## NOTE

## Coral mortality associated with dinoflagellate blooms in the eastern Pacific (Costa Rica and Panama)

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ABSTRACT: Coral reefs at Caño Island, Costa Rica, and Uva Island, Panamá, were affected during severe dinoflagellate blooms in 1985. In the second half of 1985, mass mortality of reef fishes and invertebrates, especially reef corals, occurred during blooms of the dinoflagellates  $Cochlodinium\ catenatum$ and Gonyaulax monilata. At Caño Island, up to 100% coral mortality was observed between the surface and 3 m depth, with pocilloporid species and Tubastrea coccinea most severely affected. At Uva Island, only 13 % pocilloporid mortality occurred and this was confined to the shallowest reef areas ( $\leq 3$  m). The copious amounts of mucus associated with C. catenatum, present in the water column to 1-3 m depth, and adhering to coral colonies, suggested that coral death was caused by smothering. Other conditions that may have affected reef organisms were the presence of toxic G. monilata, and possible oxygen depletion due to the high densities of phytoplankton and the decomposition of dead organisms. These dinoflagellate blooms have interfered with recovery of reefs disturbed during the 1982/83 El Niño warming event.

There are only 2 reports of coral death associated with phytoplankton blooms. Baas-Becking (1951) reported on the death of corals in the shallow waters of New Caledonia, and attributed this to the putrefaction of thick masses of *Trichodesmium thiebautii* causing a marked reduction in dissolved oxygen. Smith (1975) commented on the impact of a 1971 'red tide' on reef coral communities off the west coast of Florida, USA, with near-complete mortality of shallow-water (< 40 m) biotas over extensive areas. Smith hypothesized these mortalities were caused by bacterial and fungal infec-

tions, oxygen depletion, and hydrogen sulfide poisoning rather than by toxins of *Ptychodiscus brevis* (Davis) that were present during the event.

In this note we present observations of coral death associated with dinoflagellate blooms in the eastern Pacific – at Caño Island, Costa Rica and Uva Island, Panamá – and discuss possible mechanisms that may have resulted in coral death.

Study site and methods. Caño Island (8°43' N, 83°52'W) is situated in the southern region of the Pacific coast of Costa Rica, ca 15 km offshore (Fig. 1). Information on the coral community structure and climatic and oceanographic conditions is available in Guzmán & Cortés (1989a, b). The percentage of dead colonies of all coral species encountered at Caño Island in July 1985 was recorded. Eight permanent plots of 10 m<sup>2</sup> each at 4 localities from 0.5 to 12 m depth that have been surveyed since January 1984 (for an assessment of coral recovery after the 1982/83 El Niño event) were mapped in January 1985 before the dinoflagellate bloom and in late August 1985 near the end of the bloom. Water around the reefs was collected using 1 l bottles in July 1985 and the species composition and relative abundances of the phytoplankters were determined using a Palmer-Maloney chamber (Steidinger

Uva Island reef (7°49′N, 81°46′W) is located about 40 km offshore in the Gulf of Chiriquí, Panamá, with similar climatic conditions to those at Caño Island. Detailed information on the oceanographic conditions and coral community structure are available in Dana (1975) and Glynn (1976). The same scleractinian corals occur in both study areas; however, at Uva Island the

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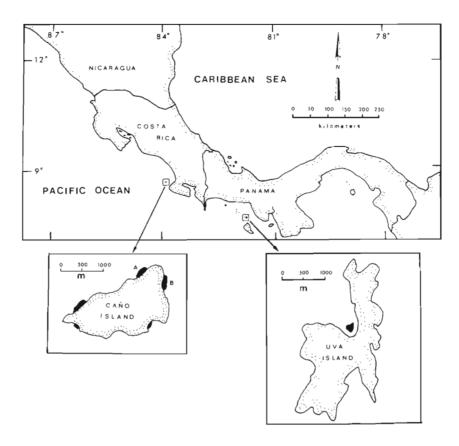


Fig. 1. Southern Central America, with detail of the islands studied: Caño Island, Costa Rica (8° 43′ N, 83° 52′ W) and Uva Island, Gulf of Chiriquí, Panamá (7° 49′ N, 81° 46′ W). Shaded areas are the coral reefs, at Caño Island A = north site and B = east site

Table 1 Percent of dead coral colonies observed on 25 July 1985, during the dinoflagellate bloom at Caño Island, Costa Rica

Taxon	Area	Depth (m)	No. colonies examined	% Dead
Pocillopora spp.	East	0.5–1	175	100
(predominantly P. elegans)		2	15	96
		2-4	96	94
		3-6	34	91
		10-25	71	1
	North	0.5-1	18	83
		1–2	5	0
		2-8	9	100
Porites lobata	East	0.5-1	13	0
		3-10	19	8
	North	1-2	9	0
		2-8	7	0
Gardineroseris planulata	East	5	1	0
	North	2-5	3	0
Pavona clavus	East	1-1.5	3	0
	North	2-5	9	0
Pavona gigantea	North	2-5	6	0
Tubastrea coccinea	East	2-10	95	95
		10	21	0
		10	17	50

relative abundance of pocilloporid corals is higher than at Caño Island. Ten permanent chain transects, between 3 and 6 m depth, were sampled at Uva Island 3 mo before and 2 mo after the dinoflagellate bloom. The chain transects were 10 m long with 73 sampling points (links) m<sup>-1</sup> and the distance covered by each species was recorded (Glynn 1984). The dinoflagellates at Uva Island were not identified to species.

Results. A severe dinoflagellate bloom, or succession of blooms, occurred around Caño Island from 3 June to 12 July 1985, with additional pulses through October (park rangers daily report, Caño Island Biological Reserve). Surface waters, from 2 to 3 m depth, were red-yellow with viscous foam presumably produced by the dinoflagellates. A surface water sample collected at Caño Island in July 1985 contained  $8.3 \times 10^5$  live phytoplankton cells  $l^{-1}$  and more than  $3 \times 10^6$  cells  $l^{-1}$ including dead cells. The relative abundances by cell number were: Cochlodinium catenatum Okamura (97.0 %), Gonyaulax monilata Howell (1.4 %), diatoms (1.2%) and other dinoflagellates (0.4%). Numerous fish were affected, with hundreds of scarids, balistids, acanthurids, pomacentrids and tetraodontids found dead on the beach, as well as dead hermit crabs, brachyuran crabs and gastropods. Some shallow water corals were also affected (Table 1), especially Pocillopora elegans Dana, P. damicornis (Linnaeus) and the azooxanthellate coral Tubastrea coccinea Lesson. The presence of live obligate crustacean symbiotes in some dead pocilloporid corals indicated recent mortality. A significant inverse relation was found between the number of dead colonies and depth (tau = -0.923, p < 0.03, n = 20, Kendall rank correlation). No recently dead or partially dead corals were observed in deeper waters ( $\geq 10$  m).

Between January and August 1985, the percentage cover of live pocilloporid corals in the permanent plots at Caño Island decreased to zero at the shallow stations (0.5 to 3 m depth), while the massive corals remained unchanged (Table 2). The deeper stations (6 to 12 m depth) showed no significant change in live coral cover between January and August 1985 (Table 2).

The Uva Island dinoflagellate bloom was observed in October and November 1985. It had a red-brown color and probably lasted several days. Numerous pocilloporid corals at 1 to 2 m depth had bleached and were sloughing tissues. The co-occurrence of viscous foam and dinoflagellates suggested that the former was produced by the latter. The viscous foam near the surface oscillated vertically with the tides over shallow-water corals, which were most affected. Transect sampling 3 mo before the dinoflagellate bloom and 2 mo after indicated minor coral mortality in the shallowest transects (3 m). In 4 transects at 3 m depth, cover of live Pocillopora spp. was reduced by 12.8 % (Table 3), but the decline was not statistically significant. The increases in coral cover in the deeper transects (Table 3, Transects 5 to 8) were due to fragmentation and the spread of live branches into the sampling area. Reductions also resulted from coral fragmentation and the transport of pocilloporid corals away from a transect (Table 3, Transect 9) and the burial of massive corals by dead pocilloporid branches (Table 3, Transect 10).

**Discussion.** The 1985 dinoflagellate bloom was the most severe reported off the Pacific coast of Costa Rica during the last 15 yr (R. Víquez pers. comm. 1988). Intense upwellings were experienced that year in the Gulf of Panamá, which were related to anti-El Niño type activity (Glynn 1989). These conditions could have caused a general shoaling of the nutricline in non-upwelling areas, ultimately leading to the dinoflagellate bloom observed in 1985.

In previous years at the Gulf of Nicoya, less than 100 km northwest of Caño Island, blooms were also dominated by *Cochlodinium catenatum*, according to Hargraves & Víquez (1981, 1985). This species has not

Table 2. Percent live hermatypic coral cover before and after a dinoflagellate bloom in 10 m<sup>2</sup> plots, Caño Island, Costa Rica. Pocilloporid corals were *Pocillopora elegans* and *Pocillopora damicornis*; other corals were *Pavona varians, Pavona gigantea, Pavona clavus, Psammocora stellata, Gardineroseris planulata* and *Porites lobata* 

Plot no.	Depth (m)	19 January 1985		29 August 1985	
		Pocilloporid corals	All corals	Pocilloporid corals	All corals
1	0.5-1	24.19	27.00	0	3.35
2	0.5-1	30.19	30.81	0	1.73
3	2-3	34.60	37.50	0	2.61
4	2-3	36.90	40.40	0	3.39
5	6	0	30.40	0	27.47
6	6	0	31.57	0	27.50
7	10-12	5.13	25.48	4.25ª	23.35
8	10-12	5.31	20.16	4.91 <sup>a</sup>	16.54

Transect no.	Depth (m)	13 August 1985		18 January 1986	
		Pocilloporid corals	All corals	Pocilloporid corals	All corals
1	3	25.9	25.9	20.7	20.7
2	3	21.5	21.5	21.0	21.0
3	3	23.6	23.6	20.6	20.6
4	3	22.5	22.9	18.9	18.9
5	4	36.8	37.1	40.8	42.0
6	4	19.3	19.7	23.8	24.1
7	5	14.7	14.7	20.8	20.8
8	5	23.7	25.8	24.9	25.5
9	5	14.4	14.4	8.5	8.5
10	6	0	27.1	0	19.7

Table 3. Percent live hermatypic coral cover before and after a phytoplankton bloom, Uva Island, Gulf of Chiriquí, Panamá. Pocilloporid corals were *Pocillopora damicornis* and *P. elegans*; other corals were *Gardineroseris planulata, Pavona varians* and *Psammocora stellata* 

been proven to be toxic, but other species of *Cochlodinium* were toxic to fish in Japan (Yuki & Yoshimatsu 1989). Even though concentrations of dinoflagellates during those blooms were higher than at Caño Island, Hargraves & Víquez (1981, 1985) did not report any massive mortality in the estuarine Gulf of Nicoya. This difference in mortality between Nicoya and Caño may have been due to a greater susceptibility of corals to changes in water quality than most estuarine organisms and because *Gonyaulax monilata* was not found at Nicoya.

The possible mechanisms by which phytoplankton blooms cause mortality include: (a) toxicity, (b) reduction of light penetration, (c) depletion of dissolved oxygen, (d) smothering by mucus, or (e) any combination of the above effects.

Mortality caused by toxic dinoflagellate blooms is well documented from boreal to tropical seas (Steidinger & Baden 1984, Anderson et al. 1985). Of the dinoflagellates observed in the blooms, only *Gonyaulax monilata* is known to be toxic (Steidinger & Baden 1984), but even at low densities its toxicity may cause the death of organisms (Schantz et al. 1975 in Steidinger 1983).

Light reduction during the dinoflagellate blooms did not seem to be critical in the death of corals. Two observations suggest this: (1) the azooxanthellate coral *Tubastrea coccinea*, which is not light dependent, was significantly affected; and (2) corals in deeper water were not affected.

Death of marine organisms attributed to anoxia after phytoplankton blooms has been reported in several marine habitats, including coral reefs (Baas-Becking 1951, Smith 1975) The majority of the fishes and crabs affected at Caño and Uva Islands were active, freeliving members of the epibenthos. Also, as oxygen levels in warm waters are often close to the lower tolerance limits of aerobic organisms (Johannes &

Betzer 1975), oxygen stress may occur on reefs, especially in the presence of decomposing organisms.

An increase in viscosity of the water due to mucus produced by dinoflagellates has been suggested as a possible cause of death of fish (Jenkinson 1989). There are no reports of coral death attributed to smothering by thick mucous layers due to phytoplankton blooms. However, a thick layer of mucus could effectively isolate corals from their environment, preventing feeding, respiration and excretion, and if the blockage were prolonged, the coral would die. Also, some bacteria thrive in the mucus produced by corals and an increase in bacterial density could cause coral death by sulfide poisoning, oxygen depletion and attack of coral tissue by the bacteria (Ducklow & Mitchell 1979, Paul et al. 1986).

Eastern Pacific coral reefs are recovering slowly from the 1982/83 El Niño warming event that caused mass coral mortalities in Panamá and Costa Rica (Glynn et al. 1988). The 1985 dinoflagellate blooms occurred less than 2 yr after this disturbance. At Uva Island, where the El Niño disturbance resulted in 70 to 95% coral mortality, few corals were affected by the dinoflagellate bloom except at shallow depths (≤3 m). At Caño Island, El Niño-associated coral mortality was lower, 25 to 75%, but mortality due to the dinoflagellate bloom was high, completely eliminating some coral species from shallow reef zones.

In conclusion, the death of reef organisms at Caño and Uva Islands was possibly caused by a combination of (a) toxicity, (b) oxygen depletion, and (c) smothering by mucus produced during dinoflagellate blooms. Adhesion of mucus to polypal areas, and the interference with polyp expansion, seemed the most likely cause of mortality in corals. These dinoflagellate blooms, occurring 2 yr after the 1982/83 El Niño warming event, have slowed the recovery of these disturbed reefs.

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