

BRIEF COMMUNICATIONS

Red tides and marine mammal mortalities

Unexpected brevetoxin vectors may account for deaths long after or remote from an algal bloom.

Potent marine neurotoxins known as brevetoxins are produced by the 'red tide' dinoflagellate *Karenia brevis*. They kill large numbers of fish and cause illness in humans who ingest toxic filter-feeding shellfish or inhale toxic aerosols¹. The toxins are also suspected of having been involved in events in which many manatees and dolphins died, but this has usually not been verified owing to limited confirmation of toxin exposure, unexplained intoxication mechanisms and complicating pathologies²⁻⁴. Here we show that fish and seagrass can accumulate high concentrations of brevetoxins and that these have acted as toxin vectors during recent deaths of dolphins and manatees, respectively. Our results challenge claims that the deleterious effects of a brevetoxin on fish (ichthyotoxicity) preclude its accumulation in live fish, and they reveal a new vector mechanism for brevetoxin spread through food webs that poses a threat to upper trophic levels.

In the spring of 2002, 34 endangered Florida manatees (*Trichechus manatus latirostris*) died in southwest Florida, and 107 bottlenose dolphins (*Tursiops truncatus*) died in waters off the Florida panhandle in the spring of 2004. In both of these unusual mortality events, extensive water surveys revealed that only low concentrations of *K. brevis* were present.

We tested for the presence of brevetoxin in the fluids and tissues of 63 of these animals (27 manatees, 36 dolphins) and found very high concentrations in the tissues of all of them (see supplementary information), confirming that the animals must have been exposed to brevetoxin. In a previous event, in which 149 manatees died, lung pathology indicated that brevetoxins had been inhaled⁵. In our examples, the absence of similar pathology excluded the possibility of poisoning through aerosol exposure, and the high toxin concentrations measured in the stomach contents indicated that the toxin was from a dietary source.

Manatee stomach contents were composed exclusively of seagrass; filter-feeding tunicates, which were suspected vectors in a 1982 mortality event³, were notably absent. Analysis of seagrass (*Thalassia testudinum*) collected at several locations in the area of death revealed high concentrations of brevetoxins (Fig. 1a), mainly in the epiphytic fraction (epiphytes, 83% of total brevetoxins; blades, 7%; rhizomes, 10%). The accumulation mechanism could involve active uptake or passive adsorption of

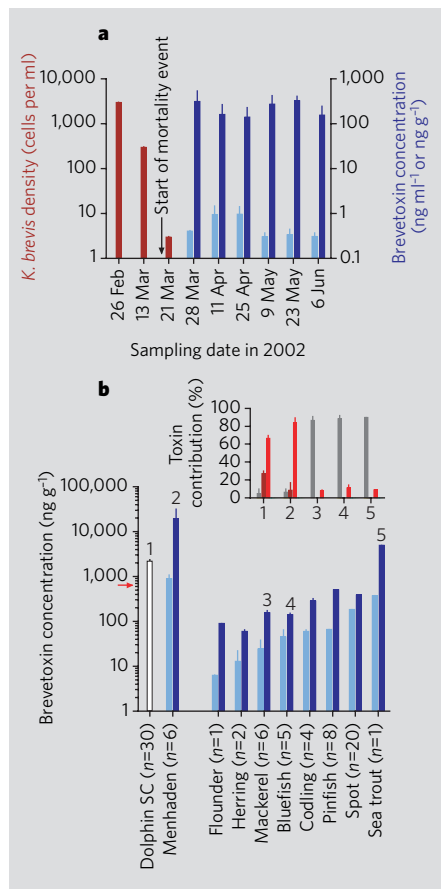


Figure 1 | Brevetoxin concentrations in seagrass and fish during mass-mortality events.

a, Density of red-tide algae *Karenia brevis* (red bars) and brevetoxin concentrations in seagrass (*Thalassia testudinum*) (dark blue bars; ng g⁻¹) and in sea water (light blue bars; ng ml⁻¹), collected during and after the 2002 manatee mortality event in Charlotte Harbor, Florida. Error bars, standard deviation between samples collected from four sites. **b**, Brevetoxin concentrations in dolphin stomach contents (SC), in undigested menhaden, and in fish collected live (flounder, *Paralichthys lethostigma*; herring, *Opisthonema oglinum*; mackerel, *Scomberomorus maculatus*; bluefish, *Pomatomus saltatrix*; codling, *Urophycis floridana*; pinfish, *Lagodon rhomboides*; spot, *Leiostomus xanthurus*; and sea trout, *Cynoscion nebulosus*) from St Joseph Bay, Florida, in spring 2004. Red arrow, regulation limit for brevetoxin in shellfish. Error bars, standard deviation between individual fish, except for pinfish and spot (pooled). Inset, toxins identified by liquid chromatography and mass spectroscopy in selected samples, as numbered in the main bar chart; those in dolphin stomach contents and in menhaden (1, 2) differed from the profile found in fish collected live two weeks after the onset of the mortality (3–5). Bars: dark red, brevetoxin-2; light red, brevetoxin-3; grey, brevetoxin-2 disulphide metabolite. For methods, see supplementary information.

the toxin. As the red tide that previously affected the area had almost dissipated by the start of the mortality event (Fig. 1a), the comparable toxin concentrations in manatee stomach contents and in the seagrass beds (up to 1,136 and 1,263 ng brevetoxin per g, respectively) indicated that seagrass was the primary source of brevetoxin for the manatees.

An extensive pathological, pathogenic and environmental investigation conducted in response to the dolphin mortalities failed to identify any consistent mortality factor other than brevetoxin⁶. Although no *K. brevis* was evident at the time, the contents of the dolphins' stomachs were acutely toxic. Stomachs were full and menhaden (*Brevoortia* spp.), a type of plankton-eating fish, were identified as the dominant prey in 50% of the 28 animals examined. Surprisingly, there was a high level of brevetoxin contamination in all undigested menhaden tested and, to a lesser extent, in all fish that were collected live two weeks after the

onset of the dolphin deaths (Fig. 1b).

Until now, it was uncertain whether live fish could accumulate and transfer brevetoxins to upper trophic levels, as brevetoxins kill fish even at low concentrations⁷ and typically result in high fish mortalities during red tides¹. To determine how brevetoxins might accumulate in fish, we exposed omnivorous and planktivorous fish to toxic shellfish (which retain brevetoxins after blooms have dissipated¹) and to bloom concentrations of healthy *K. brevis* cultures with low extracellular toxin concentrations (as sometimes observed during red tides⁸), respectively. We found that brevetoxin accumulates in both types of feeder (results not shown). Because brevetoxins are sequestered in their food (shellfish and *K. brevis* cells), the fish remained healthy while brevetoxin concentrations increased in their tissues (up to 2,675 ng g⁻¹ in the viscera and 1,540 ng g⁻¹ in the muscle of omnivorous fish exposed for two weeks to toxin-containing clams).



Florida manatees (3 metres long, on average) are susceptible to toxins from the red tide alga *Karenia brevis* (inset; cell diameter, 30–35 mm).

Brevetoxin poisoning in humans has so far been restricted to the consumption of contaminated shellfish (neurotoxic shellfish poisoning). Although the accumulation of brevetoxins in live fish to the levels measured in the menhaden is probably short-lived and unusual, this finding, together with the dolphin deaths (given that dolphins are a sentinel species⁹), raises concerns that humans could also be poisoned by contaminated fish.

These findings show not only that brevetoxin-contaminated food webs pose a threat to marine mammals, but also that toxin vectors can result in delayed or remote animal exposure. Biological toxins should therefore be considered as possible culprits when investigating unusual marine animal mortalities, even in the absence of toxin-producing algae.

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SEISMOLOGY

Earthquake risk on the Sunda trench

On 28 March 2005 the Sunda megathrust in Indonesia ruptured again, producing another great earthquake three months after the previous one. The rupture was contiguous with that of the December 2004 Sumatra–Andaman earthquake, and is likely to have been sparked by local stress, although the triggering stresses at its hypocentre were very small — of the order of just 0.1 bar. Calculations show that stresses imposed by the second rupture have brought closer to failure the megathrust immediately to the south, under the Batu and Mentawai islands, and have expanded the area of increased stress on the Sumatra fault. Palaeoseismologic studies show that the Mentawai segment of the Sunda megathrust is well advanced in its seismic cycle and is therefore a good candidate for triggered failure.

The 1,300-km-long Sumatra–Andaman rupture of the Sunda megathrust that occurred on 26 December 2004 shed stresses on to other structures in the region. We previously identified two faults of particular concern: the continuation of the Sunda megathrust to the south, beneath the islands of Simeulue and

Table 1 | Hypocentral stresses in the Sumatran earthquake of 28 March 2005

	Slip distribution	
	Ref. 8	Ref. 9
Co-seismic stress	0.005	0.110
Post-seismic stress	0.064	0.060
Total stress	0.069	0.170

Hypocentral stresses are shown in bars and are calculated using wave-form slip inversions^{8,9} and an oceanic earth structure after ref. 10. The difference in co-seismic stresses is largely due to the difference in the southward extent of the 26 December rupture in the two slip models.

Nias, and the vertical, strike-slip Sumatra fault¹. On 28 March, rupture of the Simeulue–Nias segment generated a magnitude-8.7 earthquake, which caused widespread destruction on the islands and is estimated to have killed about 2,000 people.

We have calculated the stresses induced by the Sumatra–Andaman rupture at the hypocentre of the Simeulue–Nias earthquake, including both the co-seismic² elastic effect and the effect of post-seismic³ viscoelastic relaxation of the upper mantle. The total stress perturbation at the hypocentre was small

(Table 1), between 0.07 and 0.17 bars. The size of this triggering stress illustrates the extreme non-linearity of the earthquake nucleation process.

Like its predecessor, the Simeulue–Nias earthquake has appreciably altered the state of stress in the surrounding region (Fig. 1a). Although it changed only slightly the level of stress on the section near Banda Aceh, which was most affected by the Sumatra–Andaman rupture, it has increased stresses on the Sumatra fault south of that section. As in the case of the Sumatra–Andaman rupture, the section of the megathrust just to the south has also been stressed appreciably (by as much as 8 bars on the section beneath the Batu islands and somewhat less on the segment beneath the Mentawai islands). Despite the smaller size of the Simeulue–Nias event, the magnitude of its stress perturbation on the Batu and northern Mentawai sections of the megathrust is similar to that which triggered the Simeulue–Nias earthquake. This stress may be expected to migrate further south over time as a result of viscoelastic effects.

The Batu section of the fault (Fig. 1b), from the Equator to about 0.7° S, last ruptured in 1935 during a magnitude-7.7 earthquake that resulted in about 2.3 metres of slip on a 70 km × 35 km patch of the megathrust^{4,5}. Recent palaeogeodetic studies (manuscript