



Coral bleaching and disease: contributors to 1998 mass mortality in *Briareum asbestinum* (Octocorallia, Gorgonacea)

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Abstract

High sea surface temperature associated with the recent El Niño was responsible for widespread coral bleaching and mortality around the globe in 1998. In addition to mortality caused by temperature and bleaching associated stresses, some of the coral mortality could be due to the outbreak of diseases among already weakened hosts. One possible example of this is the October 1998 epizootic affecting *Briareum asbestinum* in the Florida Keys, USA. At Carysfort, Sand Key and Western Dry Rocks, between 75 and 90% of *B. asbestinum* colonies were bleached with prevalence of necroses on bleached colonies ranging from 18 to 70%. Between October 1998 and January 1999, 18 to 91% of colonies on seven 25 × 2 m transects died (mean=68%). In addition, at Carysfort Reef, 65% of necrotic colonies that were tagged in October 1998 were dead by January of the following year. A grafting experiment revealed that lesion-causing infections were transmissible: lesions occurred on 50% of recipient colonies treated with diseased grafts whereas none of the grafts with healthy tissue resulted in disease. Preliminary work to isolate a causative agent yielded a cyanobacterium *Scytonema* sp., although work to confirm its role in the mass mortality is still on-going. By January 1999, when surviving colonies had regained their color and many lesions had healed, the cause of the *Briareum asbestinum* mass mortality or even whether a mass mortality had occurred, would have been difficult to ascertain. By any measure, this was a significant epizootic that would have gone undetected or been attributed to bleaching stress in the absence of our evaluation of the role of an infectious disease.

Introduction

The coral bleaching of 1998 was the most geographically extensive and severe in recorded history, causing significant mortality world-wide (Anon, 1998; Wilkinson et al., 1999; Hoegh-Guldberg (1999)). For the first time, coral reefs in every region of the world were affected by severe bleaching events. In some places (e.g. Singapore [Anon., 1998]) bleaching was documented for the first time. Significant coral mortality was documented in Australia, Indian Ocean, Kenya, the Maldives, the Andamans, the Lakshadweep Islands, and the Seychelles. The significant mortality of dominant acroporids marks this as the most severe bleaching event recorded for the Great Barrier Reef and the cause of a major change in community struc-

ture (Baird and Marshal, 1998). Many massive corals died as a result of the 1998 event – some as old as 700 years (Anon, 1998).

The stress for many of these corals seems to be the result of long-term exposure to high water temperatures associated with a prolonged El Niño Southern Oscillation (ENSO) event (Strong, 1998). The 1997–1998 bleaching event coincided with an unusually strong ENSO disturbance. Tropical seas have undergone warming in the past 100 years (Bottomley et al., 1990; Parker et al., 1995; Brown 1997a; Cane et al., 1997; Levitus et al., 2000). Increases in sea temperature of at least 1–2 °C are expected by 2100 in response to enhanced atmospheric greenhouse gas concentrations (Bijlsma et al., 1995; Hoegh-Guldberg, 1999). Reef-building corals, which are central to

healthy coral reefs, are currently living close to their thermal maxima, and thus become stressed and bleach (expulsion of algal symbionts) if exposed to small increases (1–2 °C) in water temperature. Concomitant with predicted rise in sea temperature, bleaching events are also expected to increase in frequency and intensity. Glynn (1993), Hoegh-Guldberg & Salvat (1995), Brown (1997b) and Hoegh-Guldberg (1999) have pointed to the significance of this trend for reef-building corals. Hoegh-Guldberg (1999) predicts events as severe as the 1998 event will become commonplace within twenty years and bleaching will occur annually in most tropical oceans by the end of the next 30–50 years.

In addition to bleaching, reports of epizootics in the ocean have increased, particularly those affecting corals (Richardson et al., 1998; Harvell et al., 1999). *Acropora palmata* and *A. cervicornis* were almost eradicated from the Caribbean by white band disease (Gladfelter 1982; McClanahan & Muthiga, 1998). *Montastrea faveolata* is declining Caribbean-wide, in part due to yellow blotch disease (Santavy et al., 1999). White Plague is significantly impacting several scleractinian species in the Florida Keys (Richardson et al., 1997). *Aspergillus sydowii* has significantly impacted sea fan corals (*Gorgonia* spp.) Caribbean-wide (Nagelkerken et al., 1996; 1997) and caused high mortality at some Florida Keys sites (Kim & Harvell, in press). Projections for damage due to bleaching have not taken into account the likelihood that some of the impacts ascribed to bleaching are actually caused by stress-facilitated disease outbreaks. Indeed, there is evidence to indicate that bleaching in one coral genus is a bacterial disease and that the probability of infection is temperature sensitive (Kushmaro et al., 1996, 1997; Toren et al., 1998).

We document here an association between bleaching and an epizootic affecting the Caribbean gorgonian coral *Briareum asbestinum*. At the peak of sea surface temperatures in October 1998, and following a sustained period of high temperatures (greater than 30 °C) in the Florida Keys, USA, we observed a bleaching event affecting many *B. asbestinum* colonies also showing necroses. To investigate the possibility that a disease was contributing to the decline of *B. asbestinum*, we recorded prevalence of bleached and necrotic colonies, tagged a subset of those necrotic colonies for monitoring, tested for transmissibility of the necrotic condition, and collected samples for microbiological examination.

Materials and methods

Monitoring

The status of *Briareum asbestinum* populations during bleaching was evaluated by surveying 2–3 transects (25 × 2 m) at Carysfort, Sand Key, and Western Dry Rocks reefs in the Florida Keys. These sites were selected based the presence of *B. asbestinum* on permanent transects which were established earlier to monitor disease of sea fan corals (*Gorgonia ventalina*). The *Gorgonia* monitoring transects radiate from a central buoy at each site and were established to monitor *Gorgonia* infected with *Aspergillus sydowii* (Kim & Harvell, in press). They are therefore haphazard with respect to *B. asbestinum* density. For each colony of *B. asbestinum* on the transects, the presence of necrotic regions were noted. Colonies were considered necrotic if they had lost their characteristic brownish purple color and instead were a pale tan or brown and if tissue was sloughing off the colony. The sloughing tissue and necrosis caused branches to break and in some cases, erode to their bases. Necrotic tissue was also apparent along lateral margins of some upright colonies. The first of two surveys were carried out in October 1998 with the second 10 weeks later in January 1999. All necrotic colonies were also bleached. A subset of bleached, necrotic colonies were tagged to directly determine the fate of necrotic individuals.

Water temperature

Sea surface temperatures (SST) presented here were provided by the National Data Buoy Center (National Oceanic and Atmospheric Agency, www.ndbc.noaa.gov). The data consists of water temperature data collected hourly by a Coastal-Marine Automated Network (C-MAN) station located at Sand Key (24.46 N, 81.88 W). SST data for a period preceding and following the bleaching event (January 1994 – October 1998) were used to calculate monthly means. Summer and winter means were calculated for the entire period using data from three of the warmest and coolest months with respect to SST (i.e., summer=July–September; winter=January–March).

Nature of pathogen

To determine the nature of the necrosis-causing agent, we established in situ experiments to assess disease transmissibility. At Western Dry Rocks (9 October 1998), we grafted two fragments (2–3 cm in length),

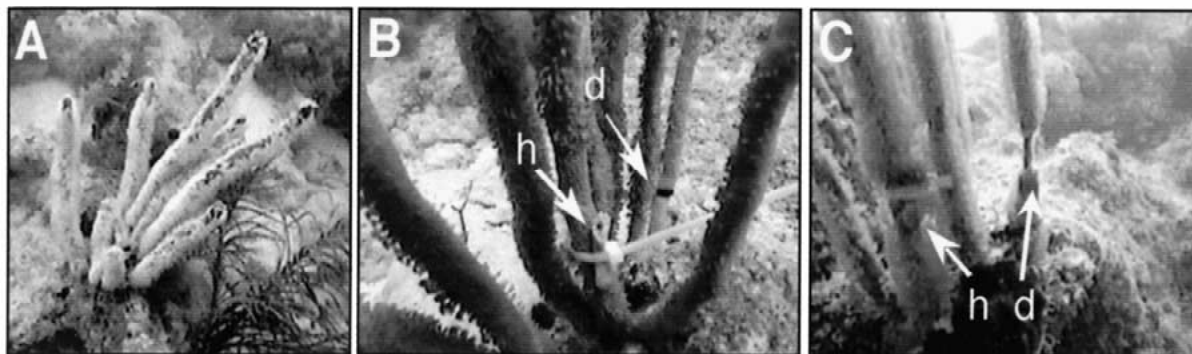


Figure 1. *Briareum asbestinum*. (A) Bleached and necrotic colony. Polyps are white from bleaching; necrosis is visible in these specimens as broken off tips on branches and areas of withdrawn polyps, (B) *in situ* infection experiment at Western Dry Rocks, October 1998. Labels are: h is a healthy graft, d is necrotic region of diseased graft, (C) Results of infection experiment, January 1999. Grafting of diseased (d) fragments resulted in necroses and loss of tissue to the axis whereas grafting with healthy (h) fragments did not.

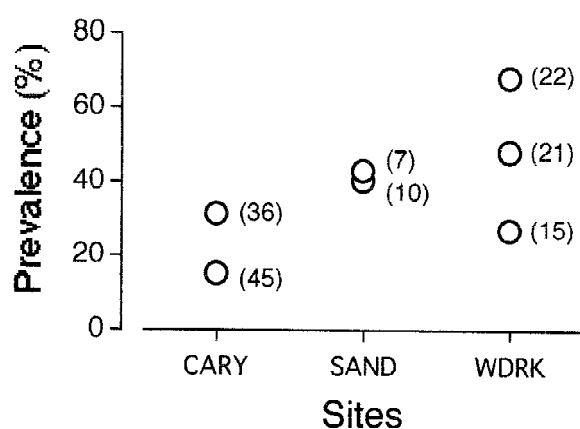


Figure 2. Prevalence of bleached and necrotic colonies from Carysfort and Western Dry Rocks in October 1998. Number colonies per transect are given in parentheses. Abbreviations: CARY=Carysfort, SAND=Sand Key, WDRK=Western Dry Rocks.

one each from diseased and healthy colonies, onto two separate branches on 11 apparently healthy, unbleached colonies (Fig. 1). After two weeks, the grafted fragments were removed and colonies examined. Visual data were collected from underwater videos of each colony. From the videotapes, we scored each lesion as (1) unblemished, (2) with a necrotic patch, (3) with necrosis and significant tissue loss extending to the axial region or (4) with necrosis eroding the fused sclerite axis with branch breakage. Because the cable tie and xenograft caused mild paling and polyp mortality even on control branches, we scored only those colonies with significant necrosis as those where tissue was visibly eroded so that the axial area was exposed.

To begin the process of identifying the infectious agent, we collected in sterile whirl packs thirteen

samples of diseased and six samples of healthy *Briareum asbestinum*. The samples were maintained in plastic bags with seawater at 4 °C to prevent further tissue decay. A cyanobacterium was consistently identified from the 13 samples of diseased tissue and was not present in the six samples of healthy tissue (Fig. 5). Isolation of the cyanobacterium was carried out using both solid and liquid media consisting of various combinations of nutrients and trace elements including nitrogen in three concentrations (25, 30, and 35 mg L⁻¹), 0.01g potassium phosphate, and 1% glucose in either 125 ml sterilized distilled water or 3.2% sterilized seawater. All combinations were kept under constant light and on a slow shaker to provide aeration.

Results

Monitoring

In October 1998, most *Briareum asbestinum* colonies at Carysfort in the northern Keys and Sand Key and Western Dry Rocks Reefs in the southern Keys, near Key West, were bleached – approximately 70–90% overall – and many bleached colonies were necrotic (Table 1). The prevalence of bleached colonies with significant necrosis ranged from 18 to 70%, depending on site (Fig. 2). No unbleached colonies were detected with any necrosis. By January 7 1999, the epizootic and bleaching episode had ended. Necrotic colonies were virtually undetectable (Table 1) and most surviving colonies had regained their pigment. In the 10 week interval, between 18 and 91% of colonies depending on the site, disappeared from the transects and

Table 1. Prevalence of necrotic *Briareum asbestinum* and mortality rates. Percents are given in parenthesis. All necrotic colonies examined October 1998 were also bleached

Site	Transect	October 1998 Necrotic	January 1999	
			Necrotic	Dead
Carysfort	1	7/45 (15)	3/22 (14)	23/45 (51)
	2	11/36 (31)	3/30 (10)	5/36 (17)
Western Dry Rocks	1	10/21 (48)	1/2 (50)	19/21 (90)
	2	15/22 (68)	0/3 (0)	19/22 (86)
	3	4/15 (27)	0/4 (0)	11/15 (73)
Sand Key	1	4/10 (40)	1/7 (14)	3/10 (30)
	2	3/7 (43)	0/1 (0)	6/7 (86)

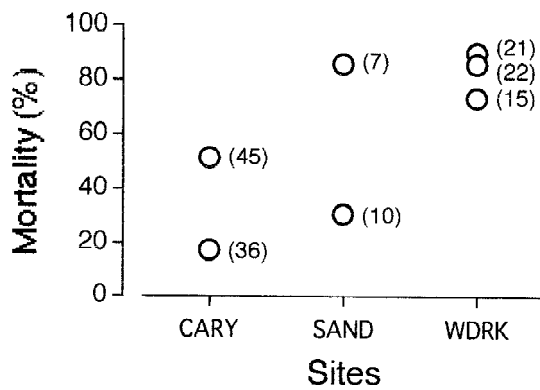


Figure 3. Mortality rates of bleached, necrotic colonies of *Briareum asbestinum* between October 1998 and January 1999. Data shown are % mortality at 2–3 transect each at three sites in the Florida Keys. Number colonies per transect are given in parentheses. Abbreviations: CARY=Carysfort, SAND=Sand Key, WDRK=Western Dry Rocks.

were counted as dead (Fig. 3). Mean mortality across all sites was approximately 68%. Although the data are limited, there appears to be substantial within-site variation in mortality. At Sand Key, mortality differed by more than 40% between the two transects. Mortality was also estimated by following the fates of individually tagged colonies at Carysfort Reef. Of 26 necrotic colonies tagged in October 1998, 17 (65%) were dead by January 1999, just 2.5 months later. Our ability to identify skeletal remains of many colonies adjacent to the numbered aluminum tags is further verification that the missing colonies on the January transects can indeed be ascribed to mortality due to disease.

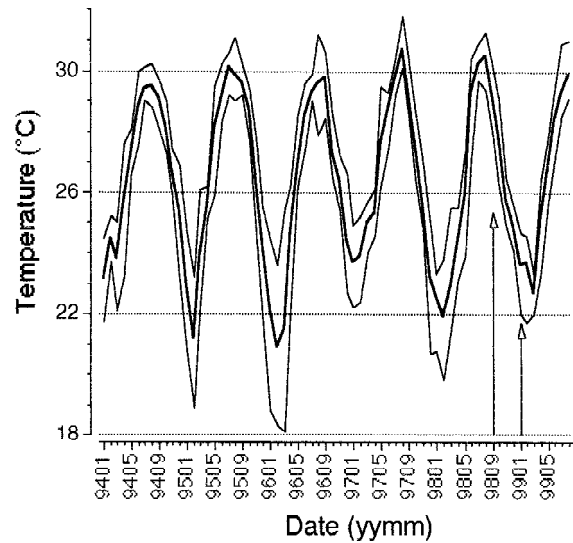


Figure 4. Sea surface temperature (SST) at Sand Key (Florida Keys, USA) for the period of January 1994 – August 1999. Mean monthly SSTs with minima and maxima are shown. Vertical arrows indicate *Briareum* monitoring dates.

Water temperature

There was clear seasonal variation in sea surface temperature (SST) with a maximal average daily SST range of 18.1 °C in the winter months to 31.8 °C in the summer (Fig. 4). Over the period between January 1994 and August 1999, mean summer and winter SST were 29.7 °C±0.192(SE) and 22.9 °C±0.241, respectively. Although it is difficult to discern clear long-term patterns from these data, the two summers preceding the October 1998 *Briareum* bleaching event were warmer than the three previous summers. The four

months immediately preceding the *Briareum* bleaching in October 1998 was marked by monthly SST greater than 30 °C. In 1997, three summer months (June–August) exceeded 30 °C. In comparison, in the three years previous to the 1997–98 warming El Niño event, monthly mean SST exceeded 30 °C only one other time in June 1997.

Infection experiment

Grafting experiments (Fig. 1C) revealed that the necrosis-causing agent is transmissible by contact. Among both treatment and control colonies, the grafts caused localized paling and the loss of polyps at the zone of contact. However, in 5 of 10 treatment grafts using diseased fragments, the recipient branches developed deep necroses, often down to the spicular axis, after 14 days. In contrast, none of the 11 control grafts using apparently healthy fragments developed such necroses.

Pathogen identification

In our culturing efforts, a cyanobacterium was consistently isolated from diseased (13/13) but not healthy *Briareum* tissue (0/6) (Fig. 5). The cyanobacterium associated with the lesions had sheathed trichomes with round ends. Heterocysts near false branches were also observed microscopically, but no akinetes were found. Based on these morphological characteristics, the cyanobacterium was tentatively identified as a member of the genus *Scytonema* (Castenholz, 1989). Efforts are on-going to identify the species. *Scytonema* spp. have been described from terrestrial, aquatic and marine environments, including marine bacterial mats. Although *Scytonema* spp. have not previously been implicated as a marine pathogen, members of this genus are known to produce tolytoxin, tantazole B and an insecticidal glycine-rich peptide (Carmeli, et al. 1990). *Scytonema* spp. tend to bind to surfaces (Castenholz, 1989) and, if bound to the unprotected surface of *Briareum asbestinum*, may then secrete cytotoxic compounds.

Discussion

In spite of increasing reports of coral bleaching and mortality events (e.g. Glynn, 1984; Coffroth et al., 1990; Fabricius, 1999), there is little known about how bleaching and mortality are causally linked. Current understanding of coral physiology suggests bleaching

(the expulsion of photo-symbionts) results in inability to feed photoautotrophically, whereas elevated water temperature causes other physiological stresses, both of which can lead to death if bleaching is prolonged (Meesters & Bak 1993; Jones 1997; Jones et al., 1997; Hoegh-Goldberg, 1999). This study indicates that in the Florida Keys, the outbreak of disease concurrent with, or following onset of bleaching may have caused or accelerated *Briareum asbestinum* mortality. We hypothesize that increased temperature above summertime averages is likely to facilitate the outbreak of opportunistic infection. We suggest that the synergistic effect of a stressed host and an increase in the growth and reproduction of the microbial pathogens at higher temperatures favor the disease outbreak. Because many marine pathogens are unidentified or not easily cultured, there is little information on temperature optima for pathogens. Alker et al. (2001) showed that the fungus *Aspergillus sydowii*, the causative agent of a sea fan epizootic (see Nagelkerken et al., 1996; 1997) has an optimal growth temperature of 30 °C, which is also the typical limit above which bleaching occurs (Strong et al., 1998). Thus, at lower temperatures the fungus may not grow fast enough to overcome host resistance; at higher temperatures the host becomes stressed and perhaps less resistant. An example of the effect of high temperature decreasing resistance in sea fans is the decreased efficacy of sea fan extracts at 30 °C relative to 25 °C (Alker et al., 2001). In another example, Black Band disease (BBD) of scleractinian corals also has an optimal temperature of between 28–30 °C (Rutzler et al., 1983). In the Florida Keys, BBD is most active during the summer and virtually disappears in the winter (Kuta & Richardson, 1996).

The *Briareum* epizootic could have been considered another bleaching related mortality. However, our observation of the bleaching event and subsequent study revealed a high prevalence of necrosis; association of a cyanobacterium implicated the role of disease in the mortality. The grafting experiments, which resulted in half the treatments developing deep lesions in contrast to none of the controls, provided evidence for the role of an infectious agent, possibly the cyanobacterium *Scytonema* sp. Nonetheless, we consider the grafting experiment preliminary because of small sample size (10 controls and 11 treatments); however the epizootic apparently ended by January 1999 and we were unable to conduct additional experiments. In no cases were unusual levels of predation

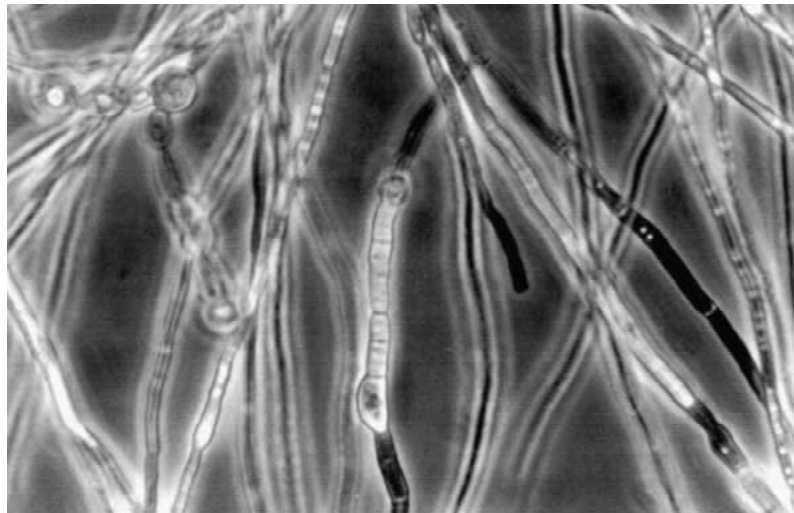


Figure 5. Confocal micrograph of an unidentified species of the cyanobacterium *Scytonema*, the putative pathogen associated with *Briareum* mortality in the Florida Keys.

observed at these sites, so we attribute the *Briareum* mortality largely to the disease agent.

An alternative explanation for the *Briareum* mortality is that the combined stress of Hurricane Georges (early October 1998) and high water temperatures caused bleaching and fatal stress which allowed colonization of necrotic regions by the cyanobacterium *Scytonema* spp. That is, the cyanobacterium could be a component of a fouling community that invades already necrotic areas of the coral. In discussing opportunistic infections of compromised individuals, a distinction needs to be made between an opportunistic infection and a secondary infection taking hold in already necrotic tissue. For example, seals infected with morbillivirus often harbor secondary viral infections (Osterhaus et al., 1994; Lahvis et al., 1995). Similarly in the case of the monk seal, there is still controversy about whether the causative agent of mass mortality in Mauritania is morbillivirus or toxic dinoflagellates (Osterhaus et al., 1997; Hernandez et al., 1998). Confirming *Scytonema* sp. as the primary pathogen and the causative agent of observed disease requires fulfilling Koch's postulates which include: (1) isolation of the agent from diseased tissue, (2) re-infection of a healthy (or in this case bleached host) with the pure culture to produce the characteristic symptoms and (3) re-isolation of the causative agent. Because the *Scytonema* is slow growing, it has not yet been possible to culture significant quantities for use in experiments to confirm it as the causative agent. These microbiological challenges are one reason why many

marine pathogens have remained either unidentified or unconfirmed (Richardson, 1998). However, we can confirm that the infectious agent associated with the *Briareum* mortality is not the sea fan pathogen *Aspergillus sydowii* as we did not detect the fungus in any of the diseased or healthy *B. asbestinum* samples.

The El Niño related bleaching and epizootic among *Briareum asbestinum* was significant, causing approximately 70% mortality at three sites in the Florida Keys (Fig 3). Mortality estimates are derived from the disappearance of colonies on permanent transects between October 1998 and January 1999. Although these estimates show the expected spatial variation both within and between sites, the overall mean mortality for this epizootic was similar to that estimated by recording the fate of individually marked colonies. Indeed, with the individually marked colonies, it was usually possible to locate small (and sometimes substantial) skeletal remnants. Similarly, on the transects were many skeletal remnants with the characteristic purple fused spicular axis of *B. asbestinum* to confirm significant mortality as the fate of missing colonies. This level of mortality will have a severe impact on populations of *B. asbestinum*, particularly because these colonies are long-lived and reproduce by brooding. *Briareum asbestinum* brood oocytes for approximately 9 months and only reproduce once per year; they further suffer high losses at reproduction because of a fertilization limitation and requirement for mates that are nearby (Brazeau & Lasker, 1992).

The *Briareum asbestinum* mortality shares characteristics similar to many marine epizootics (Harvell et al., 1999) in that: (1) it was extremely ephemeral and thus could have been easily missed, (2) the causative agent was unidentified or unconfirmed, and (3) it caused significant mortality. The *Briareum* epizootic was very short-lived; within 10 weeks 65% of colonies were dead and the survivors had recovered pigment and necroses had healed. The ephemeral nature of this and other epizootics can be problematic in the quantitative study of marine diseases. Species subject to seasonal, and temperature-related epizootics are difficult to document because there may be only short intervals in which detection and experimentation is possible. In this regard, corals, in general, may be good candidates as indicator species in the study of marine diseases. For instance, diseases of gorgonian and other corals are more likely to be detected than motile invertebrates because corals are sessile, often die slowly during disease events because of their modular construction and have durable skeletons that remain after death. Surviving *B. asbestinum* were apparent as colonies with truncated branches and healed lesions. Similarly, the sea fan corals affected by a fungal epizootic are recognizable by healed or active lesions in the sea fan blade (see Nagelkerken et al., 1997).

The high rate of bleaching during the 1997–98 El Niño clearly marks corals worldwide as stressed and it seems a reasonable hypothesis that bleaching and temperature-related stress will predispose corals and other tropical reef organisms to opportunistic infections. In addition to *Briareum asbestinum*, we observed significant mortality in two other gorgonian species. During October 1998, there were numerous bleached and necrotic colonies of *Muricea* sp. and *Pterogorgia citrina* in the Florida Keys (C. D. Harvell, unpublished observations); however, due to time constraints, prevalence and mortality data were not taken for these species. We suggest that the high levels of coral mortality throughout the world's tropical oceans, previously attributed to bleaching, may be facilitated or accelerated by opportunistic diseases infecting stressed hosts. An important question for future studies is whether other coral reef organisms are similarly affected by disease or whether the high disease rates detected in corals reflect an unusual vulnerability of this taxon to disease.

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