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NOTE

Bleaching increases likelihood of disease on *Acropora palmata* (Lamarck) in Hawksnest Bay, St John, US Virgin Islands

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Abstract Anomalously high water temperatures may enhance the likelihood of coral disease outbreaks by increasing the abundance or virulence of pathogens, or by increasing host susceptibility. This study tested the compromised-host hypothesis, and documented the relationship between disease and temperature, through monthly monitoring of *Acropora palmata* colonies from May 2004 to December 2006, in Hawksnest Bay, St John, US Virgin Islands (USVI). Disease prevalence and the rate of change in prevalence showed a positive linear relationship with water temperature and rate of change in water temperature, respectively, but only in 2005 during prolonged periods of elevated temperature. Both bleached and unbleached colonies showed a positive relationship between disease prevalence and temperature in 2005, but the average area of disease-associated mortality increased only for bleached corals, indicating host susceptibility, rather than temperature per se, influenced disease severity on *A. palmata*.

Keywords *Acropora palmata* · Coral disease · Bleaching · Temperature · Host susceptibility

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Introduction

For the past 200,000 years, *Acropora palmata* and *Acropora cervicornis* have been two of the major shallow-water, reef-building corals in the Caribbean (Pandolfi 2002). Over the last 30 years, however, both species have dramatically declined throughout their habitat range (Bruckner 2003). Paleontological evidence suggests this decline is unprecedented, at least in the last 3,000–4,000 years (Aronson and Precht 2001). In the late 1970s, white band disease, a widespread and virulent epizootic, caused precipitous declines in *A. palmata* and *A. cervicornis* throughout the Caribbean and contributed to structural changes in the shallow reef environment (Aronson and Precht 2001). The major decline of *A. palmata* throughout the Caribbean resulted in the listing of this species in 2006 as "threatened" under the United States Endangered Species Act of 1973 (Hogarth 2006).

Considered to be exclusive to *A. palmata*, white pox disease was first documented on Eastern Dry Rocks Reef off Key West, Florida in 1996 (Holden 1996). Colonies affected by white pox disease display irregularly shaped white patches where the underlying coral skeleton is exposed, typically surrounded by apparently healthy-looking tissue (Patterson et al. 2002). Since the first report of white pox disease in 1996, this disease has affected *A. palmata* throughout the Caribbean (Sutherland and Ritchie 2004). Indeed, white pox disease is reportedly responsible for an 85% decline in *A. palmata* from seven reefs in the Florida Keys between 1996 and 1999 (Patterson et al. 2002).

It has been proposed that most coral diseases, including white pox, are more prevalent during times of high water temperature (Sutherland et al. 2004), however, this relationship may be an artifact of selective monitoring during

summer months. A relationship between warm temperature anomalies and white syndrome has been documented from annual surveys along the Great Barrier Reef (Bruno et al. 2007), but was highly dependent on coral cover. More frequent surveys (i.e., monthly rather than annual) would detail the relationship between water temperature and disease. Elevated temperatures are likely associated with an increased susceptibility of corals to infectious diseases (Harvell et al. 2002), but may also directly cause the proliferation of disease pathogens (Toren et al. 1998). Some authors suggest that the recent increase in coral diseases is not from increasing pathogenic virulence, but from secondary effects of compromised hosts because of anomalously high water temperatures (Lesser et al. 2007).

The objective of this study was to determine the stressors responsible for tissue loss on the *A. palmata* population at Hawksnest Bay, St John, US Virgin Islands (USVI) and document the incidence of disease. The 2005 Caribbean-wide bleaching event allowed for comparisons of disease prevalence, and extent of disease-induced mortality, between bleached (compromised) and unbleached (less- or un-compromised) *A. palmata* colonies. It was hypothesized that both disease prevalence and the associated areas of mortality would increase with increasing water temperatures, and would be more severe on bleached compared with unbleached colonies.

Materials and methods

Study site

Virgin Islands National Park (VINP) is a protected area located at 18°N and 64°W on the island of St John, US Virgin Islands (USVI). Hawksnest Bay is located within VINP on the northwest end of the island. Three patch reefs approximately 4,240, 4,800, and 1,760 m^2 in area, respectively, extend from shore in finger-like formations with depths ranging from 0 to 5 m.

Field sampling

Coral colonies were chosen using a random number-generating algorithm in ArcView Global Information Systems (GIS) software version 3.3. Hawknest Bay reef areas were outlined using ArcView's polygon toolbox on a geo-referenced aerial map created by the National Oceanic and Atmospheric Administration Biogeography Team. Random points (100) were generated within the reef area, using Arcview's Random Points Extension toolbox, with each point allocated to a unique coordinate. Points were located in the field using a Global Positioning Systems (GPS) unit in a waterproof AquaPac. When an *A. palmata* colony was located within 1 m of the GPS coordinate, the colony was photographed with a Sony Cyber-shot T1 digital camera in an underwater housing. If no colony was present at the GPS point, the procedure was repeated for the next waypoint. A total of 60 colonies were marked after 100 points were located.

Each colony was relocated monthly, from May 2004 to December 2006, to record the presence of recent mortality, considered as exposed skeleton uncolonized by settling benthic organisms. The apparent cause of recent mortality including: disease, bleaching, physical damage, sedimentation, or predation was also documented. Recently dead areas were photographed; a metric ruler was placed adjacent to each lesion for scale. The size of each lesion was measured using ImageJ Pro software. Each area of recent mortality was traced and measured three times per photograph, and averaged to account for human error during the tracing process. Colonies with visible loss of pigment were recorded as "bleached". In situ water temperatures were recorded every 2 h by a HOBO Water Temp Pro logger (Onset Computer Corporation) attached at 1-m depth.

Prior to statistical analyses, the raw data were tested for violations of normality and homogeneity of variance assumptions; normality was assessed using the normal probability plot procedure and the Shapiro–Wilks test; homogeneity of variances were tested using Levene's test with Statistica® software. Disease prevalence in 2006 and the rate of change in disease prevalence for 2005 and 2006 did not normalize even after transformation. All other data were normal, or had a normal distribution after iterative estimates of optimal power transformations, $y = x^{\lambda}$, where x is the response variable and λ the transformation parameter (Box and Cox 1964). Simple linear regression or nonparametric correlation analyses (depending on the data set) were used to determine the relationship between in situ water temperature and (1) disease prevalence; and (2) average area of recent mortality for colonies that did or did not bleach during the 2005 Caribbean-wide coral-bleaching event. To determine the relationship between disease prevalence and in situ water temperature at Hawksnest Bay, the first derivatives, or the rates of change, of both disease prevalence and water temperature were calculated using Matlab 6.5® and examined using simple linear or nonparametric regression.

Results and discussion

Influence of disease

Disease was the most frequent apparent cause of partial mortality affecting 87% of the monitored *A. palmata*

colonies at Hawksnest Bay. On thermally stressed (i.e., bleached) colonies, disease was associated with more total colony mortality than any other stressor. Disease represented the highest apparent cause of tissue loss, being responsible for 52% of the 344 recent mortality observations. Predation by corallivorous snails, fireworms, or fishes was responsible for 17% mortality, broken branches for 18%, and sediment smothering or abrasion for 13%. Of the 180 disease incidences 80% resembled white pox, only 3% resembled white band, and 17% were undescribed white diseases. For statistical analyses, disease was not separated into the subjectively described disease "types".

2005 Caribbean bleaching event

During the summer of 2005 anomalously warm water temperatures and doldrum-like conditions coincided with Caribbean wide coral-bleaching. From April to November 2005, monthly mean water temperatures at Hawksnest Bay ranged from 0.6 to 1.6°C higher than the 14-year historical monthly means for St John (Fig. 1) (data provided by the National Park Service). At Hawksnest Bay, some *A. palmata* colonies had pale branch tips in July 2005. Bleaching peaked in October to early November, with some colonies losing all pigmentation. Prior to 2005, bleaching of *A. palmata* had not been recorded within the Virgin Islands (C. Rogers, personal observations). During the 2005 bleaching event, 50% of the *A. palmata* colonies being monitored showed some loss of pigmentation, and 17% of the colonies died between October and December 2005 from either bleaching alone or a combination of bleaching and disease. Mortality from bleaching appeared as the degeneration of tissue with no visible pigmentation, simultaneously followed by the colonization of filamentous (or turf) algae on the coral skeleton. Mortality from disease appeared as lesions with distinct borders separating living tissue (with or without pigment) from completely exposed, bare skeleton. Excluding the 2005 bleaching event, only five colonies died, all from physical damage. No colonies showed any signs of bleaching during the summers of 2004 or 2006, and no colonies suffered complete mortality from disease alone throughout the entire study.

Table 1 Summary table of simple linear regression (s) and non-parametric correlation (np) analyses comparing disease prevalence and average water temperature for 2004, 2005 and 2006

Year	R^2(s) or r_s(np)	*df*	*F*	*P*
2004 (s)	0.042	5	0.219	0.660
2005 (s)	0.604	10	15.278	0.003*
2006 (np)	0.378	11		0.203

* Indicate significant values

Disease and temperature

Disease prevalence ranged from 0 to 32.6%, with non-outbreak prevalence averaging 4.9%. All three years showed high disease prevalence (2004: 22.0%; 2005: 32.6%, 2006: 28.9%), but a positive relationship with temperature existed only in 2005 ($R^2 = 0.604$, $P = 0.003$) (Table 1; Fig. 2). While a relationship between water temperature and disease has been suggested for *A. palmata* (Patterson et al. 2002), the present study is the first to quantify this. In addition, the rate of change in disease

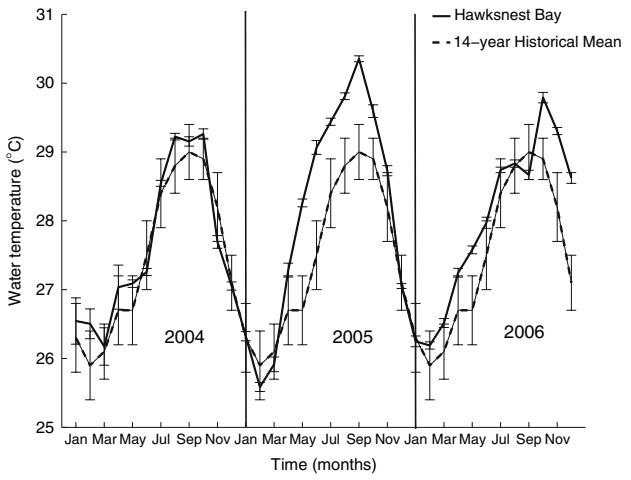

Fig. 1 Monthly mean water temperatures for Hawksnest Bay from 2004 to 2006 and the 14-year historical monthly mean water temperatures for St John. *Error bars* indicate ±standard deviation

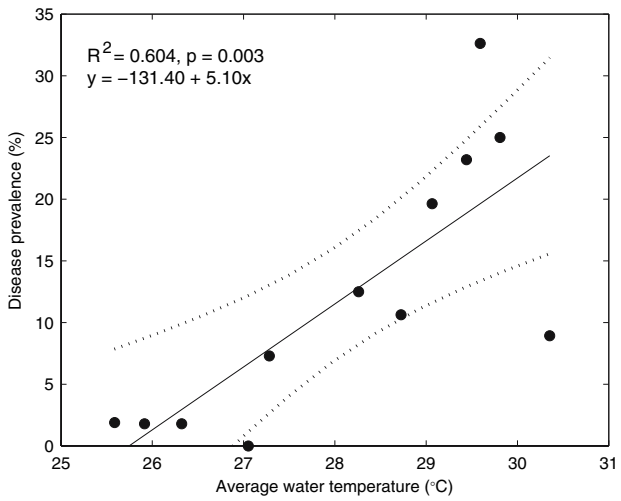

Fig. 2 Relationship between disease prevalence and average water temperature at Hawksnest Bay, St John in 2005. *Dotted lines* indicate 95% confidence limits

prevalence showed a positive correlation with rate of change in mean monthly water temperature only in 2005 (r_s = 0.692, P = 0.01) (Table 2; Fig. 3). Water temperature ranges were similar among years (2004: 25.4–30.6°C; 2005: 24.8–31.2°C; 2006: 25.4–31.2°C), but the relationship between temperature and disease prevalence was only present in 2005 suggesting that prolonged elevated temperatures increased the likelihood of disease.

Individual colonies of the same coral species often respond differently to temperature anomalies depending upon symbiotic algal genotypes (Rowan et al. 1997) and colony life history (Brown et al. 2002). In addition, Ritchie (2006) showed a decrease in the antibiotic activity of coral mucus in thermally stressed colonies of *A. palmata*, suggesting bleached colonies may be more susceptible to disease. In this study, the only year to exhibit a positive relationship between temperature and disease coincided with a coral-bleaching event indicating the responses of bleaching and disease to temperature were not independent of each other. These results contrast with a report from the Great Barrier Reef, where bleaching events showed a negative spatial correlation with coral disease outbreaks (Bruno et al. 2007).

Disease and host susceptibility

Disease prevalence of both bleached and unbleached colonies increased significantly with increasing water temperatures (Table 3) in 2005. Bleaching followed by disease was also observed on massive reef-building coral colonies on deeper reefs of St John and St Croix (Miller et al. 2006). Similarly, in 2001/2002 a coral bleaching event at Magnetic Island, Australia was followed by a disease outbreak that affected both bleached and unbleached colonies (Jones et al. 2004). In this study, bleached colonies, however, displayed a stronger positive relationship between water temperature and disease prevalence than unbleached colonies (bleached: $R^2 = 0.576$, unbleached: $R^2 = 0.408$), and judging by the regression slopes, prevalence increased at a faster rate for bleached colonies (bleached: $y = -164.77 + 6.33 \times x$, unbleached $y = -83.55 + 3.32 \times x$).

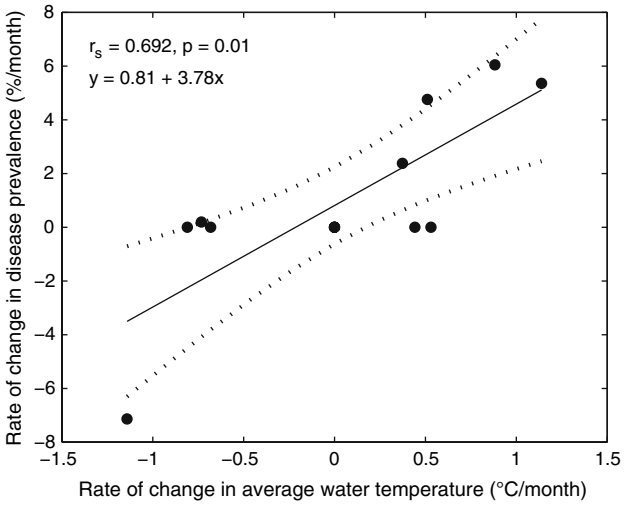

Fig. 3 Correlation between the rate of change in disease prevalence and the rate of change in average water temperature at Hawksnest Bay, St John in 2005. *Dotted lines* indicate 95% confidence limits

Although both unbleached and bleached colonies showed a significant positive relationship between disease prevalence and water temperature, prevalence measurements do not account for the extent of mortality caused by disease per incident. The measure of prevalence describes the influence of disease within specified populations, but disease can affect only part of a colony rather than the entire individual. This study showed a positive relationship between the average area of mortality and water temperature, but only for bleached colonies in 2005 ($R^2 = 0.748$, P = 0.01) (Table 4; Fig. 4). Therefore, although disease prevalence increased for both bleached and unbleached colonies, the amount of associated mortality from disease increased only when the host was bleached.

It has been suggested that increasing disease prevalence could result from a different, more virulent, pathogen present during times of elevated sea temperature. In the present case, an increase in disease prevalence was associated with high water temperatures, but colonies lost significantly more tissue from disease when bleached. There was no increase in the average area of disease-induced mortality on colonies that did not bleach, suggesting that although temperature

Table 2 Summary table of simple linear regression (s) and non-parametric correlation (np) analyses comparing the rate of change in disease prevalence and the rate of change in average water temperature for 2004, 2005 and 2006

Year	R^2(s) or r_s(np)	*df*	*F*	*P*
2004 (s)	0.013	5	0.065	0.809
2005 (np)	0.692	10		0.01*
2006 (np)	0.107	11		0.727

* Indicate significant values

Table 3 Summary table of simple linear regression analyses comparing disease prevalence and average water temperature on unbleached and bleached colonies in 2005

Effect	R^2	*df*	*F*	*P*
Unbleached	0.408	10	6.904	0.025*
Bleached	0.576	10	13.575	0.008*

* Indicate significant values

Table 4 Summary table of simple linear regression analyses comparing the area of disease-induced mortality and average water temperature on unbleached and bleached colonies in 2005

Effect	R^2	*df*	*F*	*P*
Unbleached	0.221	10	2.841	0.123
Bleached	0.748	10	29.727	0.001*

* Indicate significant values

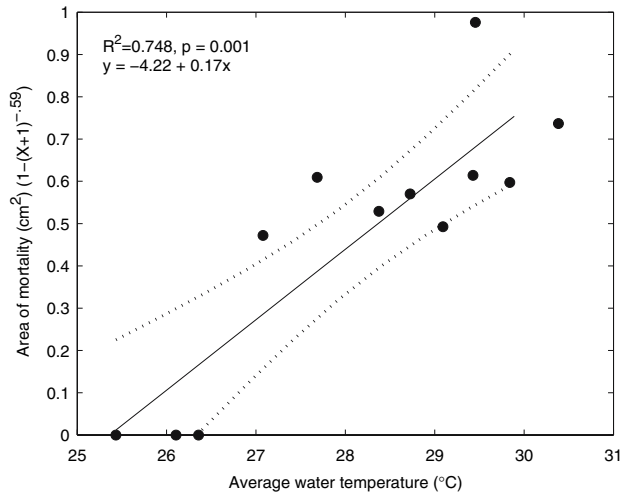

Fig. 4 Relationship between average area of disease-associated mortality and average water temperature for bleached colonies at Hawksnest Bay, St John in 2005. *Dotted lines* indicate 95% confidence limits

alone could influence disease prevalence, disease severity was also a result of host susceptibility that was caused by bleaching. Furthermore, although disease prevalence is an indicator of population status this study shows that the area of mortality caused by disease may be even more useful for determining the effects of disease severity on modular organisms, such as scleractinian corals. The significance of disease severity on a compromised host, along with positive relationships between water temperature and disease prevalence, including rates of change, give some insights into predicted responses of *A. palmata*, the already threatened coral species, to future global climate change.

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