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note

Differential disease incidence and mortality of inner and outer reef corals of the upper Florida Keys in association with a white syndrome outbreak

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ABSTRACT.—The Florida Keys Reef Tract has suffered extraordinary losses in live coral cover over the past four decades and is now battling an unprecedented coral disease outbreak. Here, colonies of *Siderastrea siderea* (Ellis and Solander, 1786) and *Pseudodiploria strigosa* (Dana, 1846) were tracked over 3 yrs (2015–2017) across two pairs of inner and outer reef sites in the upper keys, offering a unique perspective into the distribution of disease throughout the reef tract. We found that outer reef colonies of both coral species exhibited greater disease incidence and mortality associated with this ongoing epidemic, while inner patch reef colonies remained largely unaffected. These findings suggest that ecological or biological factors that differentiate coral populations across these reef zones may play an important role in susceptibility to disease.

Ocean warming and acidification are widely recognized as posing the most significant risks to coral reef health and resilience on a global scale (Hoegh-Guldberg et al. 2007). In addition to these abiotic factors, corals are also faced with the persistent threat of disease, which can cause localized mortality at levels comparable to mass bleaching events (Aronson and Precht 2001). The Florida Keys Reef Tract (FKRT), in particular, has experienced several major disease outbreaks over the past four decades that have drastically altered the health and composition of the reef community (Porter et al. 2001).

The earliest reports of disease on the FKRT documented the occurrence of what was described as coral "plague" at Carysfort Reef in the upper keys, which manifested as an advancing line of infection causing rapid tissue mortality in six species of reef-building corals (Dustan 1977). Similar disease symptoms, aptly termed *plague type II*, reemerged in the same region two decades later and spread throughout the FKRT, this time infecting at least 17 species of corals (Richardson et al. 1998). Since then, reports of new, virulent coral diseases on Caribbean reefs have increased considerably (Goreau et al. 1998, Aronson and Precht 2001, Weil 2004). These include the spread of white band and white pox diseases, which have decimated populations of *Acropora*

palmata (Lamarck, 1816) and *Acropora cervicornis* (Lamarck, 1816) (Aronson and Precht 2001), the ubiquitous, albeit less destructive, incidence of black band, yellow band, and dark spots diseases (Kuta and Richardson 1996, Gil-Agudelo et al. 2004), and most recently, a new outbreak of white plague-like symptoms on the FKRT that is unprecedented in its virulence and scale of damage (Precht et al. 2016).

This most recent outbreak was first identified in 2014 off the coast of Virginia Key (25.748°N,–80.100°W), and over the course of 3 yrs, it has radiated to the northern and southern reaches of the FKRT (Precht et al. 2016, Muller et al. 2018). Although efforts are still underway to fully understand the etiology of the epidemic, local monitoring teams have reported previously characterized symptoms of white plague, as well as several uncharacterized diseases that include "white blotch," "white bleaching band," and symptoms resembling rapid tissue loss, which have collectively affected at least 20 species of corals on the FKRT (Lunz et al. 2017). Here, we capitalized on an existing long-term monitoring effort to evaluate post-coring regrowth rate, disease incidence, and mortality outcomes of two abundant reef-building coral species, *Siderastrea siderea* (Ellis and Solander, 1786) and *Pseudodiploria strigosa* (Dana, 1846), at two cross-shore transects in the upper FKRT. Using images taken during three annual site visits from 2015 to 2017, we tested our hypothesis that the effects of the recent disease outbreak would differ between inner and outer reef sites due to cross-shore differences in environmental conditions.

Materials and Methods

Two pairs of inner and outer reef sites in the upper FKRT were visited in April 2015, April 2016, and October 2017 (Fig. 1). Inner reef sites, Basin Hill Shoals (BH) and Cheeca Rocks (CR), are located on the seaward boundary of Hawk Channel and are characterized as isolated high-relief patch reef structures interspersed among unconsolidated sand and seagrass. By comparison, outer reef sites were located outside the Carysfort (CF) and Alligator Reef (AR) Sanctuary Preservation Areas and comprise part of the continuous barrier reef system that borders the offshore edge of the continental shelf along the extent of the FKRT. Vertical relief at these sites is primarily attributable to the limestone remains of relict reef structures with relatively sparse distributions of live coral (Marszalek et al. 1977). Similar to the patterns documented by Ruzicka et al. (2013) in a keys-wide survey of reef community structure, the inner patch reef sites had greater live coral cover with many large (>1 m diameter) colonies of mounding corals, while the outer reef sites were dominated primarily by octocorals and macroalgae.

On the initial site visits in 2015, as part of an associated investigation of historical coral growth trends (Rippe et al. 2018), skeletal cores were extracted from visibly healthy colonies of *S. siderea* and *P. strigosa* at each of the four study sites. Five colonies of each species were sampled at both CF and BH, four colonies of each species were sampled at AR, while five colonies of *S. siderea* and four colonies of *P. strigosa* were sampled at CR (Online Table S1). Concrete plugs were inserted and cemented into drilled holes with Z-Spar® underwater epoxy to protect the coral colonies from erosion and further physical damage. To document tissue regrowth rates over the coring site, a 20 × 20-cm quadrat was centered on the core extraction site, and an initial image was captured using a GoPro® camera (i.e., Fig. 2). On subsequent visits, cored colonies were located and analogous images recaptured to yield three annual

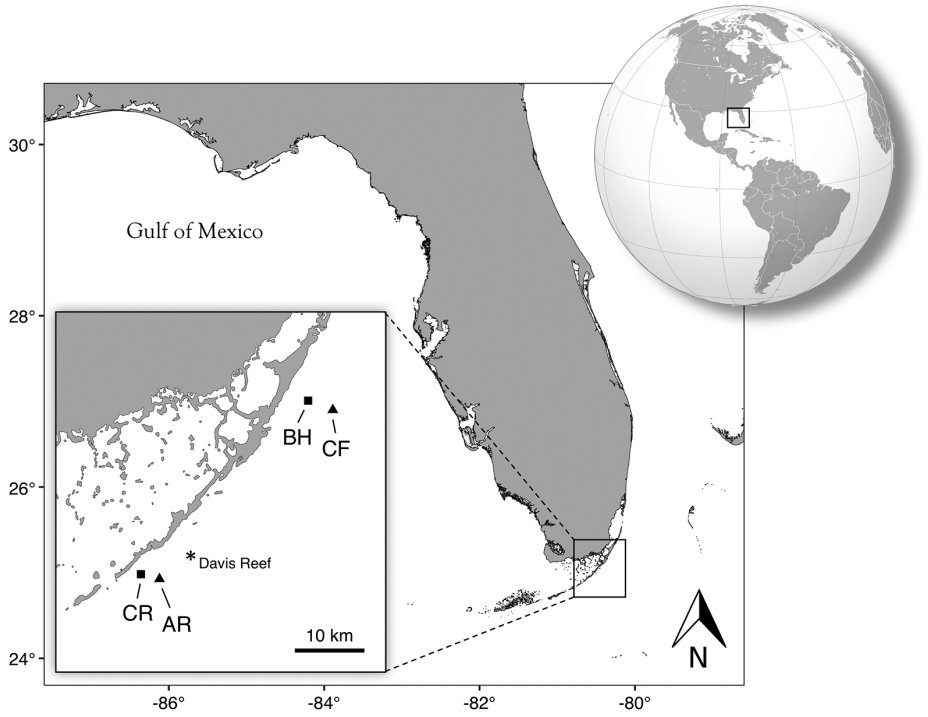

Figure 1. Squares and triangles represent inner and outer reef sites, respectively. Site abbreviations are as follows: Basin Hill Shoals (BH), Carysfort Reef (CF), Cheeca Rocks (CR), and Alligator Reef (AR). Davis Reef, referenced in the text, is indicated with an asterisk.

photographs per colony. Using ImageJ software (Schneider et al. 2012), we outlined the margin of living tissue encircling the coring hole in each image to calculate total exposed surface area, standardized using the quadrat. Across each annual interval, the difference in area was calculated as the total percent change divided by the number of days between successive images to yield an average regrowth rate for each colony. The effect of year, reef zone, and species on regrowth rate was assessed using a three-way analysis of variance (ANOVA) in R (R Core Team 2017).

Mortality and disease occurrence were assessed qualitatively for each of the imaged corals at each time point during the study period. Coral colonies were evaluated as living (<5% tissue loss), dead (>95% tissue loss), or partially dead (5%–95% tissue loss) based on estimated percentages of tissue loss visible in the images. Disease state was evaluated as either present or absent, and specific symptoms were identified based on recently published accounts of coral diseases on the FKRT (Porter et al. 2011, Precht et al. 2016). Because the etiology of the current disease outbreak remains largely uncharacterized, we use the terms *white plague* and *white syndrome* to refer to any white plague–like symptoms affecting *P. strigosa* and *S. siderea*, respectively.

Ambient environmental conditions at each site were compared using high-resolution satellite-derived daily sea surface temperature (SST) and ocean color data collected from 2003 to 2016 and 2012 to 2017, respectively. SST data were obtained at 1-km resolution from the Group for High Resolution Sea Surface Temperature (GHRSST) Level 4 Multi-scale Ultra-high Resolution (MUR) SST Analysis data set. To characterize ocean color, we used Level 2 chlorophyll-*a* and K_d490 data (approximately

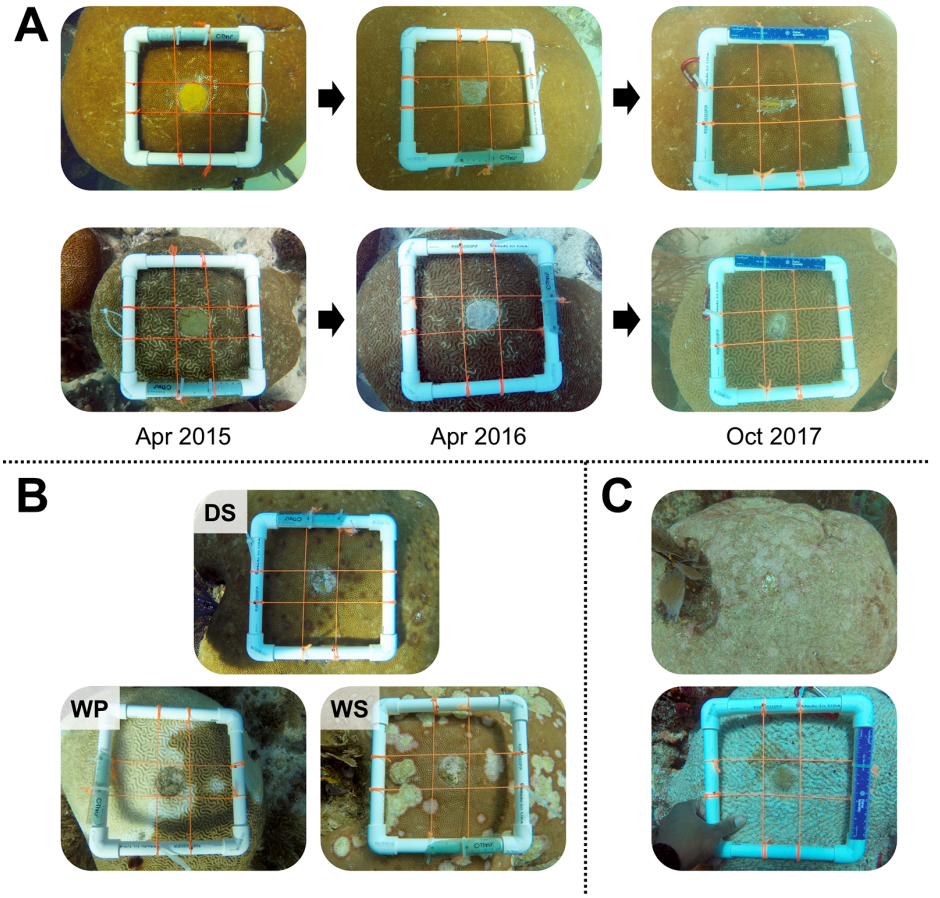

Figure 2. (A) Tissue regrowth following coring was tracked for the study species, *Siderastrea siderea* (top) and *Pseudodiploria strigosa* (bottom). Colonies exhibiting (B) symptoms of: dark spots (DS), white plague (WP), and white syndrome (WS), and (C) mortality were recorded during each site visit.

750 m resolution) collected from the Visible Infrared Imaging Radiometer Suite sensor aboard the Suomi NPP satellite and compiled by the US National Oceanic and Atmospheric Administration's CoastWatch/OceanWatch. K_d490 represents the diffuse attenuation coefficient at 490 nm wavelength (blue-green region), which can be used as a proxy for water column turbidity (Shi and Wang 2010).

Lastly, to place our findings within the context of overall coral disease trends in the area, we analyzed publicly-available observations from annual surveys conducted by Florida Reef Resilience Program (FRRP) in the upper keys region from 2014 to 2016; FRRP surveys were not conducted in the area in 2017 and thus could not be included in the analysis (Florida Reef Resilience Program 2018). Disease prevalence was measured as the total percentage of symptomatic corals (any disease) at each site, and ordinary kriging was used to spatially interpolate between survey sites. Results were then plotted on a 400 × 400 cell raster grid overlaying our study region. Analyses were performed using the gstat package in R (Pebesma 2004, R Core Team 2017). Importantly, of all disease observations in the FRRP survey dataset, 87.3% were of

dark spots disease, while only 1.8% were of white plague–related disease (i.e., white plague, white blotch, and patchy necrosis) and 8.2% were of unknown disease. Thus, the spatial and temporal patterns revealed by this data set largely reflect those of dark spots disease, not of white plague–related symptoms.

Statistical Models.—The relative effect of year, reef zone (i.e., inner and outer reef), and species on the tissue condition of the colonies (i.e., living, partial mortality, full mortality) was assessed using an ordinal logistic regression model implemented with the MASS package in R (Venables and Ripley 2002). A binomial generalized linear model was then used to assess the relative effect of year, species, reef zone, and post-coring tissue regrowth rate on the occurrence of white plague–like symptoms. Regrowth was included in this model to determine whether coring operations may have heightened susceptibility of corals to this disease outbreak. Note, because symptoms were perfectly separated across reef zones in our samples (i.e., white plague–like symptoms were not observed at inner reef sites for either species), a penalized regression was used to more reasonably reflect the true population level effect of reef zone on disease occurrence. The penalty term was computed using the cross-validation procedure implemented with the glmnet package in R (Friedman et al. 2010).

Results and Discussion

The 3-yr photographic time-lapse presented here revealed a striking distinction in the prevalence of white plague–like symptoms and mortality across inner and outer reef habitats of the upper Florida Keys. Between the initial site visits in May 2015 and final return in October 2017, all previously-labeled *P. strigosa* colonies (3 of 3) and two-thirds of *S. siderea* colonies (4 of 6) that were found at outer reef sites exhibited complete mortality (Fig. 3). Three of the four dead *S. siderea* colonies had exhibited symptoms of white syndrome in 2016; no other colonies of either species showed any signs of disease in 2016 before succumbing. The two surviving colonies of *S. siderea* that were found had experienced partial tissue mortality associated with white syndrome that continued through 2017.

Notably, none of the previously cored *P. strigosa* colonies at CF could be located in 2017; however, every *P. strigosa* colony that the dive team encountered was dead, suggesting that this species had been severely impacted at this site between 2016 and 2017. Moreover, of the five *P. strigosa* colonies imaged at this site 1 yr earlier (2016), two had completely died and two exhibited partial mortality associated with an infection of white plague. Without thorough benthic surveys, it is difficult to assign an exact measure to the past extent of this species at this site. However, like many outer reef areas of the Florida Keys Reef Tract (Ruzicka et al. 2013), this site near Carysfort Reef was largely dominated by octocorals and macroalgae with few isolated colonies of stony corals in areas of hard substrate. We estimate that fewer than 50 dead colonies of *P. strigosa* were encountered across this site, and fewer than 10 were >0.5 m in diameter. During the study period, no colonies of either species showed any sign of recovery from infection by white plague or white syndrome.

Conversely, all *S. siderea* colonies (7 of 7) and all but two colonies of *P. strigosa* (5 of 7) that were found at inner reef sites in 2017 were healthy without tissue loss (Fig. 3). Two inner reef colonies of *P. strigosa* experienced significant partial mortality

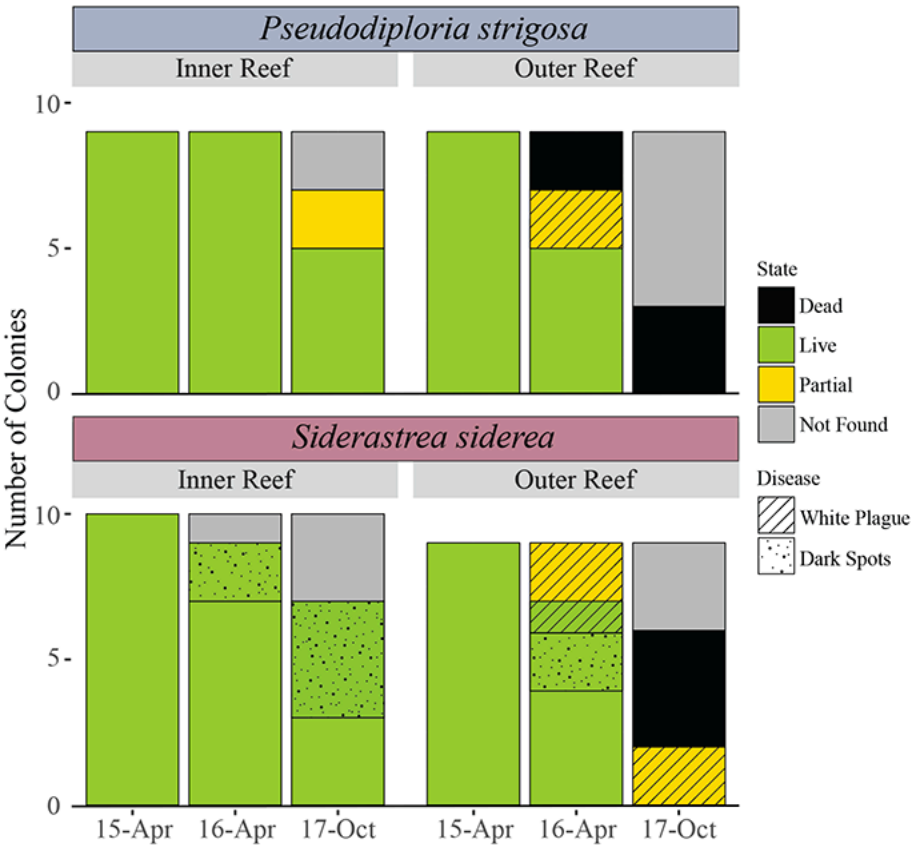

Figure 3. Total count of *Pseudodiploria strigosa* (top) and *Siderastrea siderea* (bottom) colonies exhibiting disease and mortality during each site visit. Colors denote the state of mortality (green: >95% living tissue, yellow: 5%–95% living tissue, black: <5% living tissue, gray: not found). Diagonal and spotted patterns denote symptoms of white plague or syndrome and dark spots disease, respectively.

between 2016 and 2017, but did not show any signs of disease. Thus, we cannot be certain whether the tissue mortality observed here resulted from temperature-induced bleaching, disease infection, or an unknown cause. Interestingly, all colonies of *S. siderea* at BH exhibited early stage symptoms of dark spots disease by 2017, but none had experienced associated tissue mortality (i.e., Fig. 2). All colonies of both species found at CR were visibly healthy and showed no evidence of disease throughout the sampling period.

Modeling results reinforce the observed differences in mortality and incidence of white plague–like symptoms between inner and outer reef sites. The risk of mortality was found to be significantly higher at outer reef sites (log odds 95% CI 3.21–9.32) and as year increased [log odds 95% CI 28.24–31.93 (2015–2016), 34.22–36.45 (2016–2017)], but did not differ significantly between species (log odds 95% CI –3.28 to 0.26). Likewise, logistic regression also indicated that the incidence of white plague–like symptoms was significantly higher at outer reef sites and as year increased. Estimates of effect sizes and the associated standard error of independent variables

from penalized regression models cannot be interpreted reliably due to the substantial bias introduced by the penalization procedure (Goeman 2010). However, because the cross-validation procedure removed species and regrowth from the best-fit model, we can conclude that neither factor had a significant effect on the occurrence of white plague–like symptoms.

Similar to our observations, FRRP surveys revealed an increase in overall coral disease frequency from 2014 to 2016 (Fig. 4). Note, because the large majority of disease observations in this data set were of dark spots disease (87.3%), the prevalence of disease at inner patch reef sites in the FRRP data reflects our observation of dark spots disease at the northern inner reef site (BH). Interestingly, FRRP surveys did not report significant incidence of white plague–related disease within our study region by 2016 (<3.1% at only two sites). According to anecdotal accounts, symptoms of the current outbreak had been observed as far south as Davis Reef (*see* Fig. 1) by January 2017 (FL DEP 2016); however, reports from the upper keys region at this time were inconsistent, so it is conceivable that FRRP sites had not yet been significantly impacted.

Coring operations did not influence disease incidence or mortality given the healthy status of most cored colonies in 2016 (1 yr post-coring, Fig. 3), and as verified by logistic modeling results. *Siderastrea siderea* regrew significantly faster than *P. strigosa* [0.068% (SE 0.01%) and 0.027% (SE 0.01%) d^{-1}, respectively; Table 1], an effect that was compounded by year of observation (2015–2016 or 2016–2017; Table 1). Notably, no significant differences were found between regrowth rates of inner and outer reef colonies [0.052% (SE 0.01%) and 0.043% (SE 0.01%) d^{-1}, respectively; Table 1], despite differences in disease incidence and mortality between reef zones (Fig. 3).

Preliminary evidence suggests that the onset of the current outbreak was likely associated with record high summer temperatures experienced across the FKRT in 2014, preceded by abnormally warm winter and summer seasons (Precht et al. 2016). Temperature has been shown to enhance the virulence of *Vibrio coralliilyticus* Ben-Haim et al., 2003, a ubiquitous bacterial pathogen known to cause fatal infections of corals and other marine organisms (Kimes et al. 2012). Furthermore, during the 2005 bleaching event on the FKRT, Brandt and McManus (2009) found that the frequency of white plague and dark spots disease was positively correlated with bleaching extent, and that black band disease incidence was highest during the period of warmest temperatures.

Importantly, environmental conditions vary considerably between inner and outer reef habitats, and there is evidence that such differences have implications on reef resilience to environmental stress (Kenkel et al. 2013). Inner patch reefs are generally exposed to higher dissolved nutrient concentrations, turbidity, and greater temperature variability than outer bank barrier reefs on the FKRT (Lirman and Fong 2007, Soto et al. 2011), which, rather counterintuitively, correlates with higher coral cover (Lirman and Fong 2007). Likewise, our analysis of temperature and ocean color conditions illustrates clear differences in temperature variability, mean chlorophyll-*a* and turbidity between the two inner and two outer reef sites examined in our study. We found that mean annual SST from 2003 to 2016 was similar between all sites [IR: 26.94 (SE 0.11) °C, OR: 26.96 (SE 0.10) °C], but mean annual SST range was significantly higher at inner reef sites [IR: 9.47 (SE 0.31) °C, OR: 8.90 (SE 0.33) °C]. Moreover, between 2012 and 2017, the two inner reef sites exhibited significantly higher levels

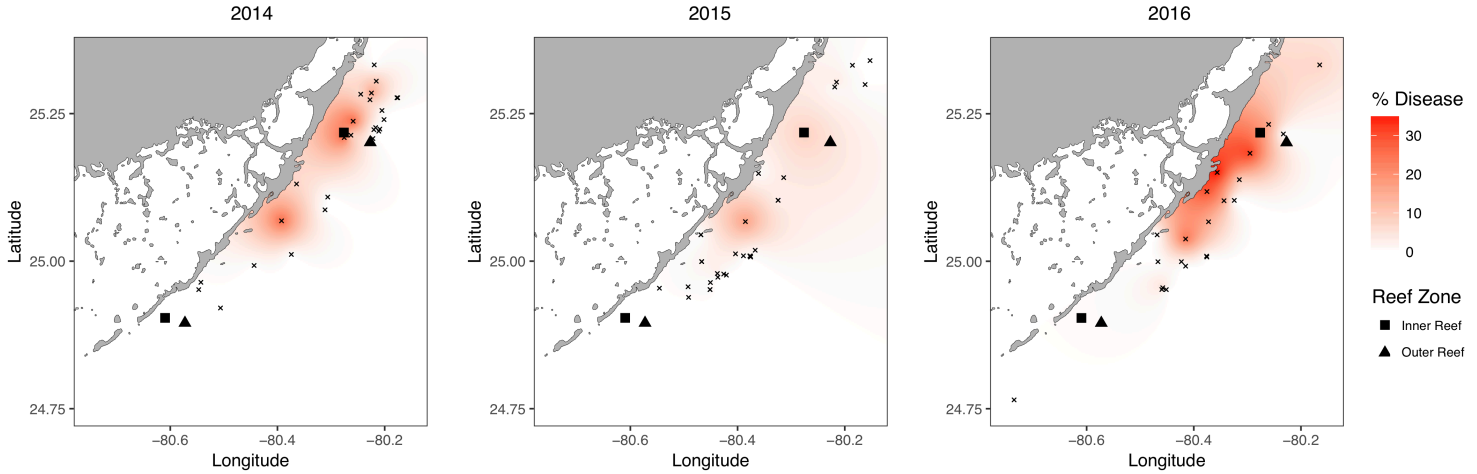

Figure 4. Disease frequency reported by Florida Reef Resilience Program (FRRP) survey teams from 2014 to 2016. Sites associated with the present study are depicted as in Figure 1. FRRP survey sites are depicted as ×. Color intensity reflects the total percentage of symptomatic corals (any disease) at each site, estimated across the seascape using spatial interpolation. Of all disease observations, 87.3% were of dark spots disease, 1.8% were of white plague–related disease (i.e., white plague, white blotch, and patchy necrosis), and 8.2% were of unknown disease.

Table 1. Tissue regrowth rates following coring operations. Regrowth rates are defined as the mean percent change in exposed surface area of the coring hole per day and are compared between species (*Pseudodiploria strigosa* or *Siderastrea siderea*), reef zones (inner or outer), and years (2015–2016 and 2016–2017) with a three-way analysis of variance. Asterisks indicate significant differences (*$P < 0.05$, **$P < 0.01$).

Variable	df	SS	*F*	*P*
Species	1	0.01920	7.632	0.0087**
Reef zone	1	0.00161	0.639	0.4289
Year	1	0.00852	3.389	0.0732
Species × reef zone	1	0.00235	0.934	0.3398
Species × year	1	0.01190	4.729	0.0358*
Reef zone × year	1	0.00002	0.007	0.9349
Residuals	39	0.09810		

of chlorophyll-*a* [IR: 2.02 (SE 0.09) mg m^{-3}, OR: 1.39 (SE 0.05) mg m^{-3}] and higher turbidity [IR: 0.27 (SE 0.02) m^{-1}, OR: 0.17 (SE 0.02) m^{-1}] than the outer reef sites.

Inner reef corals on the FKRT have demonstrated greater tolerance to experimental heat stress than their outer reef counterparts (Kenkel et al. 2013), and recent research has shown that high chlorophyll-*a* levels, typical of inner reef waters, are associated with lower prevalence of bleaching and disease during thermal stress events (van Woesik and McCaffrey 2017). Likewise, under the combined stress of heating and inoculation with a known disease-causing agent, outer reef colonies of *Porites astreoides* Lamarck, 1816 from the FKRT were found to exhibit a two- to four-fold increase in the expression of genes related to innate immune response (Haslun et al. 2018). Inner reef colonies exhibited lower levels of immune-related gene expression independent of treatment, suggesting greater resistance to biotic stress (Haslun et al. 2018). Consistent with these patterns, our findings of lower disease-associated mortality at inner reef sites suggest that the physiological or environmental factors underlying inner reef coral resilience to biotic and abiotic stress may also be buffering these areas from the more severe impacts of the current disease epidemic. However, better etiological understanding of this outbreak is necessary to substantiate this hypothesis.

It is also important to note that CR, one of the two inner reef sites (Fig. 1), has been identified as one of the most resilient patch reef communities on the FKRT. Manzello et al. (2015) found that *Orbicella faveolata* (Ellis and Solander, 1786) colonies at CR have significantly greater calcification rates than those at an adjacent outer reef site, and in a recent study, this coral community was found to experience only minimal mortality during repeat thermal stress events in 2014 and 2015 (Gintert et al. 2018). Thus, it is possible that the superior fitness of corals at this site under average and anomalous growing conditions may also be reducing their susceptibility to disease.

The present study adds to reports documenting the recent spread of coral disease throughout the FKRT. We found that corals living on outer reef sites have been more severely affected by disease than their inner reef counterparts, highlighting the potential role of habitat differences in protecting inner reef corals from the severity of this outbreak. However, it is critical to emphasize the spatial and temporal limits of our study, as these data was collected during the initial 3 yrs (2015–2017) of an ongoing disease event and from only two inner-outer reef transects on the FKRT. In fact, as of August 2018, symptoms of white syndrome have been observed at CR and at a nearby inner patch reef site adjacent to Hens and Chickens Reef, causing substantial

mortality in *Pseudodiploria* spp., *Colpophyllia natans* (Houttuyn, 1772), *Dichocoenia stokesii* Milne-Edwards and Haime, 1848, and *Montastraea cavernosa* (Linnaeus, 1767) (D Manzello, National Oceanic and Atmospheric Administration, pers comm). Thus, as monitoring efforts on the FKRT continue to elucidate the mechanisms underlying coral disease dynamics, it will be important to resolve whether the patterns observed here reflect a true disparity in resistance that has prevented or at least delayed the onset of disease or, rather, if it has arisen coincidentally during the course of the ongoing outbreak.

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