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REPORT

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Ecological aspects of black band disease of corals: relationships between disease incidence and environmental factors

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Abstract Eleven environmental factors (salinity, water depth, water temperature, nitrate, nitrite, ammonium, soluble phosphate, total phosphate, turbidity, coral diversity, and percent coral cover) were measured at 190 sites on 12 patch reefs of the Florida Keys. Each (2-m-diameter) site was centered around a coral colony with active black band disease ($n = 21$) or a haphazardly selected healthy coral of a species known to be susceptible to black band disease ($n = 169$). Statistical analysis was performed to detect any relationship between each environmental factor and black band disease incidence. Five factors (water temperature, water depth, coral diversity, and concentrations of orthophosphate and nitrite) exhibited statistically significant relationships with black band disease. These are discussed in terms of the etiology of the disease as well as the reef environment.

Keywords Black band disease · Coral reef ecology · Coral reef health

Introduction

One of the most important, yet least understood, aspects of the observed degradation of coral reefs throughout the world is the relationship between coral disease incidence and environment (Richardson 1998). There is now a clear correlation between coral bleaching and increased seawater temperature (Brown 1997), and an understanding of the relationships between thermal-induced bleaching and both coral and zooxanthellae physiologies (Fitt et al. 2001). While it has been suggested that coral disease may also be associated with a decline in reef environmental quality (Harvell et al. 1999; Porter et al. 1999; Green and Bruckner 2000), surprisingly little quantitative work has been carried out in this area.

We now know that four coral diseases are associated with high water temperature. Black band disease (Antonius 1981, 1985; Rützler et al. 1983; Taylor 1983; Edmunds 1991; Kuta and Richardson 1996; Bruckner et al. 1997), plague (Dustan 1977; Richardson et al. 1998), and the newly described dark spots disease (Gil-Agudelo and Garzon-Ferreira 2001) occur primarily during the warm months of the year when water temperature is above 28 °C. Aspergillosis, which occurs year round, was found to be most active during the warmest months (Alker et al. 2001). Nutrient (sewage) input, sedimentation, and runoff have also been implicated as potential factors that may promote coral disease incidence. Taylor (1983), Antonius (1988), and Bruckner et al. (1997) reported an increase in black band disease activity on reefs that were close to sewage outflows and/or exposed to high sediment deposition; however, no measurements were made. Only one quantitative study to date has been published in which a relationship between water quality and disease prevalence was examined (Kim and Harvell 2002). This study, an investigation of aspergillosis distribution in the Florida Keys, revealed that the disease was more prevalent in waters with relatively higher dissolved inorganic nitrogen and slightly lower water clarity (as indicated by increased turbidity and chlorophyll *a*). The authors concluded that while their results demonstrated a correlation between several measures of poor water quality and disease, the analysis was based on a limited sample set of five stations, and did not prove a causal relationship.

We report here results of a study aimed at quantitatively examining the relationships between black band disease and 11 environmental parameters that may be important in disease occurrence.

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Methods

The potential relationship between environmental (biotic and abiotic) factors and occurrence of black band disease was investigated using an adaptation of Larson and Bock's (1986) bird-centered analysis (BCA) method, originally developed for determining avian habitat preference. The premise of this approach is that identification of environmental factors that are important in the ecology of birds may be determined by comparing environmental factors at sites with and without birds.

In our study, a suite of environmental parameters that may influence black band disease incidence was measured. These were salinity, water depth and temperature, nutrients (nitrate, nitrite, ammonium, soluble phosphate, and total phosphate), turbidity, coral diversity (Shannon-Weaver index), and percent coral cover. One hundred and ninety sites (defined below) on 12 patch reefs were sampled in the middle to northern Florida Keys to provide data for the BCA analysis. These included 21 sites with black band disease and 169 haphazardly selected sites with healthy (but black band disease susceptible) corals (Table 1). Haphazard selection of sites occurred as follows. A compass bearing was determined by looking at the second hand of a dive watch and noting the position on the dial at that moment in time, such that a position of 15 s = 90°, 30 s = 180°, etc. After orienting to this bearing the position of the second hand was again viewed, and the diver then swam for this time period (number of seconds) along the bearing. The nearest coral colony of a species known to be susceptible to black band disease on these reefs was then chosen as the center of a 1-m-radius study site. Black band disease susceptible coral species on these reefs are *Montastraea annularis*, *M. cavernosa*, *Diploria labyrinthiformis*, *D. strigosa*, *D. clivosa*, and *Colpophyllia natans*. Black band diseased corals were not selected randomly; during the (six-month) study period, any coral observed to have active black band was used as a study site. The specific location of each of the black band diseased corals was recorded, and no individual black band diseased coral was resampled over the period of the study.

Our study included reefs from Alligator Reef to South Carysfort Reef, a linear distance of approximately 60 km (Fig. 1). All sites were between 2 and 5 km offshore. Sampling was conducted between April and August (1994), a period that falls within the "active" black band disease season for the reefs of the Florida Keys.

Table 1 Reefs surveyed for the BCA analysis of environmental factors associated with black band disease incidence. Each site consisted of a 1-m-radius area centered around a black band disease infection (*BBD site*) or the center of a healthy coral colony of a species susceptible to black band disease (*non-BBD site*)

Reef [a]	Total no. of sites	No. of BBD sites	No. of non-BBD sites
Algae Reef	36	8	28
Alligator Reef	10	0	10
Cannon Patch Reef	1	0	1
South Carysfort Reef	26	6	20
Elbow Reef	10	0	10
French Reef	15	0	15
Grecian Rocks	29	4	25
Hens and Chickens Reef	10	0	10
Horseshoe Reef	19	2	17
Key Largo Dry Rocks	12	1	11
Molasses Reef	21	0	21
Pickles Reef	1	0	1
Total	190	21	169

[a] All reefs are marked on local navigation charts with the exceptions of Algae and Horseshoe. These are located at 25°08.80′N, 80°17.58′W (Algae Reef) and 25°08.40′N, 80°17.63′W (Horseshoe Reef)

Underwater field sampling and measurements were performed while SCUBA diving. Water samples were collected above (< 5 cm distance) diseased and haphazardly selected healthy coral colonies using both sterile 60-ml syringes and sterile 125-ml Nalgene bottles. The syringe samples were filtered through GF/F filters, labeled, and stored on ice immediately after each dive upon return to the boat. Samples were then stored frozen until analysis for dissolved nutrients. Salinity, turbidity and total phosphorus were determined from the 125-ml bottle samples (unfiltered but similarly frozen). Water temperature at each site was measured using a laboratory thermometer. (Pressure testing of the thermometer did not reveal any demonstrable effect of pressure on temperature for the depths encountered in this study.) Water depth was determined using a diver's depth gauge, and depth measurements were standardized to tidal stage using the Tidemaster computer program. Coral cover (percent cover) and diversity (Shannon-Weaver index) were determined within a 1-m-radius circular area centered in the middle of the black band disease infection or the center of a healthy coral colony. A radius of 1 m was chosen to define the immediate environment of each coral. Within this area, percent coral cover was estimated visually and assigned to categories such that 1 = 0–25% coral cover, 2 = 25–50%, 3 = 50–75%, and 4 = 75–100% cover. Diversity (H′) was calculated based on the number of coral colonies. All scleractinian coral colonies were counted (including corals not susceptible to black band). Gorgonians were not included.

Salinity was measured using an Orion 140 salinity meter (Orion Research Inc., Boston, Massachusetts). Nutrient concentrations (nitrate, nitrite, ammonium, soluble phosphate, and total phosphate) were determined using rapid flow analysis on an ALPKEM RFA 300 (Alpkem Corp., Clackamas, Oregon). Analytical methods followed FIU's SERC standard operational procedures, as detailed in Boyer and Jones (2002). Turbidity was measured with an HF Instruments DRT15C turbidometer (HF Scientifics Inc., Ft. Meyers, Florida).

Data were statistically analyzed using the Mann-Whitney U test. This test was selected based on the small sample size involved (only 21 black band disease sites as opposed to 169 non-black band sites), which required use of a non-parametric approach.

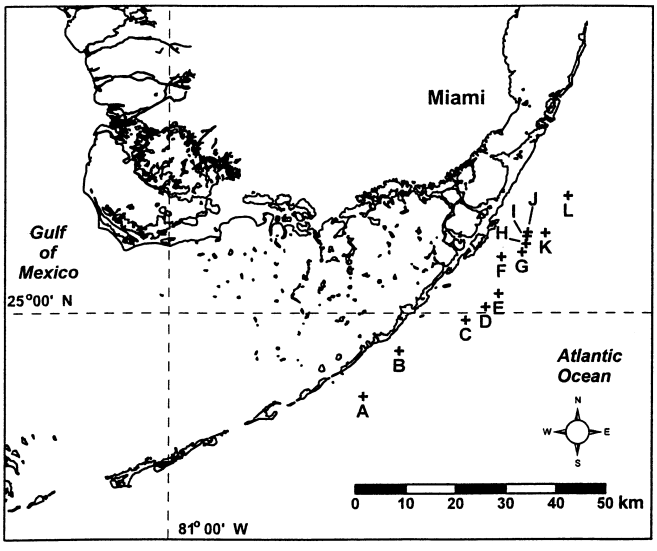

Fig. 1 Location of study reefs along the northern to middle Florida Keys. Codes are as follows: *A* Alligator Reef; *B* Hens and Chickens Reef; *C* Pickles Reef; *D* Molasses Reef; *E* French Reef; *F* Cannon Patch Reef; *G* Grecian Rocks; *H* Key Largo Dry Rocks; *I* Horseshoe Reef; *J* Algae Reef; *K* Elbow Reef; *L* South Carysfort Reef

Results

The means of five of the eleven environmental parameters studied exhibited statistically significant differences between those sites with and without black band disease (Table 2). These were water temperature, water depth, coral diversity, and concentrations of ortho-phosphate and nitrite.

Black band disease was present at sites that were significantly shallower than sites without black band disease (means of 4.1 vs. 6.1 m, $p < 0.0005$; range = < 1 to 19 m). We found no black band disease deeper than 6.6 m. Water temperature at black band disease sites was significantly higher than at non-disease sites, with average values of 29.0 vs. 28.0 °C ($p < 0.0005$). All black band disease sites had temperatures from 29.0 to 30.0 °C, with one exception of 26.0 °C. Non-black band disease sites exhibited a temperature range of 22.8 to 30 °C. Nitrite concentrations at black band sites were significantly higher than concentrations at healthy sites (means of 0.19 vs. 0.14 μM, $p < 0.005$), while ortho-phosphate concentrations were significantly lower (0.12 vs. 0.19 μM, $p < 0.05$). Coral diversity was lower at black band disease sites than at healthy sites (Shannon-Weaver index values of 1.6 vs. 2.0, $p < 0.05$). It should be noted that the standard deviations (SD) of all data reported in Table 2 were calculated with the exception of coral diversity, for which SD was estimated.

We found no statistically significant differences between sites with and without black band disease for the parameters of percent coral cover, turbidity, salinity, ammonium, nitrate, or total phosphate.

Discussion

Our finding of a statistically significant difference between black band disease and shallow vs. relatively deeper water depth agreed with the results of previous studies (Rützler et al. 1983; Taylor 1983; Antonius 1985). It has been suggested that the shallow distribution pattern of black band disease is a function of light limitation at deeper depths, since black band is dominated by a photosynthetic cyanobacterium (Antonius 1981). However, studies aimed at determining the light responses and photosynthetic capabilities of the black band cyanobacterium (*Phormidium corallyticum*) indicated that this species, like many other mat-forming cyanobacteria, prefers very low light intensities. This preference was exhibited by both in situ motility patterns in response to changing light intensity (Viehman and Richardson 2002) and laboratory studies of photosynthesis vs. irradiance of *P. corallyticum*. In the latter case, the threshold for maximum photosynthetic rate (Pmax) occurred at a light intensity of only 27.9 μE $m^{-2}s^{-1}$, a value that was consistent across varied temperatures (Richardson and Kuta 2002).

The occurrence of black band disease in relatively shallow waters may be interpreted in the context of the physiological ecology of the black band disease microbial consortium (Richardson et al. 1997). A very low light level requirement for Pmax is often accompanied by a behavioral strategy termed "self-shading" in cyanobacterial populations, in which gliding filamentous cyanobacteria (such as *P. corallyticum*) intertwine to form clumps that minimize light exposure to individual filaments (Castenholz 1982). In the case of black band disease, the response to high light (such as found in shallow reef waters) involves contraction, or clumping, of black band cyanobacterial filaments, resulting in a low-light microenvironment within the band (Richardson 1996; Viehman and Richardson 2002). This is potentially important for two of the other major black band disease consortium members, sulfide-oxidizing and sulfate-reducing bacteria (see Richardson et al. 1997). The dense cyanobacterial clumping may be integral to the development of an anoxic microenvironment within the band matrix that in turn would enrich for and support growth of populations of *Desulfovibrio* and *Beggiatoa* spp.

Our finding of a statistically significant relationship between black band disease occurrence and elevated nitrite is of interest in terms of the only other quantitative study to date of nutrients and coral disease incidence. As noted previously, Kim and Harvell (2002)

Table 2 Results of the BCA analysis of environmental factors associated with black band disease

Parameter	Mean w/BBD	SD w/BBD	Mean non-BBD	SD non-BBD	U value	*n*	df	*p*
Depth (m)	4.1	1.53	6.1	2.24	2,778.0	190	1	< 0.0005
Temperature (°C)	29.0	0.75	28.0	1.95	1231.5	190	1	< 0.0005
Nitrite (μM)	0.19	0.079	0.14	0.086	1,099.0	190	1	< 0.005
Ortho-P (μM)	0.12	0.239	0.19	0.279	2,249.0	190	1	< 0.05
Diversity	1.6	0.40	2.0	0.50	2,251.5	190	1	< 0.05
Ammonium (μM)	2.79	3.988	3.54	5.689	1,881.5	190	1	< 0.1
Salinity (ppt)	35.7	0.380	36.0	0.420	1,980.5	187	1	< 0.1
Total-P (μM)	0.08	0.014	0.08	0.030	1,621.0	187	1	< 0.1
Turbidity (NU)	0.397	0.2095	0.669	0.8386	1,578.5	187	1	< 0.1
Nitrate (μM)	0.72	0.589	0.65	0.567	1,613.0	190	1	< 0.5
Coral cover (%)	2	0.8	2	1.0	1,606.0	190	1	< 0.5

found a higher prevalence of aspergillosis on reefs of the middle and southern Florida Keys with relatively higher dissolved inorganic nitrogen (ammonium, nitrate, and nitrite). These reefs were near Key West (the southernmost of the Florida Keys), which has one of only two Florida Keys sewage (secondary) treatment plants. Both of these are located in the southern Keys, and each has an outflow to adjacent coastal waters (Kruczynski and McManus 2002). The rest of the Keys have either individual residential septic tanks or small waste-water treatment facilities with deep-injection wells for sewage disposal (Kruczynski and McManus 2002).

Three potential sources of nutrient enrichment exist for our study reefs: sewage contamination of ground waters from septic tanks and deep-well injections (Lapointe et al. 1990; Paul et al. 1995a, 1995b; Brand 2002), upwelling associated with internal tidal bores (Leichter et al. 1996; Smith and Pitts 2002), and surface water transport of relatively nutrient-rich water from Florida Bay (Porter et al. 1999; Lapointe et al. 2002). Several investigators (Lapointe et al. 1990; Paul et al. 1995a, 1995b) have hypothesized that sewage from septic systems and canals in the Florida Keys is being transported to the offshore reefs; however, supportive data are based only on samples collected in canals or directly adjacent to the shoreline. Other researchers refute this hypothesis (Szmant and Forrester 1996; Hughes and Szmant 1999). The potential for nutrient transport by upwelling is supported by a study conducted on the reef (Leichter et al. 1996) in which nitrate and nitrite concentrations associated with upwelling events were an order of magnitude higher than those of reef waters during non-upwelling. This nutrient enrichment occurred during the summer months, which agrees with the seasonality of coral diseases on these reefs. Transport of surface waters from relatively nutrient-rich Florida Bay to the reef tract through tidal channels has been documented by Brand (2002), Lapointe et al. (2002), and Smith and Pitts (2002). This process resulted in nitrogen loading of corals on reefs of the middle Keys (Cook et al. 2002). The latter was demonstrated by measurement of N:P ratios (in zooxanthellae isolated from freshly collected corals) that were twice the Redfield ratio (Cook et al. 2002).

With the above considerations in mind, which together suggest that nutrient enrichment may very well be occurring on reefs of the Florida Keys, it must be pointed out that black band diseased corals have been observed at sites that are far from human influence (Edmunds 1991). This observation thus negates a firm requirement of elevated nutrient input for disease incidence. An alternative source of our observed elevated nitrite could be black band activity itself. As black band disease moves across the coral colony it causes the lysis of the underlying coral tissue. During this process protein is broken down, resulting in the release of ammonium and numerous nitrogenous compounds (i.e. amino acids) into the environment (Briggs-Derek and Kear 1993). The newly available ammonium would be rapidly utilized by ammonia-oxidizing bacteria, resulting in an increase in the byproduct nitrite (Prosser 1989). Although nitrite-oxidizing bacteria would presumably utilize this substrate as an energy source, this physiological process occurs at a much slower rate; thus the result could be relatively elevated nitrite levels (Gottschalk 1986). Since, in our study, all water samples analyzed for nutrients were collected from within 5 cm of the coral surface, both in the diseased and non-diseased sites, and since no other nutrients showed elevated concentrations, it is quite possible that the elevated nitrite levels observed are a result of microbial mineralization associated with the coral tissue breakdown process. No significant differences were found in the concentrations of nitrate or ammonium (see Table 2), which is supportive of the above argument in that they are readily utilizable by a wide variety of organisms. If high nutrients are, in fact, required to promote (or initiate) disease activity, additional nutrient sources include fish feces and aerial deposition of nutrient-rich African dust (Shinn et al. 2000), both independent of direct (sewage) human input or terrestrial runoff.

The finding that black band disease is present in areas with statistically significant lower coral diversity in comparison with non-black band disease sites agreed with reported results from the only other field study of black band disease that included measurement of this factor (Bruckner and Bruckner 1997; Bruckner et al. 1997). We found no statistically significant difference between areas with and without black band disease and percent coral cover, which was similar to the results of studies by Edmunds (1991) and Bruckner et al. (1997), and is notable since this result suggests that black band disease was not transmitted by close proximity to infected colonies. Turbidity was also not statistically significant between black band and non-black band sites. This result differs from that of Kim and Harvell (2002), who reported increased turbidity associated with aspergillosis. No other study of coral disease included measurement of salinity (which we found to be not significant).

The results presented here did not reveal any clear relationship between degraded water quality and incidence of black band disease. The only (potentially) human-influenced environmental factors that could be considered as critical to disease development were elevated water temperature and available light. Laboratory studies of *P. corallyticum* revealed that maximum photosynthetic rates were attained above 28 °C (Taylor 1983; Richardson and Kuta 2002), and that this species prefers low light. It is our belief that the relationship between the physiological ecology of microbial pathogens of corals and the environment, for both black band and other diseases, may be the defining factor in coral disease occurrence. In the case of the black band disease cyanobacterium, the temperature and light optima for growth are in close agreement with field data documenting disease incidence. Microbial pathogens associated with bacterial bleaching (Banin et al. 2002), aspergillosis (Alker et al. 2001), and plague type II

(Remily and Richardson, unpublished data) all exhibit growth temperature optima at temperatures at or above 30 °C, a temperature at which corals become physiologically stressed (Fitt et al. 2001) and thus potentially more susceptible to infection.

In summary, our study of environmental factors associated with black band disease on reefs of the upper and middle Florida Keys is in agreement with several findings of similar studies by other groups. These include statistically significant disease occurrence in conjunction with relatively high water temperature, which has been found for black band disease, aspergillosis, dark spots disease, and plague; elevated dissolved inorganic nitrogen, also found for aspergillosis; and relatively shallow water depths, reported by many investigators for black band disease. However, our results are best interpreted within the context of the physiological ecology of the microbial coral pathogens.

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