Coral disease hotspots in the Caribbean

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Abstract. Recent outbreaks of coral diseases in the Caribbean have been linked to increasingly stressful sea-surface temperatures (SSTs). Yet, ocean warming is spatially heterogeneous and therefore has the potential to lead to hotspots of disease activity. Here, we take an epidemiological approach to examine spatial differences in the risk of white-band disease on *Acropora* spp. and yellow-band disease on *Orbicella* spp. in the Caribbean. Our analysis involved examining the spatial patterns of disease prevalence, and creating a Bayesian-risk model that tested for regional differences in disease risk. The spatial examination of disease prevalence showed several clusters of white-band disease, including high prevalence in the Turks and Caicos, Jamaica, Puerto Rico, the Virgin Islands, and Belize, whereas yellow-band disease seemed most prevalent along the Yucatan Peninsula. The Bayesian-risk model showed regional clusters of white-band disease near the southern Dominican Republic, Puerto Rico, the Virgin Islands, and the Lesser Antilles, whereas the risk of yellow-band disease was highest in the southern Caribbean. The relative risk of both diseases increased with warmer SSTs. The Bayesian-risk model allowed us to predict where we should expect future outbreaks of coral diseases at a regional scale, and suggests regions where the implementation of disease mitigation plans may be most urgent.

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INTRODUCTION

During the last three decades, reef corals have experienced unprecedented thermal stress (Glynn 1993, Hoegh-Guldberg 1999, Aronson et al. 2000). Such extreme thermal stress has led to extensive coral bleaching, coral mortality, and shifts in coral community composition (Loya et al. 2001, van Woesik et al. 2011). In addition, there has been a purported increase in the number of coral disease outbreaks in the Caribbean (Aronson and Precht 2001, Harvell et al. 2002, Altizer et al. 2013), which also has contributed to extensive declines in coral populations (Cruz et al. 2014, Loh and Pawlik 2014). Although it has been proposed that thermal stress is a driver of several coral diseases, we are just beginning to understand the environmental conditions that trigger coral disease outbreaks (Harvell et al. 2002, Lesser et al. 2007, Muller et al. 2008, Randall and van Woesik 2015). Furthermore, it is unknown whether there are emerging hotspots of coral disease activity in the Caribbean.

Geographic patterns of sea-surface temperature (SST) anomalies are predictable, although they vary spatially and temporally (Thompson and van Woesik 2009, Burrows et al. 2011). There are two main regions in the Caribbean that have experienced frequent thermal anomalies (every four to seven years) during the past few centuries—these same locations also recently have experienced the most intensive thermal stress (Thompson and van Woesik 2009). The first

region is centered on Puerto Rico and extends west to the Dominican Republic and east to the Virgin Islands. The second region is centered on eastern Costa Rica and extends north to Nicaragua and south to Panama (Randall et al. 2014). If these thermal-anomaly patterns persist into the near future, as most models suggest (IPCC 2014), then some regions in the Caribbean will receive both more intensive and more frequent thermal stress than other regions. This spatial heterogeneity in thermal stress, combined with a projected increase in frequency and intensity of thermal-stress events, will presumably influence the prevalence of coral bleaching and disease, resulting in geographic hotspots of disease.

Disease hotspots are characterized by increased rates of disease transmission and higher disease prevalence than in surrounding areas; these hotspots can serve as source areas for the dispersal of pathogens to less-infected localities (Paull et al. 2012). Coral disease hotspots can arise through a number of mechanisms driven by thermal stress. Firstly, elevated SSTs may increase the growth rate and virulence of pathogens, leading to higher pathogen densities and higher rates of infectivity (Kushmaro et al. 1998, Toren et al. 1998, Harvell et al. 2002, Rosenberg et al. 2007). Secondly, elevated SSTs may compromise coral immunity resulting in an increased number of susceptible and infected hosts (Ritchie 2006, Lesser et al. 2007, Muller et al. 2008, Mydlarz et al. 2010, Reed et al. 2010). Thirdly, warm winters increase pathogen survival rates and lengthen the duration of disease activity (Harvell et al. 2002, Randall and van Woesik 2015). Finally, thermal stress can lead to the loss of symbiotic dinoflagellates, which compromises coral health and further increases coral susceptibility to disease (Glynn 1984, Brown 1997, Muller et al. 2008). These thermal stress hotspots can result in severe consequences for coral populations, thus leading to outbreaks of disease at a regional scale (Paull et al. 2012).

Here, we take an epidemiological approach to examine whether the spatial variance in SSTs and in rates of ocean warming are associated with spatial patterns of two coral diseases, white-band disease and yellow-band disease, in the Caribbean. We examine the spatial patterns of disease prevalence and develop a Bayesian-risk model that considers disease preva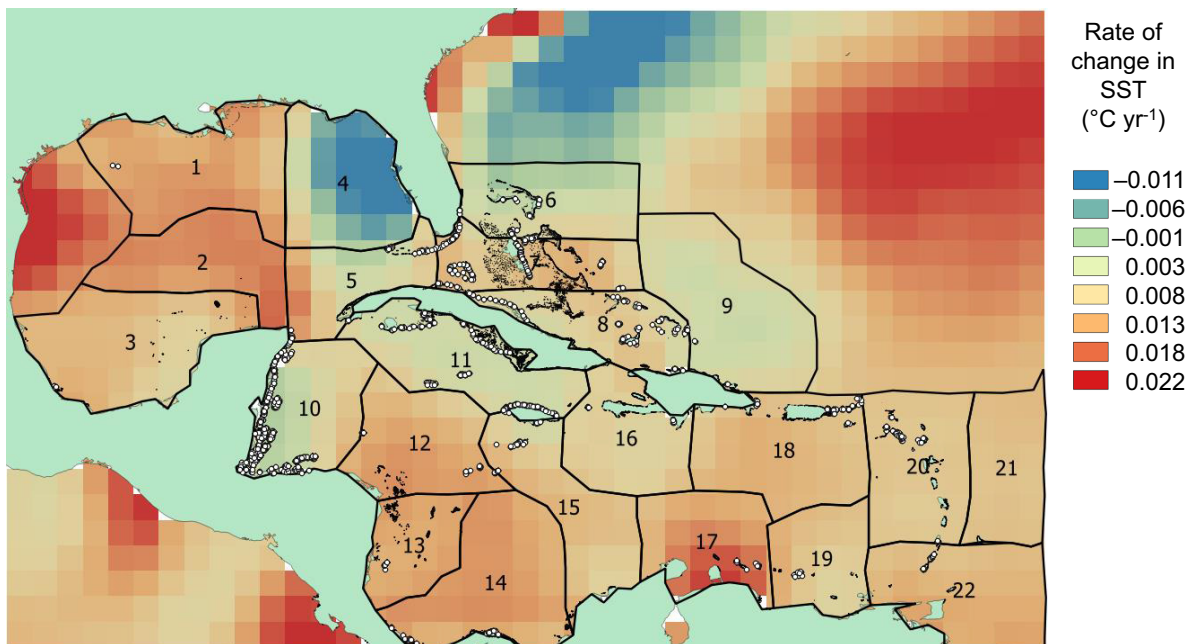lence in relation to population size, and examines spatial autocorrelation so as not to assume spatial independence of adjacent regions (Hurlbert 1984, Legendre 1993, Rangel et al. 2006). We test both models on data of white-band disease on *Acropora* spp., and yellow-band disease on *Orbicella* spp. Both diseases have caused major declines in their respective host coral populations throughout the Caribbean (Porter and Meier 1992, Aronson and Precht 2001, Miller et al. 2002, Bruckner and Bruckner 2006). Specifically, the objectives of the study were to (1) examine spatial differences in the relative risk of two coral diseases prevalent in the Caribbean (i.e., white-band disease and yellow-band disease), (2) determine whether there are relationships between the two coral diseases and SSTs, and (3) define the geographic locations of potential coral-disease hotspots in the Caribbean.

METHODS

Coral diseases and spatial models

We used count data to estimate disease prevalence. Members of the Atlantic and Gulf Rapid Reef Assessment Program (www.agrra.org) collected count data on coral diseases from 2076 sites that were surveyed between 1997 and 2014 (Fig. 1). At each site, divers recorded the total number of *Acropora* spp. and *Orbicella* spp. coral colonies, and the presence or absence of disease signs on each colony. We were interested in examining the spatial distribution and abundance of *Acropora* and *Orbicella* coral colonies, and the prevalence of white-band disease and yellow-band disease, respectively.

We were also interested in obtaining information on the risk of a particular disease relative to the regional coral population. We used N to represent the size of the total coral population, n_i to represent the population in a given region i that was potentially susceptible to a given disease, O to represent the total observed number of individual colonies with disease, and o_i to represent the observed number of individuals with a disease in each region. To obtain an estimate of risk, we compared the observed number of cases of disease, o_i, with an expected number of cases of disease, e_i, based on the population in a given region, n_i. We calculated the standardized expected ratio in the ith region, as:

ID	Region
1	Northern GOM
2	Central GOM
3	Southern GOM
4	W Florida
5	SW Florida
6	E central Florida and N Bahamas
7	S Florida and central Bahamas
8	N Cuba, S Bahamas & Turks and Caicos
9	N Dominican Republic
10	Yucatan Coast
11	S Cuba

ID	Region
12	Honduras
13	Nicaragua
14	Costa Rica and Panama
15	Jamaica to Colombia
16	S Haiti
17	W Venezuela
18	S Dominican Republic
19	Central Venezuela
20	Inner E Caribbean Islands
21	Outer E Caribbean Islands
22	E Venezuela and Trinidad

Fig. 1. The coral reefs of the Caribbean (black), with rates of change in sea-surface temperature (SST; °C yr^{-1}) over the last 45 years (1968–2012; colors), and the proposed 22 climatic regions of the Caribbean, which were defined based on rates of change in monthly SST calculated with monthly 1° by 1° HadISST data. White circles indicate coral disease survey sites. GOM, Gulf of Mexico.

$$e_i = (o_i/n_i) \times n_i. \quad (1)$$

Since we were dealing with count data, the common assumption of most disease models is that the number of cases of a disease in a region can be drawn from a Poisson distribution (Pascutto et al. 2000) as:

$$o_i \sim \mathrm{Poisson}(\lambda_i), \quad (2)$$

$$\lambda_i = \theta_i \times e_i, \quad (3)$$

where θ_i is the relative risk of the disease in region i. The Poisson distribution assumes that the mean and variance are the same, yet in nature, particularly on coral reefs, the variance of a population is frequently higher than the mean of the population. To compensate for this over-dispersion, we also constructed a mixed model (Bivand et al. 2013) using a negative binomial (nbin) distribution for relative risk (θ) and a gamma distribution to account for the variance (var) as:

$$o_i \sim \text{nbin}(\lambda_i, \text{var}_i), \qquad (4)$$

$$\lambda_i = \theta_i \times e_i, \qquad (5)$$

$$\text{var}_i \sim \text{gamma}(v, \alpha), \qquad (6)$$

$$\theta_i \sim \text{gamma}(\mu, \tau), \qquad (7)$$

where the priors of the parameters v, α, μ, and τ, for the gamma distributions, were uninformative (see the Appendix S1 for annotated R and Open-Bugs code). From these equations, we determined whether any region was characterized by higher or lower cases of disease than expected by chance alone.

We also examined whether there were any effects of spatial structure on the Bayesian-risk model using conditional autoregressive estimates (Besag et al. 1991). These estimates considered spatial adjacencies of regions, and the strength of the relationship between neighboring regions based on their size (km^2), as:

$$o_i \sim \text{Poisson}(\theta_i \times e_i), \qquad (8)$$

$$\log(\theta_i) = \alpha + \beta(x_i - \text{mean}(x_i)) + v_i \qquad (9)$$

where α is the intercept, β is the slope of the covariate x_i, and v_i is the conditional variance of the spatial autoregression specifications using geographic adjacency matrices. To reduce the open-endedness of the Markov chain Monte Carlo estimates, we censored the normal distribution of the prior on the β values between -2 and 2 (Spiegelhalter et al. 2002). The Bayesian-risk model, using conditional autoregressive estimates, was developed in Open-Bugs (Lunn et al. 2000) and was run in R (R Core Team 2016) using the packages "R2OpenBUGS" (Sturtz et al. 2005), "sp" (Pebesma and Bivand 2005), and "spdep" (Bivand and Piras 2015). Model outputs were visualized in R using the packages "rColorBrewer" (Neuwirth 2014), "raster" (Hijmans 2015), and "rgdal" (Bivand et al. 2015).

Temperature data

Mean monthly SSTs from 1968 to 2012 were obtained from the MetOffice HadISST records at a 1° by 1° spatial resolution (Fig. 1; Rayner et al. 2003). The years 1968–2012 were selected to include a 30-year SST record leading up to the first disease survey in 1997. Linear models were used to calculate the 45-year rates of change in SST for every grid cell in the Caribbean region. The rate of change in SST was then used to divide the Caribbean into 22 non-overlapping regions with similar rates of change and with similar spatial coverage (Fig. 1). We chose a course grain-size for this hotspot assessment, although the area of interest can be divided into fewer or more regions depending on the question being asked. For every region, we calculated the following SST variables from 1968 to 2012: (1) mean SST (Fig. 2, top panel), (2) maximum SST, (3) minimum SST, (4) mean rate of change in SST, (5) maximum rate of change in SST, (6) minimum rate of change in SST, and (7) range in SST. Each of the SST variables was tested in the models to determine which, if any, metrics were the best predictors of disease risk.

For each of the 22 regions, the average prevalence of disease was calculated for each host species. All models (for Eqs. 1–9) were analyzed using a Bayesian approach, with uninformative priors (Gelman et al. 2004). The models were run using 3000 Markov chain Monte Carlo simulations in OpenBUGs, which were implemented through R (R Core Team 2016) to obtain posterior probability distributions. All the R and Open-Bugs codes, the data, and the shapefiles are annotated and available online in Appendix S1.

Results

Acropora colonies were present at 38% of the sites that were surveyed (Fig. 3, top left). White-band disease on *Acropora* was particularly prevalent in the Turks and Caicos, Jamaica, Puerto Rico, the Virgin Islands, and Belize (Fig. 3, bottom left). The Bayesian-risk model, without considering spatial dependencies, showed areas of high risk of white-band disease on *Acropora*, which were located in the southern Dominican Republic, Puerto Rico, the Virgin Islands, and the Lesser Antilles (Fig. 2, center). Northern Cuba also showed relatively high risk of white-band disease (Fig. 2, center).

When spatial dependency among adjacent regions was considered in the calculations (Eq. 9), and when mean SST was used as the covariate in the autoregressive estimates of the Bayesian-risk model (Eq. 9), there were three main hotspots of high risk of white-band disease on *Acropora*— southern Hispaniola and Puerto Rico, the Virgin Islands, and western Venezuela (Fig. 2, bottom). Honduras and Nicaragua also showed relatively
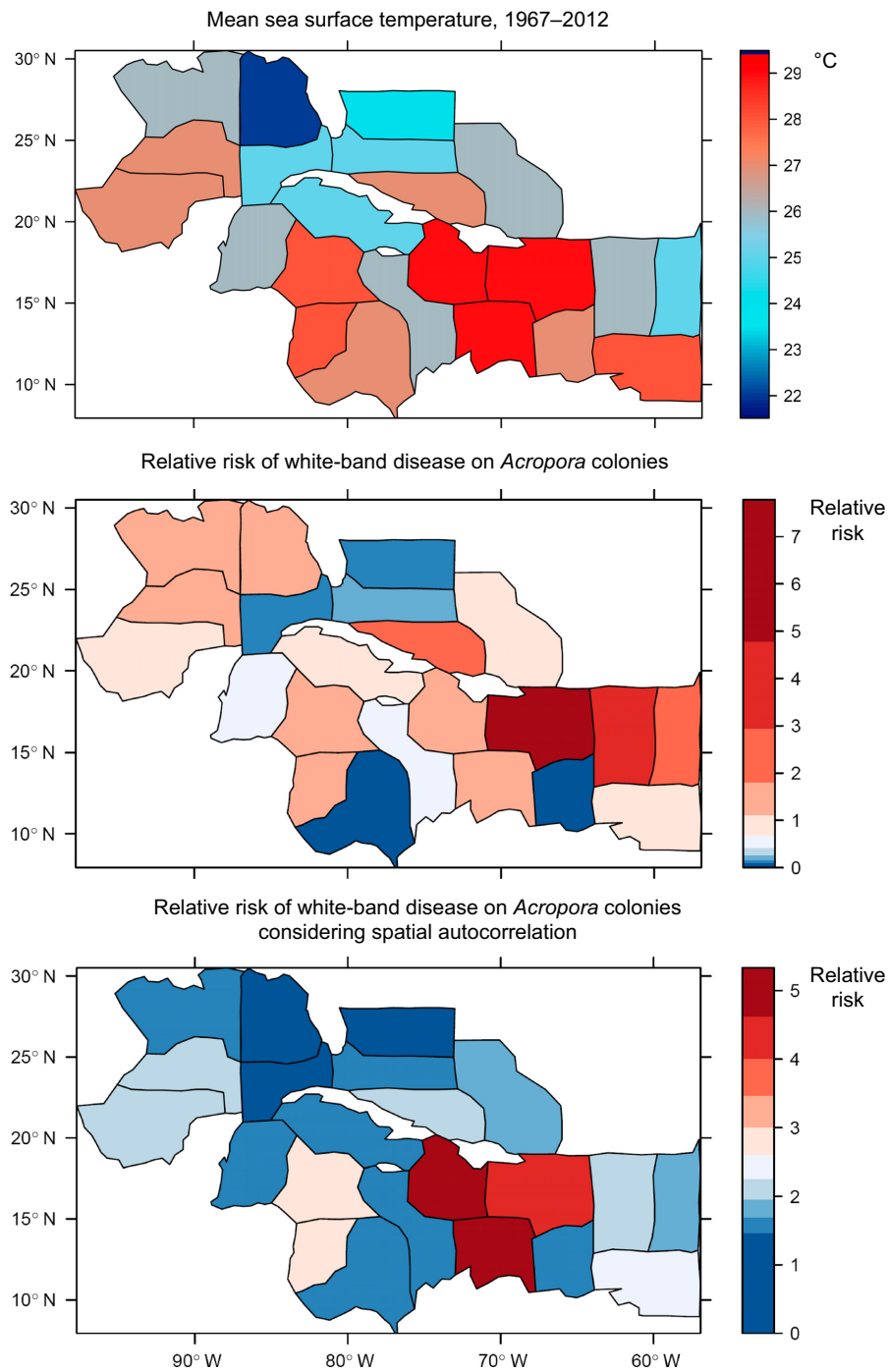
Fig. 2. Top panel: mean sea-surface temperature (SST) (°C) of 22 climatic regions from 1967 to 2012. Center panel: relative risk of white-band disease on *Acropora* spp. coral colonies, from 1997 to 2014, using Poisson-gamma relative risk estimates. Bottom panel: relative risk of white-band disease on *Acropora* spp. coral colonies, from 1997 to 2014, using Poisson-gamma relative risk estimates and examining spatial autocorrelation (Eqs. 8, 9) against the mean SST. Note that the relative risk scales vary between panels.
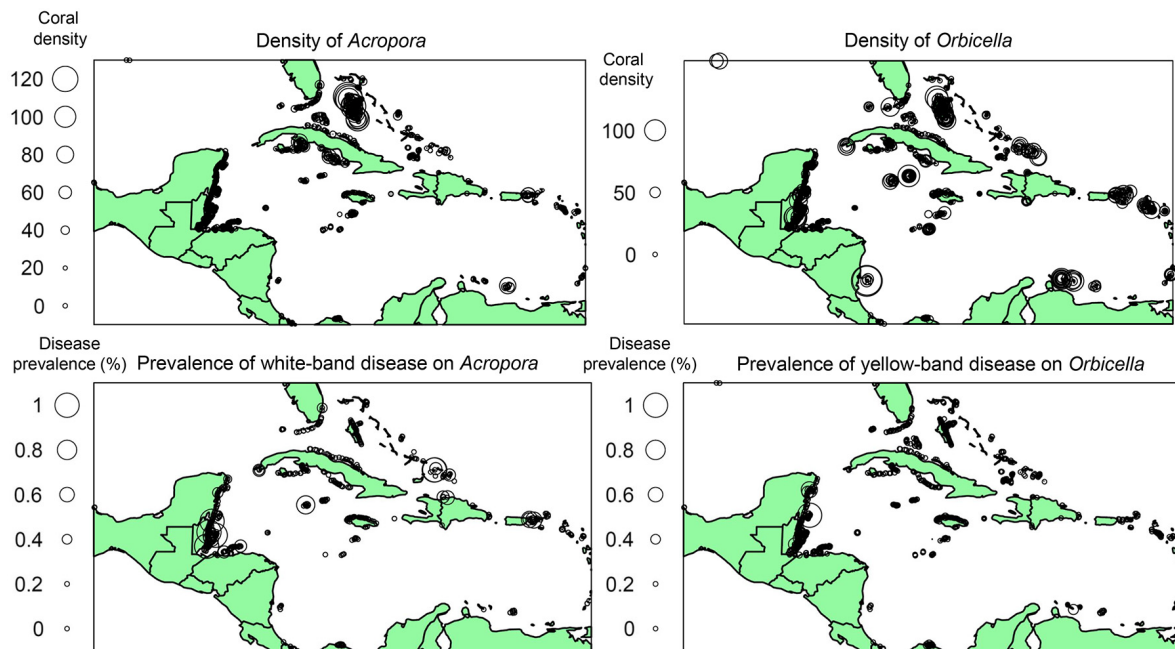
Fig. 3. The density of coral colonies per site (upper panels) and the prevalence of coral diseases per site (lower panels). The distribution and abundance of *Acropora* spp. coral colonies (upper left) and the average prevalence of white-band disease on *Acropora* (lower left) at 791 sites, from 1997 to 2014. The distribution and abundance of *Orbicella* spp. coral colonies (upper right) and the average prevalence of yellow-band disease on *Orbicella* (lower right) at 1536 sites, from 1997 to 2014.

Table 1. Results of Bayesian Poisson-gamma relative risk models used to evaluate the relationship between disease risk and sea-surface temperature (SST) variables.

Species	Variables	Beta	2.5% CI	95% CI
Acropora spp.	Mean SST	**1**	**−0.3**	**1.9**
	Maximum SST	−0.1	−1.9	1.9
	Minimum SST	0.2	0	0.4
	Average rate of change in SST	0.2	−1.9	1.8
	Maximum rate of change in SST	−0.1	−1.9	1.8
	Minimum rate of change in SST	0	−1.9	1.9
	Range in SST	−0.3	−0.8	0.1
Orbicella spp.	Mean SST	**0.6**	**−0.1**	**1.4**
	Maximum SST	−0.4	−1.7	0.8
	Minimum SST	0.2	−0.1	0.5
	Average rate of change in SST	0.1	−1.9	−1.9
	Maximum rate of change in SST	0.1	−1.9	1.9
	Minimum rate of change in SST	0.2	0	0.4
	Range in SST	−0.2	−0.5	0.1

Notes: CI, credible interval. White-band disease was evaluated on *Acropora* spp. coral colonies. Yellow-band disease was evaluated on *Orbicella* spp. coral colonies. Bolded coefficients indicate significant statistical results.

high risk of white-band disease (Fig. 2, bottom). The relationship between mean SST and white-band disease was positive and strong (β = 1, 95% credible intervals (CI) = −0.3, 1.9; Table 1). However, when the mean rates of change and the maximum rates of change in SST were considered as model covariates, the spatial relationships were weak, with high uncertainty (mean rate of change in SST β = 0.2, CI = (−1.9, 1.8); maximum rate of change in SST β = −0.1, CI = −1.9, 1.8; Table 1). These results suggest that the higher the mean SST at a given location, the more likely *Acropora* were to show signs of white-band disease.

Orbicella colonies were present at 74% of the sites (Fig. 3, top right), and yellow-band disease was particularly prevalent along the Yucatan Peninsula (Fig. 3, bottom right). Yet, the Bayesian-risk model, without considering spatial dependencies, showed that the areas at highest risk of yellow-band disease on *Orbicella* were actually in Honduras, Costa Rica and Panama, Jamaica and Colombia, and central Venezuela and Trinidad (Fig. 4, top). The autoregressive calculations of the Bayesian-risk model showed hotspots in the southern Caribbean, although the relative risk was weak, at a maximum of 2.7 (compared with 5.5 for *Acropora*; Fig. 4, bottom). The relationship between mean SST and yellow-band disease was positive and strong (mean SST β = 0.6, 95%, CI = −0.1, 1.4). Again, the maximum SST and the rates of change in SST showed weak relationships with yellow-band disease,
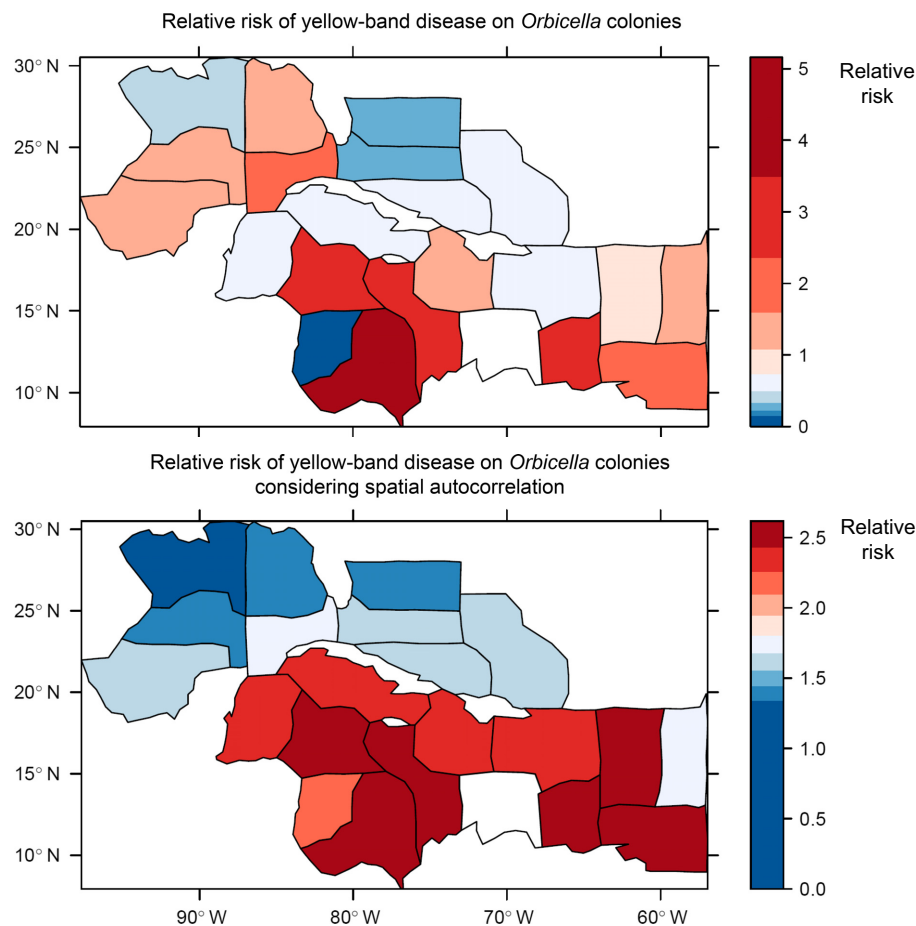

Fig. 4. Top panel: relative risk of yellow-band disease on *Orbicella* spp. coral colonies, from 1997 to 2014, using Poisson-gamma relative risk estimates (Eqs. 2, 7). Bottom panel: relative risk of yellow-band disease on *Orbicella* spp. coral colonies, from 1997 to 2014, using the Poisson-gamma relative risk estimates and examining spatial autocorrelation (Eqs. 8, 9) against the mean sea-surface temperature. Note that the relative risk scales vary between panels.

with high uncertainty (Table 1). Like white-band disease, these results suggest that the higher the mean SST at a given location, the more likely *Orbicella* showed signs of yellow-band disease.

DISCUSSION

>Here, we examined the spatial patterns in the prevalence of white-band disease on *Acropora* and yellow-band disease on *Orbicella* in the Caribbean. We applied a Bayesian-risk model to examine regional spatial patterns of disease risk and determine whether there were relationships between the diseases and SSTs. Identifying regions with a high disease prevalence is usually the first step toward the potential management of a disease (Snow 1854), yet the spatial patterns of disease prevalence that were identified did not always match the regions with increased disease risk.

The Bayesian-risk model identified spatial clusters of relative disease risk, considering regional population size and adjacency. Both the spatially dependent and spatially independent estimates showed that the highest relative risk of white-band disease on *Acropora* was evident in the southern Dominican Republic, Puerto Rico, the Virgin Islands, western Venezuela, Honduras, and Nicaragua (Fig. 2, center and bottom). Both the spatially dependent and spatially independent estimates showed that the highest relative risk of yellow-band disease on *Orbicella* was evident in Panama, Honduras, Jamaica and Colombia, and central Venezuela (Fig. 4). Interestingly, these same regions were previously identified as showing historically high return frequencies of thermal anomalies (Thompson and van Woesik 2009, Randall et al. 2014). Although the autoregressive models showed no relationship with rates of change in SST, there were strong and positive relationships between mean SST and both coral diseases.

Taking spatial autocorrelation into consideration in the relative risk models resulted in similar spatial patterns of risk compared with the model without spatial autocorrelation, but the conditional autoregressive models showed lower risk overall, and generally fewer regions at risk. These conditional autoregressive models consider spatial dependency among regions and therefore do not treat each region as completely independent. In an epidemiological context, autoregressive models that include spatial dependency might be most useful when the disease of interest stems from waterborne contagious pathogens and when there is considerable water exchange among adjacent regions that promote microbial and genetic connectivity (e.g., Precht et al. 2016). Such models may be less necessary when diseases are not contagious (e.g., Randall et al. 2016), when they are a consequence of infections by ubiquitous pathogens, or when they are driven mostly by environmental conditions (e.g., Muller et al. 2008). Because both diseases tested in this study showed strong and positive relationships with mean SST, the disease hotspots identified by both relative risk models are worth considering and developing further.

We chose a course-grained approach, using a Bayesian-risk model, to detect regional disease hotspots, although the same methods can be applied to detect hotspots or disease microrefugia at a finer scale (Mosblech et al. 2011). The same modeling approach can also be taken within a spatiotemporal framework, with all Eqs. 1–9 subscripted with a time component, t, where time-series data are available. Such an approach would not only test the dynamics of diseases through climate cycles, but would also test the long-term efficacy of management intervention to control coral diseases. While these data are not yet available, numerous restoration and monitoring programs may soon be able to make use of these models at an even finer spatial and temporal resolution.

Over the next century, the climate is predicted to continue to drive ocean temperatures considerably higher than any temperatures experienced by reef corals for over the last 700,000 years (Hoegh-Guldberg et al. 2007, Hansen et al. 2010, IPCC 2014). The results of the Bayesian-risk model suggest that the risk of white-band disease and yellow-band disease increases in regions with warmer-than-average SSTs (Lesser et al. 2007, Muller et al. 2008, Randall et al. 2014, Randall and van Woesik 2015). High SSTs will most likely continue to cause thermal stress in corals and will consequently increase the prevalence of coral diseases in these regional hotspots. Attention should be focused on reefs in the regional disease hotspots identified in the Dominican Republic, Puerto Rico, the Virgin Islands, Venezuela, Honduras, Costa Rica, and Panama to begin efforts to mitigate disease transmission in the Caribbean.

Acknowledgments

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Supporting Information

Additional Supporting Information may be found online at: http://onlinelibrary.wiley.com/doi/10.1002/ecs2.1814/full