread farther southward, and by 9 December had reached the region where all of the manatee and dolphin strandings occurred (Fig. 1). Cell densities in excess of 100 000 cells l$^{-1}$ were observed throughout this region until 9 January 2008, then declined to background concentrations (≤1000 cells l$^{-1}$) by 15 January 2008, having spread over a linear distance of ~250 miles (ca. 400 km) since the bloom began in September. Overall, high cell concentrations throughout the bloom were detected at both inshore and beachside sampling sites, as well as in the passes.

Beginning on 4 October 2007, single manatee carcasses were reported in the IRL, and reports of dead manatees continued until 12 January 2008. Temporally, the strandings were uniformly distributed throughout this period (Fig. 2). Geographically, most strandings were clustered at the confluence of the Mosquito Lagoon, Indian River and Banana River immediately inshore of Cape Canaveral, covering a linear distance of ~135 miles (ca. 220 km) across Volusia, Brevard, Indian River and St. Lucie counties (Fig. 1). In October, 5 manatee strandings occurred well before the bloom had reached the region where the strandings were reported. Additional manatee strandings (n = 5) adjacent to our study region occurred during the September 2007 to January 2008 period, but these were animals that were recovered inland along the St. Johns River or its tributaries, and so were not included as part of our sample set. The dolphin strandings were much more strongly temporally clustered than the manatees, with 10 of the 12 carcasses recovered within a 4 d period (12 to 15 December). Spatially, the extent of strandings was also smaller, covering a linear distance of ~75 miles (ca. 120 km) across Volusia and Brevard counties and primarily grouped along the Atlantic beaches adjacent to the Mosquito Lagoon. All dolphins, including the live dolphins, were sampled within the time frame of the second bloom peak (approx. 20 November 2007 to 9 January 2008).

**Dolphin analyses**

Based on total body length recorded at necropsy or capture, the age classes of the dolphins sampled were distributed as follows: 5 adults (3 males, 1 female, 1 unknown), 7 juveniles (5 males, 1 female, 1 unknown), 1 male neonate calf and 1 male fetus (Table 1). Sex class was skewed toward males, with 10 male dolphins, 2 females and 2 of undetermined sex. Gross examinations of dolphin carcasses recorded no evidence of human interaction, infectious disease or other significant pathology. Overall, 79% (11 of 14) of the

![Fig. 2. Time series of *Karenia brevis* cell concentrations and dolphin and manatee strandings, Nassau County to St. Lucie County, Florida, USA](image-url)
Table 1. Brevetoxin concentrations detected by enzyme-linked immunosorbent assays (ELISA) in bottlenose dolphin *Tursiops truncatus* samples (ng g⁻¹ or ng ml⁻¹). <dl = below limit of detection. Dates are given as mm/dd/yy.

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N                4           6           7           8           10          12          7           10          5           3          2
Min.             39          34          11          23          7           6           1          15          11
Max.             626         557         89          56          28          50          7           72          37
Mean             319         368         60          33          16          20          4           37          25          55          <dl

ᵃLive animals sampled in Mosquito Lagoon
ᵇConfirmed by liquid chromatography-mass spectrometry (LC-MS)

dolphins sampled tested positive for brevetoxin in at least 1 sample type (Table 1). For both live-rescue dolphins from the Mosquito Lagoon, only serum samples were available, and these were negative for brevetoxin. Excluding these, 92% (11 of 12) of the dead-stranded dolphins tested positive for brevetoxin. Gastrointestinal (GI) contents had the highest levels of brevetoxin, with concentrations up to 626 ng g⁻¹ in brevetoxin-positive samples (4 of 4 positive stomach content samples, 39–626 ng g⁻¹; 5 of 6 for feces, 34–557 ng g⁻¹). With respect to distribution among tissue compartments, brevetoxin was detected in nearly all other sample types collected, including liver (8 of 9 samples testing positive, 11–89 ng g⁻¹), kidney (6 of 8, 23–56 ng g⁻¹), muscle (5 of 10, 7–28 ng g⁻¹), lung (10 of 12, 6–50 ng g⁻¹), blood (4 of 7, 1–7 ng ml⁻¹), blubber (7 of 10, 15–72 ng g⁻¹), spleen (5 of 5, 11–37 ng g⁻¹) and eye (1 of 2, 55 ng g⁻¹). Brevetoxin was not detected in the 2 urine samples collected. One whole fish (species not identified) was opportunistically sampled from the esophagus of Hubbs-0777-Tt and tested positive for brevetoxin concentrations of 40 and 754 ng g⁻¹ in muscle and liver tissue, respectively (data not shown).

Brevetoxins, both parent toxins and multiple metabolites, were also confirmed by LC-MS in samples from Hubbs-0771-Tt (liver, PbTx-3) and Hubbs-0773-Tt (feces, PbTx-3, PbTx-7, S-desoxybrevetoxin-B2, and brevetoxin-B2).

Manatee analyses

Based on total carcass length recorded at necropsy, the age and sex classes of the manatees sampled were distributed as follows: 11 adults (4 males, 7 females [of which 2 were pregnant]), 6 subadults (4 males, 2 females), and 16 calves (10 males, 6 females; Table 2). Cause of death was determined as watercraft trauma in 5 manatee carcasses. Ingestion of a fishing hook and line was determined as the cause of death in 1 carcass. Two calf carcasses were stillborn, and 1 calf had possibly been orphaned or abandoned. Three animals had died from cold stress, and 14 from other natural causes. None of the carcasses presented evidence of infectious disease or other significant pathology. Extreme decomposition prevented determination of cause of death in 7 carcasses. Of the 29 manatees (plus 1 fetus) sampled, 20 (69%) tested positive for brevetoxin in at least 1 sample type (Table 2), and for 11 of these brevetoxicosis was concluded to be the probable cause of death. Brevetoxicosis cases were defined as those with vascular congestion in one or more organs or tissues on necropsy, with brevetoxin concentrations detected in multiple samples and for which no other cause of death was apparent. Badly decomposed carcasses with no detectable gross findings were considered probable brevetoxicosis cases if they were brevetoxin-positive.
in multiple tissues and GI contents and at least one such sample contained brevetoxin at a concentration of at least 50 ng g\(^{-1}\). As observed in the dolphins, GI contents had the highest levels of brevetoxin, with concentrations of 7–1173 ng g\(^{-1}\) in stomach contents (13 of 23 samples testing positive) and 30–38 ng g\(^{-1}\) in feces (2 of 3 samples positive). Brevetoxin was also detected in all other sample types, including liver (15 of 27, 7–116 ng g\(^{-1}\)), kidney (5 of 14, 13–21 ng g\(^{-1}\)), muscle (2 of 2, 13–38 ng g\(^{-1}\)), lung (3 of 26, 7–49 ng g\(^{-1}\)), and urine (6 of 12, 1–24 ng ml\(^{-1}\)). Milk opportunistically collected from one manatee (MEC0766) also contained measurable levels of brevetoxins (24 ng ml\(^{-1}\)). Stomach contents of all red tide-related carcasses contained seagrasses, indicating recent feeding, with samples of epifaunal tunicates (n = 2) collected from this seagrass material also testing positive for PbTx (Table 2).

**DISCUSSION**

Although *Karenia brevis* is often present at background concentrations in seawater and brevetoxin has been detected at low levels in marine mammals along the Atlantic coast of Florida (S. E. Fire unpubl. data), *K. brevis* blooms are relatively infrequent and marine mammal mortality events associated with

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| N    | 23  | 3  | 27  | 14  | 2  | 26  | 12  |
| Min. | 7   | 30 | 7   | 13  | 7  | 13  | 7   |
| Max. | 1173| 38 | 116 | 21  | 38 | 49  | 24  |
| Mean | 216 | 34 | 51  | 16  | 26 | 22  | 9   |
brevetoxin exposure have not previously been reported in this region (Tester & Steidinger 1997). A 1987–1988 mass mortality of bottlenose dolphins between Florida and New Jersey was initially thought to have been caused by an expatriate *K. brevis* bloom from the Gulf of Mexico (Geraci 1989), but due to a lack of adequate brevetoxin analytical methods and the presence of co-occurring morbillivirus infection, the involvement of brevetoxin remains speculative (Duignan et al. 1996, Van Dolah 2005). In this case, however, the analytical data support the connection. The severity of the bloom (with numerous samples across 6 counties exceeding 1 000 000 cells l\(^{-1}\) between September and December 2007; Fig. 1) was also similar to that of previous blooms associated with *K. brevis*-related mass mortalities along the Gulf of Mexico coast (Landsberg & Steidinger 1998, FWC 2008, Twiner et al. 2012). Negative effects of the bloom extended to other natural resources as well, including fish kills (131 reports of red tide-related fish kills were received by the FWC Fish Kill Hotline between September and December 2007; FWC unpubl. data) and prolonged closures of shellfish harvest areas in 4 counties to prevent Neurotoxic Shellfish Poisoning.

The total number of dolphins stranding during this study was fairly modest compared with similar Florida mortality events in which more than 100 dolphins died (Mase et al. 2000, Flewelling et al. 2005, Twiner et al. 2012). However, compared with historical stranding records, the number of dolphins that stranded during this event (n = 14) was several times greater than the mean number of strandings (mean = 3.5) for December over an 11 yr period of consistent response effort (Fig. 3a). Manatee mortality did not reach sufficient numbers to initiate official notification of a possible unusual mortality event, which is greater than 7 manatee carcasses or distressed manatees found in a localized area within 72 h (USFWS 1997). However, compared with historical stranding records for Volusia County to St. Lucie County, the number of manatees that stranded in this event (n = 33) was also notably greater than the mean number of strandings (mean = 20) for September to January over the previous 10 yr of consistent response effort (Fig. 3b).

Both the degree of accumulation and distribution of brevetoxin in tissues and fluids collected from the dolphins and manatees were similar to what has been reported for previous *K. brevis*-associated mortality events elsewhere in Florida (Fig. 4) (Mase et al. 2000, Flewelling et al. 2005). Compared with other types of samples, gastrointestinal contents had the highest concentrations of brevetoxin in each individual sampled (max. 626 ng g\(^{-1}\) dolphin, 1173 ng g\(^{-1}\) manatee), typically followed by liver (max. 89 ng g\(^{-1}\) dolphin, 116 ng g\(^{-1}\) manatee) and kidney (max. 56 ng g\(^{-1}\) dolphin, 21 ng g\(^{-1}\) manatee). Where similar sample types were collected and could be compared across events, brevetoxin exposure in the present study was comparable to that seen in conjunction with dolphin die-offs along the Gulf of Mexico coast of Florida in 1999–2000, 2004 and 2005–2006, and with a manatee die-off in 2002 (Fig. 4). Finfish and seagrass were observed in stomach contents of the dolphins and manatees, respectively, and high concentrations of brevetoxin were detected in the fish and epifaunal tunicates from these stomach content samples. This reinforces previous studies that have identified these dietary items as brevetoxin vectors, which, when consumed, lead to an accumulation of toxins in manatees and dolphins (Flewelling et al. 2004, Naar et al. 2007, Fire et al. 2008). Also of note was the general lack of brevetoxin detected in the
manatee lung samples, with only 3 of 26 samples testing positive. Although brevetoxins can become aerosolized and enter the respiratory tract (Pierce et al. 1990, Bossart et al. 1998), the concentrations detected in these few samples were relatively low (7–49 ng g⁻¹). Together with the high brevetoxin levels detected in stomach contents, these data suggest dietary exposure rather than inhalation of brevetoxin as the primary route of intoxication in manatees during *K. brevis* blooms. In the absence of other evidence of possible causes of death in these animals from gross pathology (human interaction, infectious disease, etc.), and in light of the evidence of brevetoxin exposure in these animals during the 2007 *K. brevis* bloom, brevetoxicosis is the presumed cause of this mortality event.

Our findings also provide new evidence that marine mammals are exposed to brevetoxin in early developmental stages. Brevetoxins were detected in milk from a lactating manatee (MEC0766) as well as in a dolphin fetus (Hubbs-0775-Tt-Fetus) and a dolphin neonate (Hubbs-0783-Tt, Table 1). The presence of brevetoxin in the dolphin fetus indicates in utero exposure during the *K. brevis* bloom; its presence in neonate tissues suggests in utero exposure and/or maternal transfer of brevetoxin through lactation, since animals of this age are nursing calves and do not forage independently (Wells et al. 1987).

These results confirm in the field what has been demonstrated experimentally in rodents: that placental transport of brevetoxin or its metabolites occurs following maternal exposure (Benson et al. 2006). This finding has serious implications for conservation efforts that protect dolphin and manatee health, since other HAB toxins are known to cause permanent neurological damage to marine mammals exposed to HAB toxins as perinates. For example, the HAB toxin domoic acid, an analog of the neurotransmitter glutamate, can cause reproductive failure and a chronic epileptic syndrome in California sea lions *Zalophus californianus* exposed to the toxin in utero or soon after birth via toxin-contaminated milk (Brodie et al. 2006, Goldstein et al. 2008). In addition, both brevetoxins and the structurally related ciguatoxins caused embryo toxicity and developmental abnormalities in experimentally dosed finfish (Kimm-Brinson & Ramsdell 2001, Colman et al. 2004). If brevetoxins are similarly able to induce neurological changes at these critical stages in dolphin and manatee development, then the health of these protected species may be threatened at the individual as well as the population level.

In conclusion, we describe here a novel brevetoxin-associated multi-species mortality event along the east coast of Florida, with evidence of brevetoxin intoxication across all age and sex classes. We further
show that red tides on Florida’s east coast, though rare, can result in mass mortality events with levels of brevetoxin exposure in dolphins and manatees similar to those reported elsewhere in Florida. We also demonstrate the first incidence of maternal transfer of brevetoxins in free-ranging marine mammals in their natural habitat during a K. brevis bloom. These findings support current management policies under which phytoplankton and marine mammals are biomonitored nationwide in efforts to assess the impacts of HABs and their associated toxins on protected marine species, ultimately informing marine policy decisions on conservation of sentinel species such as bottlenose dolphins and manatees in US coastal waters.

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NOAA disclaimer: This publication does not constitute an endorsement of any commercial product or intend to be an opinion beyond scientific or other results obtained by the National Oceanic and Atmospheric Administration (NOAA). No reference shall be made to NOAA, or this publication and its natural habitat during a K. brevis bloom. These findings support current management policies under which phytoplankton and marine mammals are biomonitored nationwide in efforts to assess the impacts of HABs and their associated toxins on protected marine species, ultimately informing marine policy decisions on conservation of sentinel species such as bottlenose dolphins and manatees in US coastal waters.

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