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Nutrient enrichment enhances black band disease progression in corals

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Abstract Infectious diseases are recognized as significant contributors to the dramatic loss of corals observed worldwide. However, the causes of increased coral disease prevalence and severity are not well understood. One potential factor is elevated nutrient concentration related to localized anthropogenic activities such as inadequate waste water treatment or terrestrial runoff. In this study the effect of nutrient enrichment on the progression of black band disease (BBD) was investigated using both in situ and laboratory experiments. Experimental increases in localized nutrient availability using commercial time release fertilizer in situ resulted in doubling of BBD progression and coral tissue loss in the common reef framework coral *Siderastrea siderea*. Laboratory experiments in which artificially infected *S. siderea* colonies were exposed to increased nitrate concentrations (up to 3 μM) demonstrated similar increases in BBD progression. These findings provide evidence that the impacts of this disease on coral populations are exacerbated by nutrient enrichment and that management to curtail excess nutrient loading may be important for reducing coral cover loss due to BBD.

Keywords Coral disease · Black band disease · Nutrient enrichment · Coral reefs

Introduction

Globally, coral reefs are undergoing detrimental loss and degradation (Hughes et al. 2003). This trend has been particularly severe on reefs of the wider Caribbean,

where an estimated 80% of coral cover has been lost over the past three decades (Gardner et al. 2003). Concurrent with losses in coral cover there have been dramatic increases in the number, frequency, geographic distribution, and host range of coral diseases (Richardson 1998; Harvell et al. 1999; Porter et al. 2001; Richardson and Aronson 2002; Sutherland et al. 2004; Weil 2004). Infectious diseases of corals are now recognized as significant contributors to the degradation observed in coral communities, particularly on Caribbean reefs (Rosenberg and Loya 2004). To date, 18 coral diseases have been described (Sutherland et al. 2004), but only six of these have been characterized in terms of pathogen identification and disease etiology.

Although the reasons for the increases in coral disease incidence and prevalence are largely unknown (Richardson et al. 1998), numerous factors including both natural and local anthropogenic impacts have been suggested as potential contributors to this phenomenon (Jackson et al. 2001; Rosenberg and Ben-Haim 2002). Of particular concern is anthropogenic nutrient enrichment. Potential human-related sources of elevated nutrients to the reef environment include inadequate sewage treatment (Lapointe et al. 1990; Paul et al. 1995; Szmant and Forrester 1996; Lipp et al. 2002), and increased terrestrial runoff related to development (Hallock et al. 1993; Costa et al. 2000). The most common natural, offshore contributions to nutrient enrichment originate from coastal upwelling, occasionally associated with internal tidal bores (Leichter et al. 1996, 2003) or volcanic events (Genin et al. 1995). Both increased nutrient availability and decreased herbivory can lead to phase shifts from coral-dominated to macroalgal-dominated communities (Littler and Littler 1984; Done 1992; Hughes 1994; Steneck and Dethier 1994). Only a limited number of studies have assessed the potential interactions between increased nutrients and reef degradation other than phase shifts. One potential effect may be an enhancement of pathogen-associated coral diseases.

To date, four published studies have included a quantitative assessment of the relationship between

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water quality and coral disease. Kim and Harvell (2002), in a field study of aspergillosis (a fungal infection of sea fans), found that although overall disease prevalence did not vary throughout the Florida Keys, higher levels of dissolved inorganic nitrogen and reduced water clarity (as indicated by chlorophyll *a* and turbidity) were correlated with increased disease severity (i.e., percent of colony area affected by aspergillosis). They emphasized that although a correlation was established, demonstration of a causative relationship would be premature due to limited sample size (five locations) and the fact that water quality parameters were determined using samples that were not collected at the same time or location as the disease surveys. Kuta and Richardson (2002) reported that black band disease (BBD) infected corals occurred in shallower water depths with higher water temperature, higher levels of nitrite, and lower levels of ortho-phosphate than healthy (but BBD-susceptible) colonies in the northern Florida Keys. They concluded, however, that the increased nitrite concentration may have been a result of microbial metabolic processes within the black band consortium itself, such as incomplete reduction of nitrate (Kuta and Richardson 2002). Kaczmarek et al. (2005) observed that both BBD and white plague type II were more prevalent at a sewage-impacted site as compared to an ecologically similar non-impacted site in St Croix, USVI. Although their study did not include measurements of nutrient concentrations during or between sewage bypass events, the relative concentrations of indicator microorganisms provided evidence that untreated sewage runoff increased coral disease prevalence.

Only one published study has experimentally examined the effects of nutrient enrichment on coral diseases. Bruno et al. (2003) found that nutrient enrichment increased the severity of both aspergillosis in sea fans and yellow band disease (YBD) on two *Montastraea* species in Akumal, Mexico. In their study, sea fans experimentally exposed to both aspergillosis (infected tissue) and increased nutrients experienced significantly higher proportions of infected tissue and diagnostic purple galls. Likewise, nutrient enrichment of *Montastraea annularis* and *Montastraea franksii* colonies with naturally occurring yellow band infections resulted in increased rates of disease progression and host tissue loss.

The aim of the present study was to experimentally determine the effect of nutrient enrichment on progression (and coral host tissue degradation) of BBD. This coral disease, caused by a pathogenic microbial consortium, is known to infect 64 scleractinian species, primarily those that form massive boulder-shaped colonies, and six octocoral species (Sutherland et al. 2004). BBD is found worldwide (Sutherland et al. 2004) and is responsible for persistent losses in coral cover (Edmunds 1991; Kuta and Richardson 1996; Borger and Steiner 2005). Furthermore, when coral tissue is lost to BBD, the exposed coral skeleton is commonly colonized by octocorallians and macroalgae and only rarely by scleractinian species (Kuta and Richardson 1997; Edmunds

2000). Outbreaks of BBD have been recorded near sewage outflows or other areas with increased pollution (Taylor 1983; Antonius 1988; Bruckner and Bruckner 1997; Goreau et al. 1998), suggesting that the distribution of BBD may be correlated with human influence. However, with the exception of Kaczmarek et al. (2005), no quantitative data regarding water quality were recorded in these studies. To specifically assess the effects of increased nutrient concentrations on the rate of BBD progression and host coral tissue loss, manipulative experiments were conducted using both naturally occurring BBD infections in situ (modeled after Bruno et al. 2003) and artificially infected coral colonies in the laboratory.

Materials and methods

Study site

This study was conducted on Horseshoe Reef (23°46'18"N, 76°5'33"W) northeast of Lee Stocking Island (LSI) in the Bahamas' Exuma Chain. This reef is characterized by a complex fringing pattern with depths ranging from 4 to 14 m. Reefs near LSI are relatively pristine with negligible environmental stress from sewage discharge, pesticide pollution, or river runoff (Dennis and Wicklund 1993). The area is geographically isolated from human population centers (45 km from Georgetown) and receives little commercial or sport fishing due to a voluntary no-take zone that surrounds LSI. These factors make it an ideal region to study coral disease dynamics with limited human influence.

In situ experiments

Between 16–17 July 2004 and 12–14 July 2005 *Siderastrea siderea* colonies with naturally occurring BBD infections were identified at Horseshoe Reef. Of these, 20 were selected in 2004 and 10 selected in 2005. The colonies were well isolated from one another (at least 5 m apart), were within similar depth ranges (7–12 m), and had lost at least 5% of host coral tissue due to disease progression. The colonies ranged from 20 to 85 cm in diameter and had no other visual anomalies beyond BBD infection. Half of these colonies were randomly selected for experimental nutrient enrichment (ten in 2004, five in 2005) and the remaining half were used as controls. Colony size (approximated by elliptical area of maximum length and perpendicular width) was tested for differences in mean size between treatment groups (*t*-tests) and correlation with disease progression over the duration of the experiment (Spearman's rank).

To assess ambient (background) nutrient availability, water samples were collected in 2004 approximately 10 cm above each of the BBD-infected colonies using sterile 60 ml plastic syringes before initiation of the nutrient dosing experiments. Additionally, water samples

were collected at five other time points prior to and within the timeframe of the experiment (2004 only) to determine ambient nutrient variability over time. These samples were collected in locations >10 m from any experimental colonies. Water samples were immediately placed on ice upon return to the boat and, upon return to shore, filtered through 25 mm diameter GF/F filters before transfer to sterile 60 ml high-density polyethylene bottles. The samples were then frozen (-40°C) for transport to Florida International University (FIU).

Nutrient concentrations near experimental colonies were manipulated using diffusive nylon bags filled with 15 g OsmocoteTM 9-6-12 time release fertilizer (Scotts, Maryville, OH, USA). The bags were attached to bare coral skeleton 5–10 cm behind the advancing BBD line using a latex-coated masonry nail and zip tie (Fig. 1a). Empty nylon bags were used for the control colonies. The fertilizer-filled bags were replaced every 5 days to maintain elevated nutrient concentrations over the duration of the experiments (20 days). Empty nylon bags (controls) were physically touched at 5-day intervals to control for diver contact. At each 5-day interval BBD disease migration was quantified to the nearest mm using calipers and two latex-coated reference nails (inserted at the beginning of the experiment). For these measurements, the caliper was placed against both the nail holding the fertilizer bag and one of the two nails marking the edge of the BBD infection at $t = 0$ to ensure that tissue loss at the exact same point was measured. The average migration distance was thus calculated at two points for each BBD infection (Fig. 1a).

To assess the increase in nutrient availability near the nutrient-dosed experimental corals, 60 ml water samples were collected in 2004 directly above three experimental colonies. The samples were collected at three time points: immediately after fertilizer addition ($t = 0$); 1 day later; and 5 days later. These samples were pro-

cessed in the same manner as the ambient nutrient samples described above. Nutrient concentrations were analyzed only in the first 5-day period of the experiment and with the assumption that reef dynamics were similar following each subsequent nutrient loader replacement.

Nutrient analysis

Samples for nutrient analysis were stored at -20°C upon arrival at FIU for 61 days until analysis. The filtered samples were analyzed for total nitrogen, nitrate, nitrite, ammonium, and soluble reactive phosphate (henceforth referred to simply as phosphate) using an ALPKEM Rapid Flow Analysis 300 (Alpkem Corp., Clackamas, OR, USA) at FIU's Southeastern Environmental Research Center (see Boyer and Jones 2002 for methods).

Laboratory nutrient enrichment

To determine the effects of nutrient addition on BBD progression under known, constant nutrient concentrations, a controlled laboratory experiment was conducted. Six colonies of healthy *S. siderea* (approximately 5 cm² each) were collected on 14 July 2005 from White Horse reef ($23^{\circ}48'14''\text{N}$, $76^{\circ}7'53''\text{W}$, located 5.2 km NW of Horseshoe Reef), a spur and groove formation 4–13 m in depth with high wave action. A no-take zone surrounding LSI prevented coral collection from Horseshoe Reef (the site of the in situ experiment). Each fragment was placed in a 1-l glass beaker inside a flow through seawater raceway in the wet laboratory (wet lab) facility at LSI. Air was bubbled at the surface to circulate water in each beaker. The wet lab was shaded by two layers of neutral density screen, reducing direct light exposure to the experimental colonies. After allowing the corals to acclimate for 1 day, four colonies

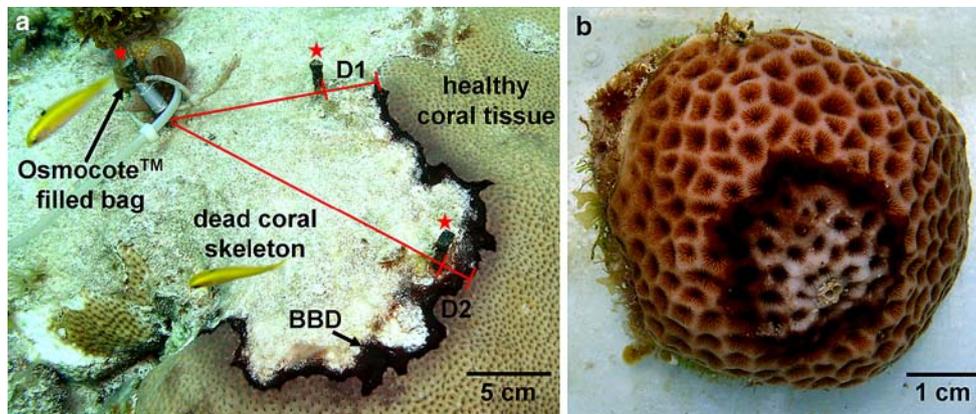


Fig. 1 *Siderastrea siderea* infected with black band disease (BBD). Advancing BBD separates dead coral skeleton from apparently healthy tissue. **a** In situ naturally infected colony with Osmocote-filled nutrient loading bag. Migration measures were recorded at two points on each colony using reference nails (indicated by stars)

as shown by the bracketed areas on the red lines. For each colony these two distances ($D1$ and $D2$) were averaged to determine migration at each time interval. **b** Artificial infection under $2\ \mu\text{M}$ nitrate treatment at day 6. Note that BBD infections in the laboratory were thinner and less dense than those tested in situ

were artificially infected with fresh BBD collected from a single naturally infected *S. siderea* colony on Horseshoe Reef. For inoculation, approximately 0.25 ml of clumped BBD was placed directly on the center of each colony and monitored visually to ensure the attachment of the BBD to the surface of each colony. The four artificially infected colonies were exposed to ambient, +1, +2, and +3 μM nitrate (NaNO_3) in filtered seawater. Two colonies were left as uninfected controls. Of these, one was exposed to ambient nutrient concentrations while the other was exposed to 2 μM nitrate. Time series photographs (e.g., Fig. 1b) were taken twice daily (morning and evening) and used with a grid overlay to estimate band migration rates (to the nearest 0.5 mm) over 10 days. A water change was performed for all treatments at day 5.

Statistical analysis

Differences in nutrient concentrations between ambient and experimentally enriched conditions were tested using the non-parametric Mann–Whitney *U*-test and Bonferroni-adjusted significance levels. To test for differences in the BBD migration distance between nutrient-enriched and control BBD infections in situ, and in the laboratory experiment, repeated measures analysis of variance (ANOVA) was used. Statistical procedures were conducted with SPSS 13.0.

Results

Quantification of in situ nutrient enrichment

Ambient nutrient concentrations were generally low with minimal variability across Horseshoe Reef (Table 1). Furthermore, ambient nutrient concentrations appeared to remain relatively constant over the duration of the experiment. Nutrient levels in samples from five

different time points on Horseshoe Reef, collected > 10 m from the experimental colonies, did not differ significantly from the ambient conditions recorded at the onset of the study (one sample *t*-tests, $\alpha = 0.05$).

Immediately after the initiation of the nutrient dosing experiment on 17 July 2004 (Table 1) nitrate, nitrite, ammonium, and phosphate concentrations increased significantly compared to ambient conditions directly above the colony (0.1 m). After 1 day nitrite, ammonium, and phosphate concentrations were significantly greater than ambient. After 5 days concentrations of all three nitrogenous compounds were significantly greater than ambient conditions, however, there was no quantifiable phosphate enrichment.

In situ experiment

Mean colony size did not differ between nutrient-dosed and control groups in 2004 or 2005 (2004: control $8,364 \pm 8,319 \text{ cm}^2$, nutrient dosed $8,875 \pm 9,130 \text{ cm}^2$; 2005: control $4,273 \pm 1,460 \text{ cm}^2$, nutrient dosed $5,576 \pm 1,972 \text{ cm}^2$ means \pm SD). There was no significant correlation between colony size and total BBD migration distance in either year.

Naturally occurring BBD infections on *S. siderea* migrated more rapidly when exposed to elevated nutrient concentrations (Fig. 2). In 2004 (Fig. 2a) repeated measures ANOVA indicate that time interval ($F = 20.90$, $p < 0.001$), treatment (between groups, $F = 80.42$, $p < 0.001$), and the interaction between the two ($F = 3.77$, $p < 0.02$) were all significant factors in BBD migration on *S. siderea* (sphericity assumed, Mauchly's $W = 0.445$, $p > 0.1$). At the conclusion of the experiment nutrient-dosed infections had migrated on average 25.4 mm compared to 10.8 mm in the controls. Likewise in 2005 (Fig. 2b) differential migration was observed between control and nutrient-dosed BBD infections. Again time ($F = 53.77$, $p < 0.001$), treatment (between groups $F = 16.77$, $p < 0.001$), and the

Table 1 In situ nutrient concentrations during the 2004 black band disease (BBD) nutrient dosing experiment

	Date	N	Nutrient concentration (μM)			
			Nitrate	Nitrite	Ammonium	Sr phosphate
Ambient	12 July	1	0.31	0.03	0.18	0.02
	17 July	20	0.28 ± 0.09	0.05 ± 0.01	0.19 ± 0.08	0.03 ± 0.02
	18 July	1	0.36	0.04	0.19	0.04
	22 July	1	0.41	0.05	0.12	0.03
	27 July	1	0.30	0.05	0.11	0.01
	2 August	1	0.40	0.06	0.21	0.02
Experimental	17 July	3	$1.36 \pm 0.49^*$	$0.14 \pm 0.03^*$	$2.49 \pm 1.40^*$	$0.25 \pm 0.11^*$
	18 July	3	0.55 ± 0.11	$0.15 \pm 0.02^*$	$2.11 \pm 0.54^*$	$0.10 \pm 0.02^*$
	22 July	3	$0.61 \pm 0.15^*$	$0.11 \pm 0.01^*$	$1.03 \pm 0.25^*$	0.03 ± 0.00

Ambient nutrient concentrations were measured before adding nutrient loaders at a distance of 0.1 m from the BBD-infected colonies ($n = 20$). Additional ambient nutrient concentrations are reported as averages of duplicate analyses on split single samples collected from locations > 10 m from experimental colonies. Experimental nutrient concentrations were measured immediately after deployment of the nutrient loaders on 17 July, and again 24 h and 5 days later. Concentrations are shown as mean \pm SD (when available). Experimentally dosed nutrient concentrations that differ significantly from ambient are indicated by an asterisk (Mann–Whitney *U*-test, $p < 0.017$).

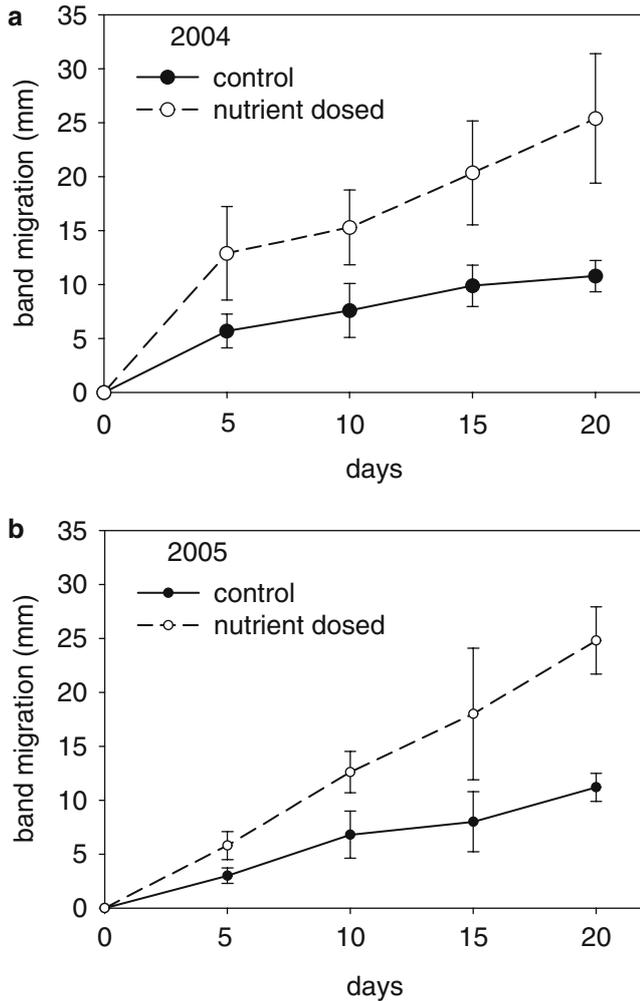


Fig. 2 Migration of in situ black band disease on nutrient dosed (opened circle) and control (filled circle) *Siderastrea siderea* colonies at 5-day intervals over 20 days (mean \pm SD). Standard deviations shown represent the variation observed among colonies within each experimental group (controls and nutrient dosed) at each 5-day interval. **a** 17 July–7 August 2004: experimental $n = 10$, control $n = 10$. **b** 15 July–5 August 2005: experimental $n = 5$, control $n = 5$

interaction between both factors ($F = 8.02$, $p < 0.01$) were significant (sphericity assumed, Mauchly's $W = 0.398$, $p > 0.2$). After 20 days nutrient-dosed BBD infections had migrated on average 24.8 mm while control infections had migrated only 11.2 mm.

Laboratory experiment

When artificially BBD-infected *S. siderea* colonies were exposed to elevated levels of nitrate in a laboratory setting, increasing rates of tissue loss were observed with increasing nitrate concentration (Fig. 3). Repeated measures ANOVA indicated differences in the slopes of BBD progression for the four nitrate treatments, evidenced by the significance of the interaction between

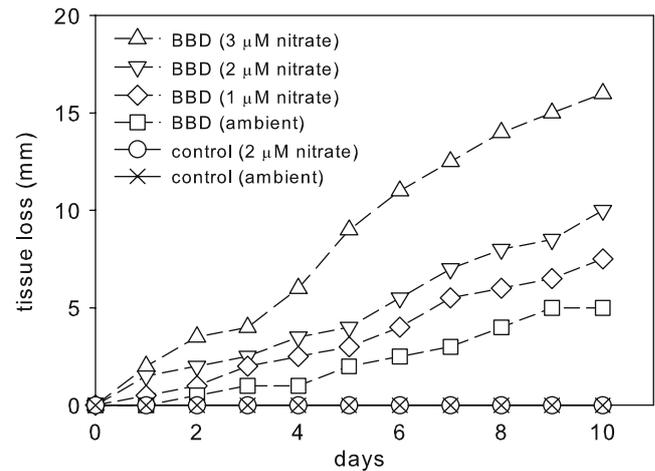


Fig. 3 Tissue loss associated with migration of black band disease on artificially infected *Siderastrea siderea* colonies and non-infected control colonies exposed to nitrate enrichment ranging from ambient to +3 μM in the laboratory

treatment and time ($F = 6.01$, $p < 0.05$). BBD migration rates in the 2- and 3- μM treatments were approximately two and three times, respectively, the rate observed for BBD exposed to ambient nutrient conditions. Neither of the two non-infected control colonies experienced tissue loss over the duration of the experiment.

Discussion

The BBD migration rates measured in this study under ambient nutrient concentrations both in situ at Horsehoe Reef ($\sim 0.6 \text{ mm day}^{-1}$) and in the laboratory ($\sim 0.5 \text{ mm day}^{-1}$) were generally low and independent of host colony size. These rates were similar to an average BBD progression of 0.81 mm day^{-1} observed by Borger and Steiner (2005) in Dominica. Only the nutrient-dosed BBD infections during the first 5-day interval in the 2004 in situ experiment (mean = 2.6 mm day^{-1}) approached the average migration rates of 3 mm day^{-1} reported elsewhere (Rützler et al. 1983; Richardson 1996). None of the infections observed in this study exhibited rates near the maximum of 6.2 mm day^{-1} reported by Rützler et al. (1983). With these generally slow migration rates over the 20-day duration of the in situ experiment, complete coral host mortality was not observed in control or nutrient-dosed colonies. The lack of total colony mortality within 20 days is not uncommon as medium to large colonies are more often affected by BBD (Borger 2005). It may take months for BBD to completely kill such coral colonies, or even years with recrudescing infections on large colonies.

The results of both the in situ and laboratory experiments in this study suggest that increases in nutrient availability can exacerbate the effect of BBD on coral hosts. In situ nutrient-dosed BBD infections migrated on average twice as quickly as control infections

(1.3 mm day⁻¹ vs. 0.6 mm day⁻¹). Nitrate concentrations also directly affected BBD migration rates in the laboratory experiment. Compared to an average rate of 0.5 mm day⁻¹ under ambient nutrient conditions, rates in the nitrate treatments were 0.7 mm day⁻¹ (1 μM), 1.0 mm day⁻¹ (2 μM), and 1.7 mm day⁻¹ (3 μM). The observed rates from the 3 μM nitrate and ambient nutrient treatments in the laboratory were similar to those observed in situ under nutrient-enriched (N and P compounds) and control treatments, respectively. While this suggests that nitrogenous compounds may be limiting, additional investigations in which both nitrate and phosphate are manipulated independently are needed to elucidate the relative roles and potential synergistic effects of these nutrients on BBD migration.

Similar increases in disease migration rates have been observed during in situ nutrient dosing experiments for YBD in studies by Bruno et al. (2003). These investigators found that nutrient enrichment increased average rates of coral host tissue loss by 1.8× relative to controls over the 90-day experiment. Bruno et al. (2003) hypothesized that increased nutrient concentrations potentially enhance pathogen fitness and virulence. However, as there is no known pathogen for YBD, the mechanism(s) by which nutrient enrichment facilitates increased YBD progression rates remain unknown. This study differed from that of Bruno et al. (2003) in that the initial mass of nutrient addition was less (15 g compared to 30 g). This factor is likely responsible for the more moderate nutrient increases observed at deployment. In both studies increases in nutrient availability diminished over time. However, Bruno et al. (2003) reported nutrient concentrations after 4 days that were still greater than ambient, while in this study nitrate and phosphate concentrations returned to ambient concentrations more quickly over time.

The mechanisms that enhance BBD migration rates under increased nutrient exposure are not yet understood but may be related to the fact that BBD is a complex microbial infection. The BBD consortium includes two populations of gliding, filamentous microorganisms, cyanobacteria, and sulfide-oxidizing *Beggiatoa* spp. (Garrett and Ducklow 1975; Rützler et al. 1983; Taylor 1983; Richardson 1996), along with sulfate-reducing bacteria (*Desulfovibrio* spp., Garrett and Ducklow 1975; Schnell et al. 1996; Cooney et al. 2002; Viehman et al. 2006) and at least 50 other heterotrophic bacterial species (Cooney et al. 2002; Frias-Lopez et al. 2002). An additional source of nutrients, i.e., other than those released by coral tissue lysis (from nutrient-enrichment manipulations or other sources as discussed above), may enhance growth rates and/or motility (Grossart et al. 2001) for one or many of the various BBD microbes.

To date there has been only one published study concerning the ecological physiology of a BBD microorganism (Richardson and Kuta 2003). In their study it was found that a culture of one BBD cyanobacterial isolate, as well as freshly collected BBD samples (both

from the Florida Keys), were unable to fix nitrogen as determined by the acetylene reduction technique (Richardson and Kuta 2003). Therefore an increase in available nitrogenous compounds, from any source, may release this organism (and other non-nitrogen fixing BBD cyanobacteria) from nitrogen limitation on the reef. The relative contributions of water column nutrients and nutrients derived from lysed coral tissues to BBD virulence and migration rates remain unknown.

The increases in BBD severity observed in this study due to nutrient enrichment may also be related to coral host stress. Multiple studies have shown decreased coral growth rates when nutrient (inorganic nitrogen and phosphorus) concentrations are increased (Stambler et al. 1991; Ferrier-Pages et al. 2000; Renegar and Riegl 2005). These short-term reductions in coral growth are likely linked to increased competition for CO₂ between calcification and photosynthesis as zooxanthellae densities increase (Szmant 2002). Nutrient enrichment has also been shown to reduce coral fecundity, fertilization, and recruitment (Hunte and Wittenburg 1992) and can lead to increased coral mortality (Kuntz et al. 2005). Elevated nutrients may reduce the coral host's ability to counteract infection by pathogenic microorganisms. There are, however, some cases in which nutrients have had no effect (Taylor 1978) or even positive effects (Atkinson et al. 1995) on coral growth.

While nutrient inputs can increase disease prevalence (Kaczmarek et al. 2005) and exacerbate infections (Bruno et al. 2003; this study), they are not necessary for coral diseases to occur. Multiple coral diseases have been observed near LSI (Voss and Richardson 2006) and in Bonaire (Weil et al. 2002), both relatively pristine regions of the Caribbean. Likewise, both BBD and aspergillosis have been reported in pristine areas by Edmunds (1991) and Nagelkerken et al. (1997), respectively. Therefore other environmental drivers with more widespread regional effects, such as rising sea surface temperatures, may be more important contributors to the observed increases in coral disease incidence and prevalence throughout the Caribbean. Nonetheless the results of this study provide evidence that localized nutrient levels play a role in BBD dynamics and support the proposal of Bruno et al. (2003) that management to curtail excess localized nutrient loading may be important for reducing coral cover loss to disease.

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