Relationships between the history of thermal stress and the relative risk of diseases of Caribbean corals

C. J. RANDALL,1,3 A. G. JORDAN-GARZA,1 E. M. MULLER,2 AND R. VAN WOESIK1

1Department of Biological Sciences, Florida Institute of Technology, 150 West University Boulevard, Melbourne, Florida 32901 USA
2Mote Marine Laboratory, 1600 Ken Thompson Parkway, Sarasota, Florida 34236 USA

Abstract. The putative increase in coral diseases in the Caribbean has led to extensive declines in coral populations. Coral diseases are a consequence of the complex interactions among the coral hosts, the pathogens, and the environment. Yet, the relative influence that each of these components has on the prevalence of coral diseases is unclear. Also unknown is the extent to which historical thermal-stress events have influenced the prevalence of contemporary coral diseases and the potential adjustment of coral populations to thermal stress. We used a Bayesian approach to test the hypothesis that in 2012 the relative risk of four signs of coral disease (white signs, dark spots, black bands, and yellow signs) differed at reef locations with different thermal histories. We undertook an extensive spatial study of coral diseases at four locations in the Caribbean region (103 km), two with and two without a history of frequent thermal anomalies (~4–6 years) over the last 143 years (1870–2012). Locations that historically experienced frequent thermal anomalies had a significantly higher risk of corals displaying white signs, and had a lower risk of corals displaying dark spots, than locations that did not historically experience frequent thermal anomalies. By contrast, there was no relationship between the history of thermal stress and the relative risk of corals displaying black bands and yellow signs, at least at the spatial scale of our observations.

Key words: Caribbean; climate change; coral reefs; disease; ocean temperature; relative risk; shallow-water reef; temperature stress; thermal stress.

INTRODUCTION

Over the last several decades, coral diseases have caused significant declines in coral cover throughout the Caribbean, and have changed the community structure of many shallow-water reefs (Aronson and Precht 2001, Bruno et al. 2011). There are now at least 14 described stony-coral diseases in the Caribbean, many of which have become widespread (Sutherland et al. 2004, Weil and Rogers 2011). While some researchers suggest that the observed increase in coral diseases is the result of an increase in human-introduced pathogens (Kaczmarsky et al. 2005, Sutherland et al. 2010, Muller 2011), other researchers argue that these outbreaks are the result of compromised corals, which have been subjected to increased environmental stressors (Lesser et al. 2007, Muller et al. 2008, Muller and van Woesik 2012), the most severe of which is the recent and continuing increase in ocean temperatures (Hansen et al. 2006, Hoegh-Guldberg et al. 2007). Our study examined whether the relative risk of corals showing signs of disease was related to the historical frequency of temperature anomalies at four reef locations in the Caribbean.

The effect of water temperature on coral disease etiology is complex and affects both the incidence of disease and the rate of disease-induced mortality. Some studies suggest that ocean temperature is likely the primary driver of coral diseases (Harvell et al. 2002, Bruno et al. 2007, Muller et al. 2008, Muller and van Woesik 2012). For example, field experiments have shown that black-band disease progressed twice as rapidly in the summer than in the winter (Boyett et al. 2007). The prevalence of black-band disease in the Florida Keys increased when water temperatures exceeded 28°C (Kuta and Richardson 2002), and the incidence of black-band disease in the United States Virgin Islands (USVI) increased with the rate of increase in water temperature (Muller and van Woesik 2011). Similarly, under laboratory conditions Cervino et al. (2004) showed that the rate of progression of Caribbean yellow-band disease increased with increasing temperature. Indeed, elevated temperature may influence diseases in many ways, including: (1) increasing the growth rate and virulence of pathogens (Toren et al. 1998, Harvell et al. 2002), (2) compromising coral immunity, thereby increasing coral susceptibility to disease (Toren et al. 1998, Alker et al. 2001, Lesser et al. 2007, Muller et al. 2008, Mydlarz et al. 2010, Reed et al. 2010), and (3) affecting potential vectors that transmit diseases (Harvell et al. 2002).

Several studies have shown that disease outbreaks often coincide with or closely follow thermal-stress

However, some studies have found no relationship between episodes of thermal stress and disease, including other studies on atramentous necrosis and white syndrome on the Great Barrier Reef (Anthony et al. 2008, Ban et al. 2012). The lack of any relationship between thermal stress and disease is not, however, necessarily evidence of coral resistance. For example, no relationship between thermal stress and disease, in some studies, may be an artifact of sampling frequency (Muller et al. 2008). Indeed, continuous monitoring through thermal-stress events is rare. It is therefore conceivable that mismatches may occur between annual coral monitoring programs and the rapid rate at which some diseases spread (Dalton et al. 2010, Roff et al. 2011). Unless sampling is frequent, monitoring programs may miss the effect of thermal stress on the subsequent change in the prevalence of coral diseases. Alternatively, there may be temperature thresholds, below which diseases are rare and above which diseases increase (Lesser et al. 2007). These examples highlight inconsistencies and a need for more comprehensive monitoring of both coral diseases and abiotic factors that potentially affect the expression of diseases.

Abiotic conditions influence host–pathogen interactions and have the potential to drive local adaptation to disease (Mitchell et al. 2005, Dionne et al. 2007). For example, Dionne et al. (2007) found that in rivers with different thermal regimes, pathogens maintained differential selective pressure on the immune system of salmon. Clearly, abiotic conditions and disturbance regimes can drive adaptation (Lytle 2001), yet it is uncertain to what extent persistent thermal-anomaly events drive the adaptation of corals and their associated diseases in a changing climate. Given repeated exposure to elevated temperature, some studies show evidence of coral acclimatization and, potentially, adaptation to thermal stress (Brown et al. 2002, Maynard et al. 2008, Thompson and van Woesik 2009, Kenkel et al. 2013). Similarly, coral populations may develop the ability to resist disease infection (Reshef et al. 2006, Grassly and Fraser 2008, Reed et al. 2010, Bruno et al. 2011). Several mechanisms have been proposed that would allow coral populations to increase their resistance to disease, including: (1) The innate immune system of corals may exhibit acquired-like immune responses that could reduce the burden of disease in harsh environments (Reshef et al. 2006, Reed et al. 2010); (2) selection by differential mortality of susceptible genotypes (see Plate 1), followed by sexual replenishment, may foster the development of disease-resistant genotypes under persistent selective pressure (Bruno et al. 2011, van Woesik and Jordán-Garza 2011); and (3) selection by differential survival of individual polyps that develop resistance through somatic mutations (van Oppen et al. 2011).

Sea surface temperatures will most likely continue to increase this century (Hansen et al. 2006, 2010, Webster et al. 2005). Localities that have historically experienced frequent thermal anomalies are also the localities that are most likely to again experience frequent thermal stress (Thompson and van Woesik 2009). It is therefore imperative to examine the extent to which thermal histories influence coral diseases. Given that most species of corals take a minimum of four to five years to reach sexual maturity (Soong 1993), experimentally subjecting corals to different thermal-stress regimes over several generations is difficult. We instead sampled coral diseases in locations that differed historically in their thermal histories. We hypothesized that the relative risk of four signs of coral disease (white signs, dark spots, black bands, and yellow signs), would differ at locations (at a scale of $10^3$ km) that have experienced frequent thermal anomalies (~4–6 years), over the last 143 years, compared with locations that have not experienced frequent thermal anomalies, over that same time period. If coral populations that experienced frequent thermal anomalies were comprised of primarily immuno-compromised corals, then we would predict a high risk of disease at these locations. By contrast, if coral populations that experienced frequent thermal anomalies had purged the susceptible individuals and were comprised of primarily disease-resistant corals, then we would predict a low risk of disease at these repeatedly stressed locations.

**METHODS**

**Location selection**

Globally, thermal-stress events vary considerably over space and time (Thompson and van Woesik 2009, Burrows et al. 2011). Thompson and van Woesik (2009) took a broad-brush approach to examine thermal anomalies. It was apparent that some localities in the Caribbean, for example, had historically experienced frequent thermal anomalies (~4–6 years), whereas other localities in the Caribbean had not. Based on the regions that were identified as either experiencing frequent or infrequent thermal anomalies in Thompson and van Woesik (2009), we selected four reef locations in the Caribbean region (Fig. 1) and examined their thermal
histories using data from the MetOffice HadISST records from 1870–2012 (Rayner et al. 2003). Two locations that did not experience frequent thermal anomalies over the last 143 years were selected: (1) Mahahual, Mexico, and (2) Tuxpan, Mexico, henceforth termed “reference” locations. We also selected two locations with a history of frequent thermal anomalies (4–6 years) over the last 143 years: (3) Bocas del Toro, Panama, and (4) St. John, United States Virgin Islands (USVI), henceforth termed “frequent-anomaly” locations. To minimize the potential effect of spatial covariates, we selected four localities that were distant from each other (a minimum of 1000 kilometers), yet still shared a similar history of thermal-anomaly frequency. These localities were also logistically feasible to survey within a two-month window at the peak of...
annual water temperatures. Furthermore, the two
two reference locations are separated by the Yucatan
Peninsula, and are in the Caribbean Sea and the Gulf
of Mexico, respectively (Fig. 1).

Comparison of thermal histories among study sites

To compare thermal-anomaly histories among loca-
tions, we conducted wavelet analyses on de-trended
records of anomalies of the mean monthly sea surface
temperature (SST), calculated as a deviation above or
below each location’s 143-year monthly average. Wave-
let-transform analyses were performed to assess the
frequency (i.e., periodicity) of anomalous thermal events
and to determine the timing of those events (i.e., when a
particular frequency of anomalies occurred), throughout
the record for each location. The power spectra of the
wavelet analyses indicated the strength of the signals, in
time–frequency space. Cross-wavelet analyses were used
in a pairwise manner to identify significant common
periodicities (i.e., common power) of temperature
anomalies, in time–frequency space, between locations.
Coherent-wavelet analyses identified the consistency of
the cross-wavelet transforms, in time–frequency space,
which were expressed as significant localized correlation
coefficients between each pair of locations. We note that
significant periodicities in the thermal anomalies do not
indicate directionality of the anomalies (i.e., they can be
either higher or lower than the mean). Wavelet-
transform analyses, cross-wavelet comparisons, and
wavelet-coherence comparisons of the time series data
were examined using the biwavelet package version
0.17.1 (Torrence and Compo 1998, Gouhier 2013) in R
(R Development Core Team 2013).

Field sampling

To assess the prevalence of coral diseases at each
location, a survey area (~1–10 km² depending on the
region’s geographic features) of hard-bottom habitat
was visually defined using Google Earth (available
online). The survey area was divided into 100 × 100 m
cells (using Ge-Path 1.4.4; available online). Within each
location, 25 100 × 100 m cells were randomly selected as
sites. These sites were defined as the primary sampling
units (Cochran 1977, Smith et al. 2011). A single 10 × 10
m quadrat was randomly placed within each site, for
field data collection. To maintain consistency across
locations and to minimize potential effects of coral-
assemblage differences, three criteria had to be met for a
site to be surveyed: (1) the depth averaged between 5 and
10 m, (2) the substrate was hard bottom, and (3) corals
were present. If any one of these criteria was not met at a
given site, it was rejected and the next randomly
generated site was selected. In total, 25, 10 × 10 m
quadrats were sampled at each location, for a total of 50
quadrats across two reference locations, for a total
survey area of 10 000 m².

All four locations were surveyed between 2 July and
1 September 2012. At each site, divers surveyed each
100-m² quadrat by systematically laying 10 contiguous
1 × 10 m belt transects onto the reef substrate. Each
coral colony with a disease sign was identified in situ
and the species and disease signs were recorded. Four
disease signs (Fig. 2) were identified: (1) White sign was
defined as a bright, white band or patch of recent
mortality adjacent to healthy-appearing tissue (i.e., the
tissue bordered a well-defined edge of exposed skeleton
not yet colonized by algae or other biofouling
organisms; sensu Bythell et al. 2004); (2) dark spot
was defined as tissue with purple, brown, or black
lesions, forming spots of irregular shapes (sensu
Goreau et al. 1998); (3) black band was defined as a
black band over the coral tissue exposing white
skeleton with different stages of biofouling (sensu
Richardson 2004); and (4) yellow sign was defined as a
yellow discoloration of tissue forming a band or
blotches (sensu Santavy et al. 1999). White signs and
black bands were associated with recent tissue loss;
yellow signs and dark spots were usually, but not
always, associated with recent tissue loss. Notably, very
few yellow bands were observed that followed the
classical description (Reeves 1994). Instead, most coral
colonies presented a patchy, nonuniform yellowing of
the tissue; therefore, the condition was termed “yellow
sign.” Additionally, any area of recently exposed white
skeleton, which was not clearly caused by predation or
a competitive interaction, was recorded as a white sign,
including white plagues, white bands, and white pox.
The white-sign diseases were not differentiated because
of similar- or identical-appearing signs, unknown
etiologies for several diseases, and the possibility that
the diseases were caused by the same pathogens
(Bythell et al. 2004, Ainsworth et al. 2007). Coral
colonies were occasionally recorded with two or more
signs of disease, when those signs appeared to be
spatially independent on the colonies.

Relative-risk assessments

A relative-risk assessment was conducted to test the
hypothesis that coral populations in localities with a
history of frequent thermal anomalies differed in their
risk of developing a disease compared with coral
populations in reference locations. We examined relative
risk using the following equation:

\[
\text{relative risk} = \frac{a/(a + b)}{c/(c + d)}
\]

where \( a \) is the number of sites with a specific disease at a
frequent-anomaly location, \( b \) is the number of sites
without a specific disease at a frequent-anomaly
location, \( c \) is the number of sites with a specific disease
at a reference location, and \( d \) is the number of sites
without a specific disease at a reference location (Sistrom

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4 http://earth.google.com/
5 http://www.sgrillo.net/googleearth/gepath.htm
and Garvan 2004). Estimates of relative risk were first calculated by combining all coral taxa, referred to here as the “assemblage-level analyses.” Diseases were recorded as either present or absent at a site, while only including sites that supported the coral species known to host the disease of interest. However, white signs on Acropora spp. were considered separately, as the causative agents of white signs on Acropora spp. (white pox and white bands) appear to be unique to the genus (Ritchie and Smith 1998, Patterson et al. 2002, Sutherland et al. 2010). Relative-risk estimates were also calculated using the presence and absence of each disease on individual coral taxa (considering only sites that supported the specific coral genus or species of interest), referred to here as the “taxa-specific analyses.” Ten coral taxa that are important reef-builders or that were common at all four locations were selected for the taxa-specific analyses. A series of analyses was undertaken by combining data from locations with similar thermal histories. Another series of analyses used data
from each location in pairwise comparisons. Sites were not included in the estimates of relative risk when the coral taxa of interest were absent.

Relative risk was calculated using a Bayesian approach (Gelman et al. 2004, Lawson 2009) and was estimated using a binomial likelihood distribution and a uniform-Beta prior distribution. To obtain an estimate of relative risk, Markov chain Monte Carlo simulations (100,000 iterations with a burn-in of 10,000) were used with Gibbs sampling in OpenBUGS 3 (available online; see the Supplement for the Bayesian analysis code). A 95\% credible interval was calculated for each estimate of relative risk. Credible intervals that did not include a value of one were considered significant, with a credible interval above one signifying a higher risk of a coral colony inflicted with disease at the frequent-anomaly locations. A credible interval below one signified a higher risk of disease at the reference locations.

Furthermore, the range of the 95\% credible interval provided a general measure of the confidence in the relative-risk estimate, with a large range signifying low confidence in the estimate (Kruschke 2011). We added one to each observation total to prevent observations of zero yielding undefined values of relative risk. The OpenBUGS code is available in the Supplement.

### RESULTS

#### Comparison of thermal histories among study sites

Wavelet-transform analyses identified the cycles that corresponded to anomalous temperature frequencies of 4–6 years. Based on these frequencies, the anomalies were most likely related to El Niño Southern Oscillation (ENSO) events and were therefore probably high-temperature anomalies. These events were more consistently significant over the last 143 years in Bocas del Toro, Panama (Fig. 3c), and St. John, United States Virgin Islands (Fig. 3d), than in Mahahual (Fig. 3a) and Tuxpan, Mexico (Fig. 3b). Further wavelet comparisons are available in the supplemental material (Appendix C: Figs. C1 and C2).

#### Field sampling

The four signs of disease were observed in all four locations, affecting a total of 2469 colonies of 27 coral species. Overall, white signs affected the greatest number of coral species (28), whereas yellow signs affected the least number of species (3; the only genus affected was *Orbicella*; Table 1). *O. annularis* was the only species affected by all four disease signs (Table 1). Bocas del Toro, Panama (frequent-anomaly location), had the

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6 http://www.openbugs.net/w/Downloads
The greatest number of coral species that were diseased (20), whereas Tuxpan, Mexico (reference location), had the least number of coral species that were diseased (12; Table 1). Ninety-five percent of the diseased colonies were observed with a single disease sign, whereas 5% of the diseased colonies were observed with multiple signs of disease (3% with white and yellow signs, 2% with white signs and dark spots, <1% with dark spots and yellow signs, and a single O. annularis with white signs, dark spots, and yellow signs; note that these signs appeared to be spatially independent on the colonies).

Relative risk

Locations that historically experienced frequent thermal anomalies had a significantly different risk of corals displaying white signs and dark spots than locations that did not historically experience frequent thermal anomalies (Fig. 5). At the assemblage level, the estimate of the relative risk of white signs (with and without Acropora spp.) was significantly higher in locations with a history of frequent thermal stress than in locations without a history of frequent thermal stress (relative risk = 1.2, 95% credible interval = 1.1–1.4; Fig. 5; Appendix B: Table B1). While a history of frequent thermal stress increased the relative risk of white signs at the assemblage level, that pattern was less evident at the taxa level; only one genus, Porites, exhibited a significantly increased relative risk of white signs at frequent-anomaly locations (relative risk = 1.9, 95% credible interval = 1.2–3.1) and Colpophyllia natans showed the opposite pattern (i.e., higher risk at the reference locations, relative risk = 0.4, 95% credible interval = 0.2–0.6; Fig. 6; Appendix B: Table B2). All other coral species, including Acropora spp., had credible intervals that included one, indicating that the risk of disease did not differ significantly with thermal history. However, many of those credible intervals were large, suggesting high variance across sites and low confidence in the estimate of the likelihood of a relationship between risk and thermal history (Fig. 6; Appendix B: Table B2). By contrast, the relative risk of dark spots was significantly lower in locations with a history of frequent thermal stress than in locations without that history (relative risk = 0.8, 95% credible interval = 0.6–0.9; Fig. 5; Appendix B: Table B1). That pattern persisted within the taxa-specific analyses for the common hosts of dark-spot syndrome, Siderastrea spp., Orbicella annularis spp. complex, and Stephanocolenia intersepta (Fig. 7; Appendix B: Table B2).

At the assemblage level, there was no detectable relationship between the relative risk of either yellow signs or black bands and the frequency of thermal anomalies (Fig. 5; Appendix B: Table B1). Taxa-specific analyses of yellow signs were not conducted because the only genus that hosted yellow signs was Orbicella and was therefore equivalent to the assemblage level analysis. For the taxa-specific analyses of black bands, relative risk did not differ significantly with the frequency of thermal anomalies, and the credible intervals were wide, indicating low confidence in the estimate to detect a relationship (Fig. 8; Appendix B: Table B2).

Discussion

Our results indicate that locations in the Caribbean with a history of frequent thermal anomalies had a significantly higher risk of corals displaying white signs, and a significantly lower risk of corals displaying dark spots, than locations without frequent thermal stress. Estimates of the relative risk of white signs were also higher at locations with frequent thermal anomalies for ~80% of the coral taxa examined, but most of the estimates also had wide credible intervals, which included one (Fig. 6; Appendix B: Table B2). These results suggest that there was high variance in the prevalence of white signs on individual coral species across locations, although low confidence in some estimates may be simply a consequence of species rarity. For example, Acropora spp. were uncommon (observed in only 18 of 100 quadrats) due in part to the restricted depth range (5–10 m) of our sites (Appendix B: Table B2). Alternatively, common species may infrequently
show signs of a particular disease (for example, see white signs on *Montastraea cavernosa*; Appendix B: Table B2), and therefore, the variance among locations would be high. High variance among locations could also be caused by differences in location-specific coral assemblages (Appendix A). For one species, *Colpophyllia natans*, frequent-anomaly locations actually had a significantly reduced risk of white signs (Fig. 6). Indeed, that species was most abundant in Tuxpan, Mexico (a reference location), and rarer at the other three locations, suggesting that observations from Tuxpan were most likely driving that relative-risk result.

Despite high variance in the relative risk of white signs at the taxa-specific level, the risk of white signs was significantly higher for the coral assemblages at frequent-anomaly locations compared with reference locations. Although potentially different white signs were not distinguished in situ (with the exception of *Acropora* spp.), it is generally regarded that most white-sign diseases are infectious and caused by bacterial agents (Ritchie and Smith 1995, Richardson et al. 1998a, b, Patterson et al. 2002, Denner et al. 2003). Elevated temperatures increase the rates of growth and division of many marine bacteria (White et al. 1991), which could lead to an increase in the virulence of infectious, bacterial pathogens (Toren et al. 1998). A positive relationship between temperature and disease virulence has been demonstrated for some bacterial pathogens associated with coral diseases, including *Vibrio shiloi* and *V. coralliilyticus*, supporting the notion that high temperatures may increase the virulence of bacteria that putatively cause white-sign diseases (Kushmaro et al. 1998, Banin et al. 2001, Ben-Haim and Rosenberg 2002).

Interestingly, at the assemblage level, the relative risk of dark spots was higher at reference locations than at frequent-anomaly locations (Figs. 5 and 7). These results were driven primarily by the presence of dark spots on three coral genera, *Oorbicella*, *Siderastrea*, and *Stephanocoenia*. Dark spots is one of the most common and widespread syndromes in the Caribbean (Gil-Agudelo et al. 2004, Gochfeld et al. 2006), yet its etiology is poorly understood. It is unknown whether dark spots are indicative of infectious disease or are instead physiological responses to stress (Borger 2005).
Lesser et al. 2007, Muller and van Woesik 2012). Indeed, dark spots may be the result of: (1) a single infectious disease with slightly different signs on different hosts, (2) multiple infectious diseases with similar morphological signs (Weil 2004, Porter et al. 2011), (3) a physiological response to increased environmental and physical stressors experienced by the host (Borger 2005), or (4) a symptomatic precursor of another disease (Muller and van Woesik 2012).

Given that the etiology of dark spots is unknown, it is difficult to identify the mechanism by which an increase in thermal-anomaly frequency may reduce the relative risk of this sign. However, we suggest two possible explanations which clearly warrant further investigation. First, locations with a history of frequent thermal-stress events may support coral populations that had already acclimatized to thermal stress at the time of the 2012 survey, and were therefore less susceptible to dark spots. Dark spots do not always progress or result in tissue loss and when they do, the rate of progression is slow, at 0.12 mm/d (Borger 2005) compared with white plague, which can advance up to 20 mm/d (Richardson et al. 1998a).

Furthermore, Siderastrea siderea, a species commonly affected by dark spots, is considered to have a generalist life history strategy, producing abundant offspring (Lewis 1997, Miller et al. 2000, Darling et al. 2012). Over several decades, S. siderea may have experienced differential selective pressure in locations with persistent thermal anomalies, resulting in a population with a higher frequency of alleles that are resistant to dark spots or to the environmental stress that manifests as dark spots. Second, dark spots may be a sign of stress that could precede future disease outbreaks (Borger 2005, Lesser et al. 2007, Muller and van Woesik 2012). If dark spots are an indicator of stress and are usually followed by a disease, then the abundance of dark spots at reference locations may be indicative of the assemblages currently contending with stress. By contrast, assemblages at frequent-anomaly locations may have already succumbed to stress and show few signs of dark spots. A small percentage of colonies with dark spots (<3%) also had multifocal signs of other diseases (white and yellow signs). These cases could be illustrative of a transition from a stress response to an infectious disease and we suggest that these potential, intermediate-transition stages warrant further attention. Clearly, describing the etiology of these diseases is urgently needed to understand the potential mechanisms driving the patterns that were observed in the field.

A history of frequent thermal stress did not appear to be related to the risk of black bands or yellow signs, either at the assemblage level, or at the taxa level (Figs. 5 and 8; Appendix B: Tables B1 and B2). Black-band disease is caused by a microbial consortium (Garrett and Ducklow 1975, Ducklow and Mitchell 1979, Rutzler et al. 1983, Cooney et al. 2002, Frias-Lopez et al. 2002), and yellow signs, assumed to be indicative of Caribbean...
yellow-band disease, are associated with *Vibrio* spp. bacteria (Santavy et al. 1999, Cervino et al. 2004). Evidence suggests that elevated temperatures directly increase the progression rate of black-band disease (Kuta and Richardson 2002, Boyett et al. 2007) and Caribbean yellow-band disease (Cervino et al. 2004), but interestingly, the present study found no relationship between thermal-anomaly frequency and disease risk, for either of these diseases.

While black bands were observed on coral colonies at all four survey locations, they were uncommon, and responsible for <1% of the observed diseases. Although black bands were present in more quadrats in the high-frequency locations than in the reference locations (nine vs. four), there was high variance among locations. During an outbreak of black-band disease, up to 5% of the host assemblage may become infected (Bruckner et al. 1997, Muller and van Woesik 2011), which indicates that we did not sample during an outbreak. Furthermore, outbreaks of black-band disease are generally short lived (Sato et al. 2009). Therefore, finding a relationship between black-band disease and an environmental factor may require a much larger sample size than reported here, as the disease appears to persist at relatively low prevalence (Page and Willis 2006).

Unlike black bands, yellow signs were common at all locations (Appendix B: Table B1), although we found no relationship between thermal-anomaly history and the risk of yellow signs. It was surprising to find no evidence of a relationship between thermal history and yellow signs, especially since Caribbean yellow-band disease is sensitive to temperature (Cervino et al. 2008). Yet, Caribbean yellow-band is usually a disease that progresses slowly (Cervino et al. 2004, Bruckner and Bruckner 2006) and disease signs might remain on large (>1 m diameter) *Orbicella* spp. colonies for many years (Bruckner and Bruckner 2006; A. G. Jordan-Garza, personal observation). The turnover of *Orbicella* spp. populations also occurs slowly. Therefore, an effect of thermal history might be confounded by the presence of large colonies that show yellow signs for long periods of time. It is also possible that other environmental factors may be driving the prevalence and abundance of black bands and yellow signs. For example, elevated nutrients are positively correlated with the progression rates of both black-band disease (Voss et al. 2007) and Caribbean yellow-band disease (Bruno et al. 2003), suggesting that multiple environmental factors may interact with temperature to cause these diseases.

Indeed, variability in environmental conditions, including temperature and temperature history, may affect the dynamics of coral diseases, resulting in differential selective pressure. With an increase in the frequency and
extent of coral disease epizootics over the last several decades, it is likely that disease-driven selective pressure will affect coral populations. Our results also indicate that the relative risk of some coral diseases is related to the history of thermal stress. For example, the higher relative risk of white signs at locations with high return frequencies of anomalous temperatures, suggests that susceptible colonies have not been purged from those populations. Persistent, selective pressure has the potential to result in disease resistance, if disease-resistant genes become fixed in populations (Hildemann et al. 1977, Potts 1984, Mydlarz et al. 2010, Reed et al. 2010, van Woesik and Jordán-Garza 2011). How rapidly genes become fixed in a population, and hence, the response time, will also depend on variations in the life history traits of the organisms found in the different thermal environments. In summary, the prevalence of some coral diseases was clearly related to thermal history in the Caribbean, although the relationship was not uniform across all four signs of disease nor was the effect uniform among coral species.

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**Literature Cited**


Supplemental Material

Appendix A

Location and assemblage descriptions (Ecological Archives E095-173-A1).

Appendix B

Results of all pairwise relative-risk assessments (Ecological Archives E095-173-A2).

Appendix C

Pairwise Morlet cross-wavelet and coherence-wavelet analyses (Ecological Archives E095-173-A3).

Supplement

Source code for the relative-risk model (OpenBUGS) (Ecological Archives E095-173-S1).