



Eosinophilia and biotoxin exposure in bottlenose dolphins (*Tursiops truncatus*) from a coastal area impacted by repeated mortality events[☆]

Lori H. Schwacke^{a,*}, Michael J. Twiner^{b,1}, Sylvain De Guise^c, Brian C. Balmer^d, Randall S. Wells^d, Forrest I. Townsend^e, David C. Rotstein^f, Rene A. Varela^g, Larry J. Hansen^h, Eric S. Zolman^b, Trevor R. Spradlin^f, Milton Levin^c, Heather Leibrecht^c, Zhihong Wang^b, Teresa K. Rowles^f

^a National Oceanic and Atmospheric Administration, National Ocean Service, Center for Human Health Risks, 331 Fort Johnson Road, Charleston, SC 29412, USA

^b National Oceanic and Atmospheric Administration, National Ocean Service, Center for Coastal Environmental Health and Biomolecular Research, 219 Fort Johnson Road, Charleston, SC 29412, USA

^c University of Connecticut, Department of Pathobiology and Veterinary Science, 61 North Eagleville Road, U-89, Storrs, CT 06269, USA

^d Chicago Zoological Society, c/o Mote Marine Laboratory, 1600 Ken Thompson Parkway, Sarasota, FL 34236, USA

^e Bayside Hospital for Animals, 251 N.E. Racetrack Road, Fort Walton Beach, FL 32547, USA

^f National Oceanic and Atmospheric Administration, National Marine Fisheries Service, Office of Protected Resources, Marine Mammal Health and Stranding Response Program, 1315 East West Highway, Silver Spring, MD 20910, USA

^g Ocean Embassy Inc, 6433 Pinecastle Blvd, Ste 2, Orlando, FL 32809, USA

^h National Oceanic and Atmospheric Administration, National Marine Fisheries Service Southeast Fisheries Science Center, 101 Pivers Island Road, Beaufort, NC 28516, USA

ARTICLE INFO

Article history:

Received 21 October 2009

Received in revised form

23 April 2010

Accepted 7 May 2010

Available online 26 May 2010

Keywords:

Marine mammal

Biotoxin

Domoic acid

Immunology

Eosinophil

ABSTRACT

Bottlenose dolphins (*Tursiops truncatus*) inhabiting coastal waters in the northern Gulf of Mexico have been impacted by recurrent unusual mortality events over the past few decades. Several of these mortality events along the Florida panhandle have been tentatively attributed to poisoning from brevetoxin produced by the dinoflagellate *Karenia brevis*. While dolphins in other regions of the Florida coast are often exposed to *K. brevis* blooms, large-scale dolphin mortality events are relatively rare and the frequency and magnitude of die-offs along the Panhandle raise concern for the apparent vulnerability of dolphins in this region. We report results from dolphin health assessments conducted near St. Joseph Bay, Florida, an area impacted by 3 unusual die-offs within a 7-year time span. An eosinophilia syndrome, manifested as an elevated blood eosinophil count without obvious cause, was observed in 23% of sampled dolphins. Elevated eosinophil counts were associated with decreased T-lymphocyte proliferation and increased neutrophil phagocytosis. In addition, indication of chronic low-level exposure to another algal toxin, domoic acid produced by the diatom *Pseudo-nitzschia* spp., was determined. Previous studies of other marine mammal populations exposed recurrently to *Pseudo-nitzschia* blooms have suggested a possible link between the eosinophilia and domoic acid exposure. While the chronic eosinophilia syndrome could over the long-term produce organ damage and alter immunological status and thereby increase vulnerability to other challenges, the significance of the high prevalence of the syndrome to the observed mortality events in the St. Joseph Bay area is unclear. Nonetheless, the unusual immunological findings and concurrent evidence of domoic acid exposure in this sentinel marine species suggest a need for further investigation to elucidate potential links between chronic, low-level exposure to algal toxins and immune health.

Published by Elsevier Inc.

[☆] **Funding sources:** This work was conducted as a partnership of the National Oceanic and Atmospheric Administration (NOAA), Chicago Zoological Society and the University of North Carolina, Wilmington (UNCW). Funding for the dolphin health assessment fieldwork was provided by NOAA's Marine Mammal Health and Stranding Response Program and conducted under Permit No. 932-1489 (as amended) issued by NOAA's National Marine Fisheries Service. Protocols for the dolphin capture-release and tagging were approved by the UNCW Internal Animal Care and Use Committee, Permit No. 2004-012.

* Corresponding author. Fax: +1 843 762 8737.

E-mail address: Lori.Schwacke@noaa.gov (L.H. Schwacke).

¹ Current address: University of Michigan, Department of Natural Sciences, 4901 Evergreen Road, Dearborn, Michigan 48128, USA.

1. Introduction

Unusual mortality events (UMEs) of bottlenose dolphins, manifested by a marked increase in the number of carcasses washing ashore, have occurred repeatedly over the past two decades in the northern Gulf of Mexico and specifically along the Florida Panhandle. Some of the events have been tentatively attributed to exposure to brevetoxin (PbTx), produced by the dinoflagellate, *Karenia brevis*. Although definitive evidence of brevetoxicosis as the primary cause of death has been elusive,

circumstantial evidence points to *K. brevis* as the most likely culprit in at least some of the Florida Panhandle mortality events. A UME spanning 9 months (August 1999–May 2000) was spatially and temporally coincident with *K. brevis* blooms and the associated deaths of fish, sea turtles, invertebrates and sea birds (NOAA, 2004). Early in the event, dolphins primarily stranded in and around St. Joseph Bay (Fig. 1), strandings later spread westward to St. Andrew's Bay and Choctawhatchee Bay, eventually totaling over 150 bottlenose dolphin carcasses.

A subsequent event in the spring of 2004 remained localized to the St. Joseph Bay area and lasted only a single month but still resulted in more than 100 dolphin mortalities (NOAA, 2004). There was a concurrent absence of significant densities of *K. brevis* in the region, but stomach contents sampled from stranded dolphins showed high levels of PbTx, with many of the dolphins stranding with full stomachs (N. Barros, pers. comm.), suggesting an acute toxic effect rather than chronic disease or a lack of food resources as likely causal factors (Flewelling et al., 2005; NOAA, 2004).

Bottlenose dolphin strandings spiked again in the beginning of September 2005 and continued to be elevated through April 2006, with dispersed strandings across much of the Florida Panhandle early in the event, and then a peak of mortalities occurring in spring 2006 in the more western Choctawhatchee Bay. *K. brevis* blooms were noted in the first few months of the event, but then dissipated. Although measured seawater cell counts of *K. brevis* were negligible by March/April 2006 (Naar et al., 2007), the number of bottlenose dolphin strandings was still elevated and PbTx was detected in the stomach contents of many of the recovered carcasses as well as in prey species (MJT unpublished data). This suggests that the dynamics of *K. brevis* blooms are complex and measured cell counts of *K. brevis* may not always correlate with risks to marine wildlife and/or human populations.

It is puzzling as to why *K. brevis* blooms, which have historically been rare in the Panhandle area, would prompt such massive mortalities. Dolphin mortality events of the same frequency or intensity have not been noted in other portions of

the Florida coast where *K. brevis* blooms are relatively commonplace. In fact, *K. brevis* blooms have been documented almost annually along the south and central west coast of Florida since the 1940s, but only a few dolphin mortality events have been documented (1946–1947, 1953–1955, 2005) and these were in conjunction with severe and prolonged blooms (TKR unpublished data; Gunter et al., 1948; Steidinger et al., 1972; Steidinger and Joyce Jr., 1973). Why dolphin die-offs have occurred recently along the Florida Panhandle and seem to be related to atypical blooms of *K. brevis*, yet are not seen in southern areas where blooms are frequent, is a driving question for ongoing research (Gaydos, 2007). One potential hypothesis is that dolphins in the Panhandle area differ with regard to prey preference and/or availability, thus creating potential vectors of exposure that are not as likely for bottlenose dolphin populations along the Florida west coast. Alternatively, Panhandle dolphins may be exposed to some yet unknown environmental stressor, rendering them more susceptible to the toxic effects of *K. brevis* and thus more vulnerable to mortality events. It is also possible that the PbTx to which animals were exposed differed qualitatively between events. Understanding the toxin exposure of dolphins and correlated health impacts is important not only for the management of this protected species, but also to elucidate the potential for human health risks.

As part of ongoing research to address these hypotheses, capture–release efforts were conducted in and around St. Joseph Bay to evaluate dolphin health and background toxin exposures. The first health assessment was conducted in April, 2005, one year following the die-off centralized within St. Joseph Bay at approximately the same time of year. The 2005/2006 die-off began approximately 4 months later. A second health assessment effort was conducted in July, 2006 approximately 2 months after the longer term and less localized 2005/2006 UME had subsided. The objectives of the health assessment efforts were to (1) evaluate a number of health and functional immune parameters for dolphins in St. Joseph Bay and compare these health endpoints with those measured from other wild populations and (2) measure baseline exposure of dolphins to the biotoxin PbTx. Levels of domoic acid (DA), produced by the diatom *Pseudo-nitzschia* spp., were also measured in tissues of the sampled dolphins.

2. Materials and methods

2.1. Sampling

Bottlenose dolphin health assessments were conducted during April 18–28, 2005 and July 17–28, 2006 in and around St. Joseph Bay, FL (Fig. 1). Methods for the capture–release health evaluation generally followed those described by Wells et al. (2004). Dolphin groups that included young calves (seemingly < 2 yr) were avoided. Dolphins were captured by encircling them with a net and restraining them by hand. Adult female dolphins were examined via ultrasound to determine reproductive condition; pregnant females had blood drawn and were then released. Non-pregnant females and males had blood drawn and were weighed and hoisted aboard a processing boat for complete physical examination, skin assessment, morphometric measurement, diagnostic ultrasound and collection of urine, feces, blowhole swab sample, gastric fluid, and blubber samples. Standardized data collection forms developed for dolphin health assessment studies in Sarasota, FL and other NOAA studies along the southeast coast, were used for physical examination and skin assessment. This facilitated comparison with other dolphin study sites. Freezebrands and VHF tags were also applied to enable post-capture follow-up (Balmer et al., 2008).

2.2. Age class determination

A single tooth was extracted under local anesthesia from most individuals and age was determined by reading dentinal and cemental growth layer groups (GLGs) (Hohn et al., 1989). A tooth was not obtained from some dolphins due to their presumed young age ($n=3$), or pregnancy status ($n=3$). Also, two dolphins were

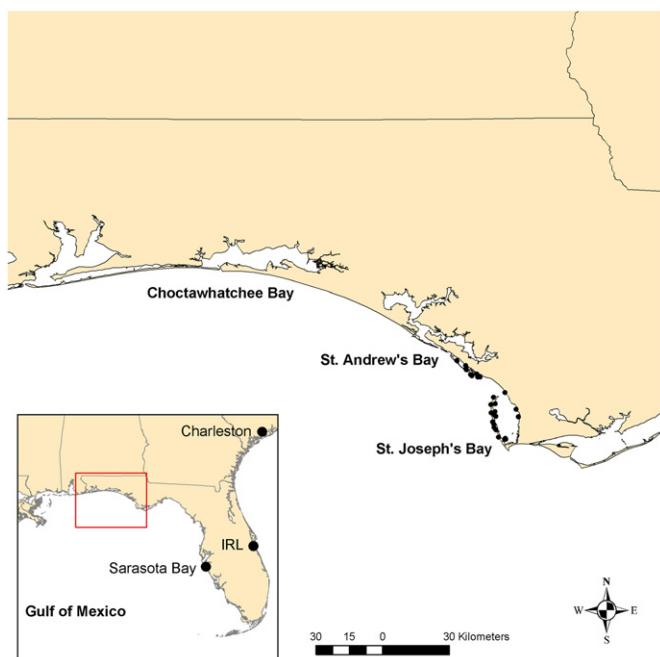


Fig. 1. Map of Florida Panhandle. Black circles represent locations where dolphins were sampled.

Download English Version:

<https://daneshyari.com/en/article/4470349>

Download Persian Version:

<https://daneshyari.com/article/4470349>

[Daneshyari.com](https://daneshyari.com)