Host density and anthropogenic stress are drivers of variability in dark spot disease in Siderastrea siderea across the Florida Reef Tract

Aeby, Greta S.; Williams, Gareth J.; Whitall, David ; Davies, Andrew; Fromuth, Elizabeth; Walker, Brian K.

Bulletin of Marine Science

E-pub ahead of print: 27/06/2023

Peer reviewed version

Running title: Host density and anthropogenic stress drive patterns of dark spot disease

Title: Host density and anthropogenic stress are drivers of variability in dark spot disease in *Siderastrea siderea* across the Florida Reef Tract

Greta S. Aeby[1*], Gareth J. Williams[2], David R. Whitall[3], Andrew J. Davies[4,5], Elizabeth Fromuth[6], Brian K. Walker[6]

[1] *Smithsonian Marine Station, 701 Seaway Dr, Fort Pierce, Florida, 34949, United States*

[2] *School of Ocean Sciences, Bangor University, Askew St., Menai Bridge, Anglesey, LL59 5AB, United Kingdom*

[3]*National Centers for Coastal Ocean Science, National Ocean Service, NOAA, 1305 East-West Hwy, Silver Spring, Maryland 20910, United States*

[4]*Department of Biological Sciences, University of Rhode Island, 120 Flagg Rd, Kingston, Rhode Island, 02881, United States*

[5]*University of Rhode Island, Graduate School of Oceanography, 215 South Ferry Road, Narragansett, Rhode Island 02882, United States*

[6]*Halmos College of Arts and Sciences, Nova Southeastern University, 8000 N. Ocean Dr, Dania Beach, Florida 33004, United States*

**Corresponding author email: <greta@hawaii.edu>*

ABSTRACT

Dark spot disease (DSD) was first reported within Florida's coral reefs in the 1990s but factors affecting its spatial distribution have not been well studied. We used a 14-year (2005-2019) coral monitoring data set, utilizing 2,242 surveys collected along Florida's coral reefs (~530 linear km) to explore the spatial and temporal patterns of DSD occurrence. We built predictive statistical models to test for correlations between a suite of environmental and human impact factors and the occurrence of DSD in the reef coral, *Siderastrea siderea*. DSD in *S. siderea* is a chronic disease which occurred in all 14 years of the study. Annual DSD prevalence ranged from 0.45% to 4.4% and the proportion of survey sites that had DSD ranged from 4.8% to 30.9%. During the study period, DSD became more widespread across Florida's coral reefs and affected a higher proportion of *S. siderea* populations. Spatial variations in DSD correlated with environmental and human factors which together explained 64.4% of the underlying variability. The most influential factors were concentration of silica in the surface waters (a proxy for freshwater input), the total number of coral hosts, and distance to septic areas. DSD occurred in all regions, but the highest cumulative prevalence occurred in the upper Keys on reefs around major urban centers with links to coastal water discharges. Our results support the hypothesis that coastal water quality is a key component of DSD disease dynamics in Florida and provides motivation for addressing land-sea connections to ameliorate disease occurrence in the region.

INTRODUCTION

Disease is a normal component of host populations, but increased levels of disease can indicate changes in host-pathogen ecology often due to shifting environmental conditions (Schrag and Wiener 1995, Daszak et al. 2000). Climate change and environmental degradation from local human impacts are cited as major influences contributing to disease outbreaks in wildlife populations (Daszak et al. 2000, Dobson and Foufopoulos 2001, Lafferty and Holt 2003) including reef corals (Harvell et al. 1999, 2007, Carpenter et al. 2008, Maynard et al. 2015). Diseases in corals have become a problem worldwide with outbreaks reported from the Indo-Pacific (Willis et al. 2004, Myers and Raymundo 2009, Aeby et al. 2011a, 2015, 2016, Haapkylä et al. 2011), Persian Gulf (Howells et al. 2020, Aeby et al. 2020), Indian Ocean (Thinesh et al. 2009, Raj et al. 2016) and western Atlantic (Patterson et al. 2002, Miller et al. 2009, Voss and Richardson 2006, Croquer and Weil 2009, Brandt et al. 2012, Croquer et al. 2021). The most devastating coral disease in recent history, stony coral tissue loss disease, emerged in Florida in 2014 (Precht et al. 2016) and continues to spread across the Caribbean, causing significant mortality on affected coral reefs (Walton et al. 2018, Alvarez-Filip et al. 2019, Sharp et al. 2020, Heres et al. 2021, Estrada-Saldivar et al. 2021). Coral disease is a major threat to the survival of coral reefs and identifying the primary factors affecting coral disease dynamics is key to designing effective local mitigation strategies, prioritizing disease intervention resources, and identifying areas suitable for reef restoration.

Environmental factors can affect host-pathogen dynamics by impairing host immune responses and enhancing pathogen abundance, transmission, or virulence (Daszak et al. 2001, Dobson and Foufopoulos 2001, Harvell et al. 2007) and several environmental conditions have emerged as important in driving increased disease levels in corals. Thermal stress has been linked to numerous disease outbreaks (Bruno et al. 2007, Miller et al. 2009, Bruno 2015,

Brodnicke et al. 2019, Howells et al. 2020) with higher ocean temperatures thought to impair coral immune responses as well as enhance pathogen abundance and virulence (Mydlarz et al. 2009, Maynard et al. 2015, Ushijima et al. 2016, Vega Thurber et al. 2020). Lamb et al. (2018) found that disease establishment was facilitated via abrasion of corals by plastics, allowing pathogen invasion, reducing coral resources for immune function due to wound healing and by the colonization of plastics by pathogens. Poor water quality due to dredging (Pollock et al. 2014), land-based runoff (Haapkylä et al. 2011, Sheridan et al. 2014, Aeby et al. 2016) and eutrophication (Bruno et al. 2003, Vega Thurber et al. 2014) have also been implicated in increasing disease prevalence. A common component of all these studies is the link between increases in coral disease and changes in environmental conditions due to anthropogenic actions.

Dark spot disease (DSD), also called dark spot syndrome (DSS), is a disease reported on coral reefs across the wider Caribbean (23 countries) affecting 16 coral species (Work and Weil 2016). DSD manifests as multi-focal or coalescing spots of dark discolored tissue (Fig. 1) that can spread across the coral surface causing slow, progressive tissue loss (Work and Weil 2016). DSD lesions can also resolve and appear elsewhere on the colony (Borger et al. 2005, Gochfeld et al. 2006, Porter et al. 2011) and it is unclear what determines the trajectory of these lesions. Corals with similar disease signs from the Indo-Pacific (Work et al. 2008, 2014), and Red Sea (Aeby et al. 2021) are termed ‘endolithic hypermycosis’ as histopathology showed the lesions to be associated with endolithic fungal infections (Work et al. 2008, 2014, Aeby et al. 2021). In the Caribbean, the etiology of DSD is less certain, although Renegar et al. (2008) also found endolithic fungal infections in DSD-affected *Siderastrea siderea.* Cervino et al. (2001) showed that zooxanthellae from dark spot lesions are swollen and darker in pigment and suggested DSD was primarily a disease of the symbiotic zooxanthellae found within the coral host. The lesions

identified as DSD may have multiple underlying etiologies, as proposed for tissue loss diseases (white syndromes) (Bourne et al. 2014) or may instead be a general stress response, as suggested by Borger et al. (2005). Although DSD affects multiple coral species on coral reefs across the Caribbean, it is not well studied (Work and Weil 2016).

Dark spot disease (DSD) has been affecting Florida's corals since the 1990s (Santavy et al. 2001) but little is known about the ecology of DSD in this region. We used a 14-year (2005-2019) coral disease monitoring data set, collected along Florida's extensive Coral Reefs Tract (~530 linear km), to explore DSD occurrence along a gradient of human and environmental factors. Specifically, we examined the spatial and temporal patterns of DSD prevalence (percent of corals affected) and frequency of occurrence among survey sites in the common coral host, *Siderastrea siderea*, and used statistical modeling to explore some of the underlying co-factors that correlate with DSD occurrence. This is the first regional-scale investigation of DSD patterns along the Florida Reef Tract.

METHODS

Study area

The Florida Reef Tract is a predominantly continental reef system which spans approximately 595 linear km of coastline from the Dry Tortugas National Park in the southwest to Martin County in the northeast (Walker et al. 2013). It is a large barrier reef ecosystem composed of over 45 species of stony corals, and is a critical ecosystem that provides coastal protection, supports a large tourism industry, and hosts a wide variety of commercially and ecologically important marine species.

Disease surveys

We used data collected as part of the Florida Reef Resilience Program's (FRRP) Disturbance Response Monitoring program (DRM)(https://frrp.org/coral-monitoring/). The DRM uses a stratified random sample design across 28 discrete reef zones in 10 sub-regions. At each site, two independent 1x10 m belt transects were surveyed with all coral colonies with maximum diameter >4 cm identified to species, enumerated and their health condition noted. The data are available from Florida Fish and Wildlife Conservation Commission (https://ocean.floridamarine.org/FRRP/). For our analysis, we used a subset of the data, limiting it to the coral species, *Siderastrea siderea*, and the health condition noted as dark spot disease (DSD). *Siderastrea siderea* is a widespread species occurring across the Caribbean and is the most common host of DSD (Gil-Agudelo et al. 2001, Weil 2004, Borger 2005, Gochfeld et al. 2006, Voss and Richardson 2006). The dataset used in our analyses included 2,442 individual surveys from 2005-2019, excluding 2017 due to low and inconsistent sampling effort that year after hurricane Irma (Fig. 2). From these data, we calculated DSD prevalence, defined as the percent of colonies surveyed at each site that had a lesion consistent with DSD. Overall prevalence was the proportion of all colonies that had a DSD lesion (all surveys combined). Frequency of disease occurrence (FOC) is a measure of spatial distribution of disease and was calculated as the percentage of survey sites having one or more coral colonies manifesting DSD. There was a large increase in DSD prevalence and FOC between the time periods 2005-2012 and 2013-2019. Thus, differences in DSD prevalence and FOC between these two time periods were analyzed using a non-parametric Wilcoxon 2-group test (JMP vers. 16.1). Regional patterns of DSD prevalence were explored with the data split by ecoregions (Walker 2012). Differences in DSD prevalence among ecoregions was examined by calculating the cumulative average DSD prevalence for surveys between 2005 and 2019 (excluding 2017).

Modeling the association between DSD and multiple human and environmental factors

Several predictor variables were hypothesized to be linked to spatial variations in coral disease dynamics, including 1) The Nature Conservancy's (TNC) Ocean Wealth Index (as a proxy of reef "use" by people), 2) impacts from wastewater, 3) septic tanks (potential sewage pollution), 4) local land use (as indicators of local coastal development), 5) water quality, and 6) local human populations. These were quantified using multiple data sources and across a range of scales and combined with measurements of survey depth, susceptible coral host abundance (i.e., the abundance of *Siderastrea siderea* at each site), survey year (to account for the temporally variable nature of the disease data), and a range of metrics that captured variations in reef habitat (Table 1). For our predictor groups 1– 6 above, we quantified each of them within a radial distance of each survey location over a range of spatial scales using a Fibonacci sequence (1, 2, 3, 5, 8, 13 and 21 km). We examined different spatial scales for predictor variables as the influence each predictor might have on our disease response variable could vary depending on their distance from the survey locations. In some cases, there was a high proportion of missing data values over the smaller scales for some predictor variables (e.g., 1 – 8km for water quality estimates and wastewater treatment facilities) so these scales were excluded prior to model fitting (see Table 1 for the final scales included for each predictor). Despite our knowledge that some of the predictor variables were likely collinear, given the large replication in the response variable, and the fact that our chosen modeling framework (see below) is robust to the inclusion of spurious predictors, we included all predictors in the model fitting process.

Statistical models were built using a boosted regression tree (BRT) framework to test the ability of the predictor variables to explain variation in the number of diseased coral hosts across

the Florida Reef Tract. Unlike many modeling techniques that aim to fit a single parsimonious model, BRT incorporates machine learning decision tree methods (Breiman et al. 1984) and boosting, a method to reduce predictive error (Elith et al. 2008), to build an additive regression model in which individual terms are regression trees, fitted in a forward stage-wise manner (i.e., sequentially fitting each new tree to the residuals from the previous ones). In summary, BRT gives two crucial pieces of information, namely the underlying relationship between the response and each predictor, and the strongest statistical predictor (among the simultaneously tested predictors) of the response variable in question. Due to their flexible use and improved predictive power, the use of BRTs has increased over recent years to model non-linear ecological relationships at a range of spatial scales including coral reef-environment associations (e.g., Williams et al. 2010, Gove et al. 2015, Aston et al. 2019).

BRTs were constructed using the *gbm.step* routine (Elith et al. 2008) in the *dismo* package (Hijmans et al. 2017) for R (www.r-project.org) and all model outputs were visualized in *ggplot2* using *ggBRT* (Jouffray et al. 2019). The data were modeled using a Poisson distribution, as our response variable was the number of corals in each survey location showing signs of DSD (i.e., number of disease cases). Number of disease cases was used rather than disease prevalence (# cases/total # colonies surveyed) as host abundance was already included in the model. We used a 10-fold cross-validation approach to test the model against withheld portions of the data (iterated thousands of times) and the cross-validated percentage deviance explained, calculated as (1 – (cross-validated deviance/mean total deviance)), as our measure of model performance (Jouffray et al. 2019). To optimize model predictive performance, we varied three core parameters of the BRT algorithm: the *bag-fraction* (bf, proportion of data to be selected at each step), the *learning rate* (lr, used to shrink the contribution of each tree as it is added to the

model), and the *tree complexity* (tc the number of terminal nodes in a tree). Using a customized loop routine (Richards et al. 2012), we identified the combination of these three parameters that resulted in the lowest cross-validation deviance (CVD) over bf-values 0.5, 0.7, and 0.8, lr-values 0.001, 0.0001, and 0.00001, and tc-values 1–5, while maintaining a minimum of ≥ 1000 fitted trees and a maximum of 50,000 trees. This identified the following optimal model parameter settings: bf = 0.8, lr = 0.001, tc = 2. We calculated the relative importance of each predictor based on the number of times a variable was selected for splitting, weighted by the squared improvement to the model as a result of each split, and averaged over all trees (Friedman and Meulman 2003, Elith et al. 2008). To assess the relative contribution of each predictor, we only considered predictors with a relative influence above that expected by chance (100/number of variables) (Muller et al. 2013) and then rescaled their influence to 100%. The interactive effect of the most influential predictors was then visualized using the *ggInteract_3D* function within *ggBRT*. All input data (DSD.csv) and R code (DSD_Cases_BRT_Florida.R) used to execute our BRT models are provided as Supplementary Materials.

RESULTS

DSD prevalence through time within S. siderea populations across the Florida Reef Tract

Between 2005 and 2019, mean (±1SE) DSD prevalence among all sites and all years was 2% (± 0.4%). DSD was observed in every year of the survey period (14 years) and mean DSD prevalence ranged from a low of 0.45% in 2006 to a high of 4.4% in 2016. Overall, DSD prevalence (all sites combined) increased through time, with mean DSD prevalence before 2013 (2005-2012) equaling 1.1% (±0.17%) compared to 2.9% (±0.27%) from 2013-2019 (Fig. 3) (Wilcoxon 2-group test, Z=2.9, p=0.0037).

Distribution of DSD through time across the Florida Reef Tract

Region-wide distribution of DSD within our study period was 14.6% with 357 of the 2,442 survey sites having colonies with DSD signs (all years combined). Frequency of occurrence ranged from a low of 4.8% (n=84 sites) in 2005 to a high of 30.9% (n=188 sites) in 2018 (Fig. 4). Through time, DSD occurrence increased from 7.4% ($\pm$0.8%) of survey sites between 2005 and 2012 to an average of 22.1% ($\pm$2.8%) of survey sites between 2013-2019 (Wilcoxon 2-group test, Z=2.9, p=0.0037).

Association between DSD and suspected human and environmental drivers

Our predictive model explained 64.4% of the underlying spatial deviance in the number of DSD cases across the entire dataset (Table 2). The top three predictors contributed 45.5% of the relative influence within the model and included the concentration of silica in the surface waters within a 13 km radius (proxy for freshwater input), the total number of susceptible coral hosts (*S. siderea*), and the number of septic areas within an 8 km radius. When predicting to new data from the training data, 47.1% of the cross-validated percentage deviance was explained, suggesting a reasonable ability to predict to new data. The probability of DSD occurrence increased as both surface water silica concentration and coral host abundances increased and became maximized where silica exceeded 1.15 µM/L and where there were >100 coral hosts within the survey area (Fig. 5A). DSD occurrence was also higher in areas with >13 km^2 septic areas within 8 km regardless of coral host abundance but was highest where there were also >100 coral hosts (Fig. 5B).

Other significant variables in the model were year of the study (consistent with our findings of increased DSD levels through time), survey depth, habitat characterization (URM ClassLv4, Reef Zone)(https://myfwc.com/research/gis/regional-projects/unified-reef-map/), measures of water quality, total carbon in surface waters within 8 km and in bottom waters within 21 km, and total phosphorous in bottom waters within 21 km, and surface waters within 8 km, distance to pollution sources (number of sewer systems within 21 km and number of septic systems within 8 km) and variables associated with human impacts (amount of spending within 1 and 5 km which indicates degree of human presence and degree of urbanization within 8 km which could influence coastal runoff and pollution).

Regional distribution of DSD and the association with predictor variables across the Florida Reef Tract

S. siderea colonies with DSD were found throughout the Florida Reef Tract but there was a distinct spatial clustering of DSD colonies on reefs around the upper Keys (Fig. 6) where there was a threshold concentration of silica in the water and a threshold area of septic systems within 8 km of the survey sites (Fig. 7).

DISCUSSION

In the first long-term study examining dark spot disease (DSD) occurrence across the Florida Reef Tract, DSD in *Siderastrea siderea* was found to be a chronic disease occurring in the annual monitoring surveys in all 14 years of the study. Between 2005 and 2019, *S. siderea* DSD prevalence ranged from less than 0.5% to over 4% which is less than from prior studies in Florida and regions in the Caribbean. In Florida, DSD prevalence in *S. siderea* was found to be

between 4.3% to 13.3% (Porter et al. 2011), and in the Caribbean reported studies found 25% in Columbia (Gil-Agudelo & Garzon-Ferreira 2001), 53% in Bonaire, 58% in Turks and Caicos, and 42% in Grenada (Cervino et al. 2001). We only found an average DSD prevalence of 2% (all years combined) but the spatial and temporal coverage from the number of surveys we examined (2508 surveys across 15 years) far exceeded the spatial and temporal scale of other studies which might explain these differences. Indeed, we did find higher DSD prevalence at smaller spatial and temporal scales. As example, in the upper Florida Keys, DSD prevalence was 19.4% in 2014 and 22% in 2016. In addition, human and environmental variables are also important factors underlying disease occurrence which also vary in time and space.

DSD became more widespread across Florida's coral reefs and affected a higher proportion of *S. siderea* populations through time. DSD was found in 7% of the total surveys between 2005 and 2012 but occurred in 22% of the surveys between 2013 and 2019. Similarly, average DSD prevalence was approximately 1% of the surveyed corals from 2005 to 2012 but affected nearly 3% of *S. siderea* colonies thereafter. As with most coral diseases, pinpointing a cause to this increase in DSD occurrence through time is challenging. We ruled out changes in methods or field personnel and there were no obvious acute environmental events (bleaching, hurricanes, annual rainfall) that could explain changing disease levels. Previous studies have shown that DSD is dynamic, with colonies recovering and becoming reinfected over time (Gochfeld et al. 2006, Porter et al. 2011). However, our data indicate a persistent and increasing annual DSD prevalence from 2012 – 2019, indicative of a chronic source or stressor. Florida's coral reefs have been increasingly impacted by humans with densely populated coastlines, higher visitor numbers, and chronic polluted terrestrial run-off for many decades (Jackson et al. 2014). Florida has also suffered an acceleration of both acute and chronic thermal stress over the past 30

years with reefs experiencing six mass bleaching events since the late 1980s (Manzello 2015) with the most recent event occurring in 2014 (Eakin et al. 2019, van Woesik and McCaffrey 2017). It is plausible that Florida's coral reefs have reached the limit of their ability to tolerate multiple, possibly additive or synergistic stressors which may be affecting the coral's ability to resist disease.

Statistical modeling found host density, indicators of freshwater input, and proximity to septic systems were the strongest predictors of DSD, suggesting that environmental and anthropogenic stress are important drivers of this disease across the Florida Reef Tract. It must be noted that other factors may be important in determining DSD levels in regions where pollution is less problematic. Identifying the cause of DSD will help in teasing out all relevant environmental co-factors. However, for Florida's reefs polluted waters have been identified as a major problem for decades (Zhao et al. 2013). Florida's long history of coastal pollution has resulted in harmful algal blooms, seagrass die-offs, and declining coral reefs, with wastewater and on-site sewage treatment and disposal systems (OSTDS) representing major nitrogen sources contributing to ecosystem impacts (Lapointe et al. 2004, 2015). In addition to excess nitrogen, sewage pollution also results in coastal influxes of freshwater, phosphorous, pathogens, endocrine disrupters, suspended solids, sediments, heavy metals, and other toxicants (Wear and Vega Thurber 2015). Evidence of sewage pollution is common in Florida's nearshore environments with human fecal contamination of nearshore or offshore coral reefs found along the Florida Reef Tract (Lipp et al. 2002, Futch et al. 2010, 2011, Staley et al. 2017). Sewage can also act as a reservoir for potential pathogens as evidenced by Sutherland et al. (2011) who found human wastewater to be a source of the pathogen causing white pox disease in the reef coral, *Acropora palmata*. Additionally, freshwater stress alone can increase a coral's susceptibility to

bacterial infections (Shore-Maggio et al. 2018) and numerous field studies show a link between rainfall, terrestrial run-off, and disease outbreaks (Haapkylä et al. 2011, Sheridan et al. 2014, Aeby et al. 2016).

It is not usually a single stressor that results in the emergence of disease but multiple co-factors. Coastal runoff exposes nearshore coral reefs to sediments, freshwater, excess nutrients, and other pollutants. Other studies support the importance of multiple stressors impacting coral reefs. Oberle et al. (2019) found a combination of nutrient loading and low salinities from groundwater discharge explained an outbreak of black band disease on coral reefs in Hawaii. Considering the long-term problems Florida has had with polluted waters, it is not surprising that we found a link between DSD levels, proximity to sewage sources and indicators of land-based freshwater influx. Similarly, Porter et al. (2001) showed a five-fold increase in number of survey stations with disease over time in the Florida Keys and this trend was especially evident on reefs close to the population centers of Key West and Key Largo. In additional support of our findings, Vega Thurber et al. (2014) showed a direct link between excess nitrogen and DSD development in *S. siderea.* In an *in situ* nutrient enrichment experiment, they found *S. siderea* in treatment plots, had a 100% increase in DSD compared to corals in control plots.

Spatial analyses among regions showed that the progressively higher DSD prevalence through time, was especially prominent in the upper Florida Keys. The upper Florida Keys were also associated with higher levels of silica indicating an influx of freshwater, and larger septic areas, two of the top predictor variables explaining DSD occurrence along the Florida Reef Tract. Several changes in local water management have occurred around the upper Keys since 2012 that might have influenced the amount of freshwater, and thus levels of DSD, occurring in that region, namely the development of wastewater management systems, and changes in the

management of the Everglades canal system. Wastewater is recognized as a historic source of excess nutrients into local Florida waters (Barreras et al. 2019) and billions of dollars have been spent developing wastewater management systems to convert septic systems to sewer. In the upper Keys, there are two water management districts, Key Largo Wastewater Treatment District (KLWTD) and the North Key Largo Utility Corporation (Barreras et al 2019). The KLWTD advanced wastewater treatment system was completed in 2010 and effluent from this system is deep-well injected to reduce pollution in nearshore marine waters (Reich et al. 2002, Chanton et al. 2003). Implementation of these systems has shown improvements in water local quality (Barreras et al. 2019, Kelly et al. 2021) so wastewater seems a less likely cause of increased disease within this region. However, deep-well injected wastewater on the island of Maui in Hawaii was found to be a significant source of eutrophication on adjacent coral reefs, contributing to their decline (Dailer et al. 2010, Murray et al. 2019).

Conversely, freshwater from the heavily managed and controlled Everglades canal system has been shown to affect much of the Florida Keys coral reef system (Lapointe et al 2019). This water contains runoff from both urban and agriculture areas that can result in 'black water' events where patches of dark colored water develop due to high concentrations of phytoplankton and colored dissolved organic matter (Hu 2004, Zhao et al. 2013). Lapointe et al. (2019) implicate this water as the main source of enriched nutrients to the reef system and a primary driver of coral reef decline in the lower Florida Keys over the past few decades. Given that increasing the freshwater flows from the Everglades has been a priority for managers for decades (NOAA 1996), it is also possible that these flows are increasing DSD. Clearly, more research is needed to understand the source and dynamics of DSD in the Florida Keys.

Host density was also a significant factor in explaining DSD occurrence across the Florida Reef Tract. A positive relationship between host abundance and disease has been demonstrated for numerous host-pathogen systems (Altizer and Augustine 1997, McCallum et al. 2004, Lafferty 2004) including DSD (Borger and Steiner 2005) and other coral diseases (Bruno et al. 2007, Haapkylä et al. 2009, Aeby et al. 2010). High host density can result in increased transmission of disease throughout a population as distance between individuals decreases. However, this would only apply toward infectious diseases, and it is not known whether DSD is infectious. DSD is not well studied, but to date, there is no evidence that DSD is infectious (Randall et al. 2016). However, if DSD were an environmentally induced disease, then host density could still play a role in predicting disease occurrence (e.g., more corals exposed to the adverse environmental conditions would lead to higher local disease levels).

On the Florida Reef Tract, *S. siderea* densities have been increasing through time (Hayes et al. 2022), and it is thought that weedy species, such as *S. siderea*, are becoming more common due to the declines of historically dominant species (Burman et al. 2012, Jones et al. 2020). The species that have declined the most were also the most stenotopic species, i.e., those able to tolerate a narrower range of environmental conditions (Burman et al. 2012). Increasing thermal stress events combined with multiple disease outbreaks have led to this shift in coral community structure (Burman et al. 2012, Jones et al. 2020). Understanding disease in *S. siderea* is becoming even more important as its numerical dominance increases throughout the Florida Reef Tract.

Several studies have suggested DSD is caused by the overgrowth of endolithic fungi (Work et al. 2008, 2014, Renegar et al. 2008) and it is plausible that DSD is an environmentally induced disease whereby environmental stressors are disrupting the coral holobiont facilitating

fungal overgrowth. If so, the environmental stressors and thus underlying drivers of DSD may also differ between regions explaining why pollution may be important in driving DSD on Florida's reefs but perhaps not in other regions. Corals live in a dynamic partnership between the cnidarian host, its endosymbiotic dinoflagellates, and a suite of microbiota that includes archaea, bacteria, viruses, fungi, and endolithic algae (Rosenberg et al. 2007, Bourne et al. 2009, Boilard et al. 2020). Environmental conditions that disrupt the delicate balance among partners create imbalances or dysbiosis which can result in disease (Lesser et al. 2007, Bourne et al. 2009, Vega Thurber et al. 2020). MacKnight et al. (2021) examined the link between microbial dysbiosis and disease susceptibility of seven Caribbean coral species exposed to white plague disease. They found that coral species that showed comparatively higher microbial dysbiosis had the greatest susceptibility to infection upon exposure to white plague. Diseases in marine organisms are more often due to dysbiosis and the emergence of opportunistic pathogens rather than infection by a single pathogen (Lesser et al. 2007, Burge 2014, Egan et al. 2014. Egan and Gardner 2016). In other cases, pathogens can live as a component of a host's normal microbiome but under certain conditions switch from a commensal to a pathogen. For example, the marine bacterium *Phaeobacter gallaeciensis* BS107 is a commensal on the microalgae, *Emiliania huxleyi*, and produces compounds that are beneficial to its algal host (Seyedsayamdost et al. 2011). However, in response to p-coumaric acid, a breakdown product symptomatic of aging algae, *Phaeobacter gallaeciensis* BS107 initiates production of potent algaecides becoming an opportunistic pathogen of its algal host. Whether or not DSD is caused by a disruption of the symbiosis between the coral host and its fungal endoliths will require further research on the ecology and etiology of this disease.

Interestingly, human population densities were not significant in explaining DSD occurrence unlike studies of other coral diseases (e.g., growth anomalies: Aeby et al. 2011b) where human populations were associated with disease. Instead, the amount of spending was an important factor reflecting Florida's resident population as well as its extensive visitor numbers. Florida's resident population is around 22 million (https://www.populationu.com/us/florida-population) whereas it hosted 122 million visitors in 2021 (https://www.visitflorida.org/resources/research/). For Florida, this metric (spending) appears to be a better indicator of potential degree of human impacts rather than simply resident human population. Other important variables identified in the model (measures of water quality, distance to pollution sources, degree of urbanization that influence coastal runoff and pollution) confirm anthropogenic impacts as a major influence on coral disease occurrence.

Florida's coral reefs have declined over the past several decades with increased coastal populations, high visitor numbers, overfishing, and impaired water quality contributing to their decline. Florida is also considered a "coral disease hotspot" with disease outbreaks occurring repeatedly since the 1970s (Dustan 1977, Richardson 1998, Green and Bruckner 2000, Precht et al. 2016, van Woesik and McCaffrey 2017). The downward trend of Florida's coral reefs continues with a recent study on southeast Florida coral populations by Walton et al. (2018) who found region-wide declines in coral diversity, density and live tissue area which was attributed to disease. Our study documents the long-term persistence of disease in *S. siderea*, a common reef coral across Florida, and our modeling results suggest that coastal water quality is a key component to coral reef disease management, including maximizing resiliency in the face of global climate change. Disease not only impact host populations but can also result in ecosystem-wide impacts due to mortality of keystone species (Burge et al. 2014). Diseases are

predicted to increase with global climate change and anthropogenic pressures (Gattuso et al. 2015, Maynard et al. 2015), placing more emphasis on the need to further understand drivers of marine diseases.

ACKNOWLEDGEMENTS

We thank the Florida Department of Environmental Protection's Office of Resilience and Coastal Protection and to NOAA's National Centers for Coastal Ocean Science for supporting these efforts. Thank you to J Stein and the Florida Fish and Wildlife Conservation Commission Fish and Wildlife Research Institute's Florida Reef Resilience Program Disturbance Response Monitoring for facilitating data requests and advice. Thanks to J Maynard and D Tracey at SymbioSeas for help with data analysis and GIS methods development. Funding was provided by the Florida Department of Environmental Protection Award No. B7B6F3. The manuscript was greatly improved by comments from C. Woodley, E. Davenport and J. Guyon. The views, statements, findings, conclusions, and recommendations expressed herein are those of the authors and do not necessarily reflect the views of the State of Florida or any of its sub-agencies.

LITERATURE CITED

Aeby GS, Ross M, Williams GJ, Lewis TD, Work TM. 2010. Disease dynamics of *Montipora* white syndrome within Kaneohe Bay, Oahu, Hawaii: distribution, seasonality, virulence, and transmissibility. Dis Aquat Org 91:1–8

Aeby GS, Bourne DG, Wilson B, Work TM. 2011a. Coral diversity and the severity of disease outbreaks: A cross-regional comparison of *Acropora* white syndrome in a species-rich

region (American Samoa) with a species-poor region (Northwestern Hawaiian Islands). J Mar Biol 2011:1–8

Aeby GS, Williams GJ, Franklin EC, Haapkyla J, Harvell CD, Neale S, Page CA, Raymundo L, Vargas-Ángel B, Willis BL, et al. 2011b. Growth anomalies on the coral genera *Acropora* and *Porites* are strongly associated with host density and human population size across the Indo-Pacific. PLoS ONE 6:e16887

Aeby GS, Work TM, Runyon CM, Shore-Maggio A, Ushijima B, Videau P, Beurmann S, Callahan SM. 2015. First record of black band disease in the Hawaiian archipelago: response, outbreak status, virulence, and a method of treatment. PLoS ONE 10:e0120853

Aeby GS, Callahan S, Cox E, Runyon C, Smith A, Stanton F, Ushijima B, Work T. 2016. Emerging coral diseases in Kāneʻohe Bay, Oʻahu, Hawaiʻi (USA): two major disease outbreaks of acute *Montipora* white syndrome. Dis Aquat Org 119:189–198

Aeby GS, Howells E, Work T, Abrego D, Williams GJ, Wedding LM, Caldwell JM, Moritsch M, Burt JA. 2020. Localized outbreaks of coral disease on Arabian reefs are linked to extreme temperatures and environmental stressors. Coral Reefs 39:829–846

Aeby GS, Shore A, Jensen T, Ziegler M, Work T, Voolstra CR. 2021. A comparative baseline of coral disease in three regions along the Saudi Arabian coast of the central Red Sea. PLoS ONE 16:e0246854

Altizer SM, Augustine DJ. 1997. Interactions between frequency–dependent and vertical transmission in host–parasite systems. Proc R Soc Lond B 264:807–814

Alvarez-Filip L, Estrada-Saldívar N, Pérez-Cervantes E, Molina-Hernández A, González-Barrios FJ. 2019. A rapid spread of the stony coral tissue loss disease outbreak in the Mexican Caribbean. PeerJ 7:e8069

Aston E, Williams GJ, Green J, Davies A, Wedding L, Gove J, Jouffray J, Jones T, Clark J. 2019. Scale-dependent spatial patterns in benthic communities around a tropical island seascape. Ecography 42:578–590

Barreras H, Kelly EA, Kumar N, Solo-Gabriele HM. 2019. Assessment of local and regional strategies to control bacteria levels at beaches with consideration of impacts from climate change. Mar Pollut Bull 138:249–259

Boilard A, Dubé CE, Gruet C, Mercière A, Hernandez-Agreda A, Derome N. 2020. Defining coral bleaching as a microbial dysbiosis within the coral holobiont. Microorg 8:1682

Borger JL. 2005. Dark spot syndrome: a scleractinian coral disease or a general stress response? Coral Reefs 24:139–144

Bourne DG, Ainsworth TD, Pollock FJ, Willis BL. 2014. Towards a better understanding of white syndromes and their causes on Indo-Pacific coral reefs. Coral Reefs 34:233–242

Bourne DG, Garren M, Work TM, Rosenberg E, Smith GW, Harvell CD. 2009. Microbial disease and the coral holobiont. Trends Microbiol 17:554–562

Brandt ME, Ruttenberg BI, Waara R, Miller J, Witcher B, Estep AJ, Patterson M. 2012. Dynamics of an acute coral disease outbreak associated with the macroalgae *Dictyota* spp. in Dry Tortugas National Park, Florida, USA. Bull Mar Sci 88:1035–1050

Breiman L, Friedman, J, Olshen A, Stone C. 1984. Classification and Regression Trees. Belmont, CA: Wadsworth. International Group 432:151-166

Brodnicke OB, Bourne DG, Heron SF, Pears RJ, Stella JS, Smith HA, Willis BL. 2019. Unravelling the links between heat stress, bleaching and disease: fate of tabular corals following a combined disease and bleaching event. Coral Reefs 38:591–603

Bruno JF. 2015. The coral disease triangle. Nature Clim Change 5:302–303

Bruno JF, Petes LE, Drew Harvell C, Hettinger A. 2003. Nutrient enrichment can increase the severity of coral diseases. Ecol Letters 6:1056–1061

Bruno JF, Selig ER, Casey KS, Page CA, Willis BL, Harvell CD, Sweatman H, Melendy AM. 2007. Thermal stress and coral cover as drivers of coral disease outbreaks. PLoS Biol 5:e124

Burge CA, Eakin M, Friedman CS, Froelich B, Hershberger PK, Hofmann EE, Petes LE, Prager KC, Weil E, Willis BL, et al. 2014. Climate change influences on marine infectious diseases: implications for management and society. Annu Rev Mar Sci 6:249–277

Burman S, Aronson R, van Woesik, R. 2012. Biotic homogenization of coral assemblages along the Florida reef tract. Mar Ecol Prog Ser, 467, 89-96. doi:10.3354/meps09950

Carpenter KE, Abrar M, Aeby G, Aronson RB, Banks S, Bruckner A, Chiriboga A, Cortés J, Delbeek JC, DeVantier L, et al. 2008. One-third of reef-building corals face elevated extinction risk from climate change and local impacts. Science 321:560–563

Cervino J, Goreau TJ, Nagelkerken I, Smith GW, Hayes R. 2001. Yellow band and dark spot syndromes in Caribbean corals: distribution, rate of spread, cytology, and effects on abundance and division rate of zooxanthellae. *In:* Porter JW, editor. The Ecology and Etiology of Newly Emerging Marine Diseases. Springer Netherlands, Dordrecht, p. 53–63

Chanton J P, Burnett, WC, Dulaiova H, Corbett, DR, Taniguchi M. 2003. Seepage Rate Variability in Florida Bay Driven by Atlantic Tidal Height. Biogeochem, 66:87-202

Cróquer A, Weil E. 2009. Changes in Caribbean coral disease prevalence after the 2005 bleaching event. Dis Aquat Org 87:33–43

Cróquer A, Weil E, Rogers CS. 2021. Similarities and differences between two deadly Caribbean coral diseases: white plague and stony coral tissue loss disease. Front Mar Sci 8:709544

Dahlgren C, Pizarro V, Sherman K, Greene W, Oliver J. 2021. Spatial and temporal patterns of stony coral tissue loss disease outbreaks in the Bahamas. Front Mar Sci 8:682114

Dailer ML, Knox RS, Smith JE, Napier M, Smith CM. 2010. Using δ15N values in algal tissue to map locations and potential sources of anthropogenic nutrient inputs on the island of Maui, Hawai‘i, USA. Mar Pollut Bull 60:655–671

Daszak P. 2000. Emerging infectious diseases of wildlife-- threats to biodiversity and human health. Science 287:443–449

Daszak P, Cunningham AA, Hyatt AD. 2001. Anthropogenic environmental change and the emergence of infectious diseases in wildlife. Acta Tropica 78:103–116

Dobson A, Foufopoulos J. 2001. Emerging infectious pathogens of wildlife. Phil Trans R Soc Lond B 356:1001–1012

Dustan P. 1977. Vitality of reef coral populations off Key Largo, Florida: Recruitment and mortality. Environ Geol 2:51–58

Eakin CM, Sweatman HPA, Brainard RE. 2019. The 2014–2017 global-scale coral bleaching event: insights and impacts. Coral Reefs 38:539–545

Egan S, Fernandes ND, Kumar V, Gardiner M, Thomas T. 2014. Bacterial pathogens, virulence mechanism and host defense in marine macroalgae: bacterial pathogens of macroalgae. Environ Microbiol 16:925–938

Egan S, Gardiner M. 2016. Microbial dysbiosis: rethinking disease in marine ecosystems. Front Microbiol 7:991

Elith J, Leathwick JR, Hastie T. 2008. A working guide to boosted regression trees. J Anim Ecol 77:802–813

Estrada-Saldívar N, Molina-Hernández A, Pérez-Cervantes E, Medellín-Maldonado F, González-Barrios FJ, Alvarez-Filip L. 2021. Reef-scale impacts of the stony coral tissue loss disease outbreak. Coral Reefs 39:861–866

Friedman J, Meulman J. 2003. Multiple additive regression trees with application in epidemiology. Statistics in medicine 22:1365–1381

Futch JC, Griffin DW, Lipp EK. 2010. Human enteric viruses in groundwater indicate offshore transport of human sewage to coral reefs of the upper Florida Keys. Environ Microbiol 12:964–974

Futch JC, Griffin DW, Banks K, Lipp EK. 2011. Evaluation of sewage source and fate on southeast Florida coastal reefs. Mar Pollut Bull 62:2308–2316

Gattuso J, Magnan A, Bille R, Cheung W, Howes E, Joos F, Allemand D, Bopp L, Cooley S, Eakin M, et al. 2015. Contrasting futures for ocean and society from different anthropogenic CO2 emissions scenarios. Science 349

Gil-Agudelo DL, Garzón-Ferreira J. 2001. Spatial and seasonal variation of dark spots disease in coral communities of the Santa Marta area (Colombian Caribbean). Bull Mar Sci 69:619-629

Gochfeld D, Olson J, Slattery M. 2006. Colony versus population variation in susceptibility and resistance to dark spot syndrome in the Caribbean coral *Siderastrea siderea*. Dis Aquat Org 69:53–65

Gove J, Williams G, McManus M, Clark S, Ehses J, Wedding L. 2015. Coral reef benthic regimes exhibit non-linear threshold responses to natural physical drivers. Mar Ecol Prog Ser 522:33–48

Green EP, Bruckner AW. 2000. The significance of coral disease epizootiology for coral reef conservation. Biol Cons 96:347–61

Haapkylä J, Unsworth RKF, Flavell M, Bourne DG, Schaffelke B, Willis BL. 2011. Seasonal rainfall and runoff promote coral disease on an inshore reef. PLoS ONE 6:e16893

Harvell CD, Jordan-Dahlgren E, Merkel S, Rosenberg E, Raymundo L, Smith G, Weil E, Willis B. 2007. Coral disease, environmental drivers, and the balance between coral and microbial associates. Oceanography 20:172–195

Harvell CD, Kim K, Burkholder JM, Colwell RR, Epstein PR, Grimes DJ, Hoffmann EE, Lipp EK. 1999. Emerging marine diseases: climate links and anthropogenic factors. Science 285:1505–1510

Hayes NK, Walton CJ, Gilliam DS. 2022. Tissue loss disease outbreak significantly alters the Southeast Florida stony coral assemblage. Front Mar Sci 9:975894

Heres MM, Farmer BH, Elmer F, Hertler H. 2021. Ecological consequences of stony coral tissue loss disease in the Turks and Caicos Islands. Coral Reefs 40:609–624

Hijmans RJ, Phillips S, Leathwick J, Elith J. 2017. Dismo: species distribution modeling. R package version 1.1–4. See https://CRAN.R-project.org/ package=dismo

Howells EJ, Vaughan GO, Work TM, Burt JA, Abrego D. 2020. Annual outbreaks of coral disease coincide with extreme seasonal warming. Coral Reefs 39:771–781

Hu C. 2004. Linkages between coastal runoff and the Florida Keys ecosystem: A study of a dark plume event. Geophys Res Lett 31:L15307

Jackson EJ, Donovan M, Cramer K, Lam V. 2014. Status and Trends of Caribbean Coral Reefs: 1970-2012. Global Coral Reef Monitoring Network, IUCN 306

Jones NP, Figueiredo J, Gilliam DS. 2020. Thermal stress-related spatiotemporal variations in high-latitude coral reef benthic communities. Coral Reefs 39:1661–1673

Jouffray JB, Wedding LM, Norström AV, Donovan MK, Williams GJ, Crowder LB, Erickson AL, Friedlander AM, Graham NAJ, Gove JM, et al. 2019. Parsing human and biophysical drivers of coral reef regimes. Proc R Soc B 286:20182544

Kelly E, Gidley M, Sinigalliano C, Kumar N, Solo-Gabriele HM. 2021. Impact of wastewater infrastructure improvements on beach water fecal indicator bacteria levels in Monroe County, Florida. Science of The Total Environment 763:143024

Lafferty KD. 2004. Fishing for lobsters indirectly increases epidemics in sea urchins. Ecol App 14:1566–1573

Lafferty KD, Holt RD. 2003. How should environmental stress affect the population dynamics of disease? Ecol Lett 6:654–664

Lamb JB, Willis BL, Fiorenza EA, Couch CS, Howard R, Rader DN, True JD, Kelly LA, Ahmad A, Jompa J, Harvell CD. 2018. Plastic waste associated with disease on coral reefs. Science 359:460–462

Lapointe BE, Barile PJ, Matzie WR. 2004. Anthropogenic nutrient enrichment of seagrass and coral reef communities in the Lower Florida Keys: discrimination of local versus regional nitrogen sources. J Exp Mar Biol Ecol 308:23–58

Lapointe BE, Herren LW, Debortoli DD, Vogel MA. 2015. Evidence of sewage-driven eutrophication and harmful algal blooms in Florida's Indian River Lagoon. Harmful Algae 43:82–102

Lapointe BE, Brewton RA, Herren LW, Porter JW, Hu C. 2019. Nitrogen enrichment, altered stoichiometry, and coral reef decline at Looe Key, Florida Keys, USA: a 3-decade study. Mar Biol 166:108

Lesser MP, Bythell JC, Gates RD, Johnstone RW, Hoegh-Guldberg O. 2007. Are infectious diseases really killing corals? Alternative interpretations of the experimental and ecological data. J Exp Mar Biol Ecol 346:36–44

Lipp EK, Jarrell JL, Griffin DW, Lukasik J, Jacukiewicz J, Rose JB. 2002. Preliminary evidence for human fecal contamination in corals of the Florida Keys, USA. Mar Pollut Bull 44:666–670

MacKnight NJ, Cobleigh K, Lasseigne D, Chaves-Fonnegra A, Gutting A, Dimos B, Antoine J, Fuess L, Ricci C, Butler C, et al. 2021. Microbial dysbiosis reflects disease resistance in diverse coral species. Commun Biol 4:679

Manzello DP. 2015. Rapid recent warming of coral reefs in the Florida Keys. Sci Reports 5:1–10

Maynard J, van Hooidonk R, Eakin CM, Puotinen M, Garren M, Williams G, Heron SF, Lamb J, Weil E, Willis B, Harvell CD. 2015. Projections of climate conditions that increase coral disease susceptibility and pathogen abundance and virulence. Nature Clim Change 5:688–694

McCallum H, Kuris A, Harvell C, Lafferty K, Smith G, Porter J. 2004. Does terrestrial epidemiology apply to marine systems? Trends Ecol Evol 19:585–591

Miller J, Muller E, Rogers C, Waara R, Atkinson A, Whelan KRT, Patterson M, Witcher B. 2009. Coral disease following massive bleaching in 2005 causes 60% decline in coral cover on reefs in the US Virgin Islands. Coral Reefs 28:925–937

Muller D, Leitao P, Sikor T. 2013. Comparing the determinants of cropland abandonment in Albania and Romania using boosted regression trees. Agricultural systems 117:66–77

Murray J, Prouty NG, Peek S, Paytan A. 2019. Coral skeleton δ15N as a tracer of historic nutrient loading to a coral reef in Maui, Hawaii. Sci Rep 9:5579

Mydlarz L, Couch C, Weil E, Smith G, Harvell C. 2009. Immune defenses of healthy, bleached, and diseased *Montastraea faveolata* during a natural bleaching event. Dis Aquat Org 87:67–78

Myers R, Raymundo L. 2009. Coral disease in Micronesian reefs: a link between disease prevalence and host abundance. Dis Aquat Org 87:97–104

Neely KL, Lewis CL, Lunz KS, Kabay L. 2021. Rapid population decline of the pillar coral *Dendrogyra cylindrus* along the Florida Reef Tract. Front Mar Sci 8:656515

NOAA Florida Keys National Marine Sanctuary Final Management Plan/Environmental Impact Statement. 1996. https://www.govinfo.gov/content/pkg/CZIC-qh91-75-f56-f583-1996-v-1/html/CZIC-qh91-75-f56-f583-1996-v-1.htm

Oberle FKJ, Storlazzi CD, Cheriton OM, Takesue RK, Hoover DJ, Logan JB, Runyon C, Kellogg CA, Johnson CD, Swarzenski PW. 2019. Physicochemical controls on zones of higher coral stress where black band disease occurs at Mākua Reef, Kaua‘i, Hawai‘i. Front Mar Sci 6:552

Patterson KL, Porter JW, Ritchie KB, Polson SW, Mueller E, Peters EC, Santavy DL, Smith GW. 2002. The etiology of white pox, a lethal disease of the Caribbean elkhorn coral, *Acropora palmata*. Proc Nat Acad Sci 99:8725–8730

Pollock FJ, Lamb JB, Field SN, Heron SF, Schaffelke B, Shedrawi G, Bourne DG, Willis BL. 2014. Sediment and turbidity associated with offshore dredging increase coral disease prevalence on nearby reefs. PLoS ONE 9:e102498

Porter JW, Dustan P, Jaap WC, Patterson KL, Kosmynin V, Meier OW, Patterson ME, Parsons M. 2001. Patterns of spread of coral disease in the Florida Keys. *In:* Porter JW. Editor. The Ecology and Etiology of Newly Emerging Marine Diseases. Springer Netherlands, Dordrecht, p. 1–24

Porter JW, Torres C, Sutherland KP, Meyers MK, Callahan MK, Ruzicka R, Colella M. 2011. Prevalence, severity, lethality, and recovery of dark spots syndrome among three Floridian reef-building corals. J Exp Mar Biol Ecol 408:79–87

Precht WF, Gintert BE, Robbart ML, Fura R, van Woesik R. 2016. Unprecedented disease-related coral mortality in southeastern Florida. Sci Rep 6:31374

Raj KD, Aeby GS, Mathews G, Bharath MS, Rajesh S, Laju RL, Arasamuthu A, Kumar D, Edward JKP. 2016. Patterns in the abundance of fish and snail corallivores associated

with an outbreak of acute tissue loss disease on the reefs of Vaan Island in the Gulf of Mannar, India. Proc 13th Int Coral Reef Symp, Hono., HI. 85–89

Randall CJ, Jordán-Garza AG, Muller EM, van Woesik R. 2016. Does dark spot syndrome experimentally transmit among Caribbean corals? PLoS ONE 11:e0147493

Reich CD, Shinn EA, Hickey TD, Tihansky AB. 2002. Tidal and meteorological influences on shallow marine groundwater flow in the upper Florida Keys. The Everglades, Florida Bay, and coral reefs of the Florida Keys: an ecosystem sourcebook. pp 659–676

Renegar D-EA, Blackwelder P, Miller JD, Gochfeld DJ. 2008. Ultrastructural and histological analysis of dark spot syndrome in *Siderastrea siderea* and *Agaricia agaricites*. Proc 11th Int Coral Reef Symp, Ft. Lauderdale, FL. Session 7.

Richards BL, Williams ID, Vetter OJ, Williams GJ. 2012. Environmental factors affecting large-bodied coral reef fish assemblages in the Mariana Archipelago. PLoS ONE 7:e31374

Richardson L, Goldberg W, Carlton R, Halas J. 1998. Coral disease outbreak in the Florida Keys: Plague Type II. Rev Biol Trop 46:187–198

Rosenberg E, Koren O, Reshef L, Efrony R, Zilber-Rosenberg I. 2007. The role of microorganisms in coral health, disease, and evolution. Nat Rev Microbiol 5:355–362

Santavy DL, Mueller E, Peters EC, MacLaughlin L, Porter JW, Patterson KL, Campbell J. 2001. Quantitative assessment of coral diseases in the Florida Keys: strategy and methodology. *In:* Porter JW, editor. The Ecology and Etiology of Newly Emerging Marine Diseases. Springer Netherlands, Dordrecht, pp 39–52

Schrag SJ, Wiener P. 1995. Emerging infectious disease: what are the relative roles of ecology and evolution? Trends Ecol Evol 10:319–324

Seyedsayamdost MR, Case RJ, Kolter R, Clardy J. 2011. The Jekyll-and-Hyde chemistry of *Phaeobacter gallaeciensis.* Nature Chem 3:331–335

Sharp WC, Shea CP, Maxwell KE, Muller EM, Hunt JH. 2020. Evaluating the small-scale epidemiology of the stony coral tissue loss disease in the middle Florida Keys. PLoS ONE 15:e0241871

Sheridan C, Baele JM, Kushmaro A, Fréjaville Y, Eeckhaut I. 2014. Terrestrial runoff influences white syndrome prevalence in SW Madagascar. Mar Environ Res 101:44–51

Shore-Maggio A, Aeby GS, Callahan SM. 2018. Influence of salinity and sedimentation on *Vibrio* infection of the Hawaiian coral *Montipora capitata*. Dis Aquat Org 128:63–71

Staley C, Kaiser T, Gidley ML, Enochs IC, Jones PR, Goodwin KD, Sinigalliano CD, Sadowsky MJ, Chun CL. 2017. Differential impacts of land-based sources of pollution on the microbiota of southeast Florida coral reefs. Appl Environ Microbiol 83:e03378-16

Sutherland KP, Shaban S, Joyner JL, Porter JW, Lipp EK. 2011. Human pathogen shown to cause disease in the threatened elkhorn coral *Acropora palmata*. PLoS ONE 6:e23468

Thinesh T, Mathews G, Edward JKP. 2009. Coral disease prevalence in Mandapam group of islands, Gulf of Mannar, Southeastern India. Ind J Mar Sci 38:444-450

Ushijima B, Videau P, Poscablo D, Stengel JW, Beurmann S, Burger AH, Aeby GS, Callahan SM. 2016. Mutation of the *toxR* or *mshA* genes from *Vibrio coralliilyticus* strain OCN014

reduces infection of the coral *Acropora cytherea*: virulence genes in *Vibrio coralliilyticus*. Environ Microbiol 18:4055–4067

van Woesik R, McCaffrey KR. 2017. Repeated thermal stress, shading, and directional selection in the Florida Reef Tract. Front Mar Sci 4:182

Vega Thurber RL, Burkepile DE, Fuchs C, Shantz AA, McMinds R, Zaneveld JR. 2014. Chronic nutrient enrichment increases prevalence and severity of coral disease and bleaching. Glob Change Biol 20:544–554

Vega Thurber R, Mydlarz LD, Brandt M, Harvell D, Weil E, Raymundo L, Willis BL, Langevin S, Tracy AM, Littman R, et al. 2020. Deciphering coral disease dynamics: integrating host, microbiome, and the changing environment. Front Ecol Evol 8:575927

Voss J, Richardson L. 2006. Coral diseases near Lee Stocking Island, Bahamas: patterns and potential drivers. Dis Aquat Org 69:33–40

Walker BK. 2012. Spatial analyses of benthic habitats to define coral reef ecosystem regions and potential biogeographic boundaries along a latitudinal gradient. PLoS ONE 7:e30466

Walker BK, Gilliam DS. 2013. Determining the extent and characterizing coral reef habitats of the northern latitudes of the Florida Reef Tract (Martin County). PLoS ONE 8:e80439

Walton CJ, Hayes NK, Gilliam DS. 2018. Impacts of a regional, multi-year, multi-species coral disease outbreak in southeast Florida. Front Mar Sci 5:323

Wear SL, Thurber RV. 2015. Sewage pollution: mitigation is key for coral reef stewardship. Ann NY Acad Sci 1355:15–30

Weil E. 2004. Coral reef diseases in the wider Caribbean. *In*: Rosenberg E., Loya Y. (eds) Coral Health and Disease. Springer Berlin Heidelberg, Berlin, Heidelberg, pp 35–68

Williams GJ, Aeby GS, Cowie ROM, Davy SK. 2010. Predictive modeling of coral disease distribution within a reef system. PLoS ONE 5:e9264

Willis BL, Page CA, Dinsdale EA. 2004. Coral disease on the Great Barrier Reef. *In:* Rosenberg E, Loya Y, editors. Coral Health and Disease. Springer Berlin Heidelberg, Berlin, Heidelberg, p. 69–104

Work TM, Aeby GS, Stanton FG, Fenner D. 2008. Overgrowth of fungi (endolithic hypermycosis) associated with multifocal to diffuse distinct amorphous dark discoloration of corals in the Indo-Pacific. Coral Reefs 27:663–663

Work TM, Aeby GS, Lasne G, Tribollet A. 2014. Gross and microscopic pathology of hard and soft corals in New Caledonia. J Invert Path 120:50–58

Work TM, Weil E. 2015. Dark-Spots Disease. *In:* Woodley CM, Downs CA, Bruckner AW. Porter JW, Galloway SB, editors. Diseases of Coral. John Wiley and Sons, Inc, Hoboken, NJ, p. 354–360

Zhao J, Hu C, Lapointe B, Melo N, Johns E, Smith R. 2013. Satellite-observed black water events off southwest Florida: implications for coral reef health in the Florida Keys National Marine Sanctuary. Remote Sensing 5:415–431

Table 1. Predictor variables with their description and units used to model potential human and environmental drivers of dark spot disease (DSD) across the Florida Reef Tract.

Predictor variable	Description and units	Min	Max	Data source
Depth	Depth (m) of coral at time of survey	1.1	21.6	Recorded in situ
Host abundance	Total number of host colonies along transect	1	193	Recorded in situ
Year	Survey year	2005	2019	
Habitat characterization	Reef Zone -Thirteen mutually exclusive zones identified from shore to shelf edge corresponding to typical insular shelf and coral reef geomorphology. Examples include Reef Flat, Lagoon, Back Reef, Reef Crest, Fore Reef, Bank/Shelf, Bank/Shelf Escarpment. (Zitello et al 2009)	NA	NA	https://myfwc.com/research/gis/regional-projects/unified-reef-map/
	URM GeoForm - Distinct and non-overlapping broad geomorphological structure types that can be mapped by visual interpretation of remotely sensed imagery. Examples include Coral Reef and Hardbottom, Unconsolidated Sediment, Other Delineations, and Unknown. (Zitello et al 2009)	NA	NA	
	URM GeoFormDet - Distinct and non-overlapping more descriptive geomorphological structure types that can be mapped by visual interpretation of remotely sensed imagery. Examples include Rock Outcrop, Boulder, Spur and Groove, Individual Patch Reef, Aggregated Patch Reefs, Aggregate Reef, Reef Rubble, Pavement, Pavement with Sand Channels, Rhodoliths, Sand, Mud, Sand with Scattered Coral and Rock, Artificial, Land, and Unknown. (Zitello et al 2009)	NA	NA	
	URM Class Lv - (Lv 0-4) The most detailed classification that incorporates GeoFrom and dominant live biotic cover estimates. Examples include Aggregate Reef-Algae (Continuous), Aggregate Reef-Algae (Discontinuous), Aggregate Reef-Algae (Patchy), Aggregate Reef, Live Coral (Discontinuous), Aggregate Reef-Live Coral (Patchy), Aggregate Reef- Live Coral (Sparse), Aggregate Reef-Uncolonized	NA	NA	
The Nature Conservancy Ocean Wealth Data	Mean number of coastal tourist visits that year (within 1, 2, 3, 5, 8, 13, 21 km radius)	0	6,305	https://oceanwealth.org
	Mean spend ($) by coastal tourists that year (within 1, 2, 3, 5, 8, 13, 21 km radius)	0	2,202	
Wastewater: proxy of human presence/influence (impacts to coastal areas)	Number of wastewater treatment facilities (within 13 and 21 km radius)	1	57	https://geodata.dep.state.fl.us/
	Mean design capacity (millions gallons/day) of wastewater treatment facilities (within 13 and 21 km radius)	0	113	
	Amount of wastewater (millions gallons/day) permitted to be processed (within 13 and 21 km radius)	0	113	
Septic/Sewer Areas: proxy for potential sewage pollution	Septic area (area in km^2 known/likely to drain to septic within 1, 2, 3, 5, 8, 13, 21 km radius)	0	230	https://www.floridahealth.gov/environmental-health/onsite-sewage/research/flwmi/index https://ww10.doh.state.fl.us/pub/bos/Inventory/FloridaWaterManagementInventory
	Septic count (total number of known/likely septic 'areas' that intersect 1, 2, 3, 5, 8, 13, 21 km radius)	0	97416	
	Sewer area (area in km^2 known/likely to drain to sewer within 1, 2, 3, 5, 8, 13, 21 km radius)	0	7,437	
	Sewer count (total number of known/likely sewer 'areas' that intersect 1, 2, 3, 5, 8, 13, 21 km radius)	0	403,628	
Land Use: degree of urbanization (affects coastal runoff & pollution)	High intensity land use area (in km2) = Constructed materials account for 80 – 100 % of the total cover. Vegetation, if present, occupies less than 20 % of the landscape.	0	289	https://coast.noaa.gov/digitalcoast/data/ccapregional.html
Water Quality: direct measurements at water surface and reef floor	Mean total phosphorus (um/L) (within 8, 13, 21 km radius)	0.033	0.531	http://serc.fiu.edu/wqmnetwork/
	Mean silica (um/L) (within 8, 13, 21 km radius)	0.005	7.16	
	Total organic carbon (um/L)	18.55	135.24	
	TN-ANTEK 9000=tot nitrogen (um/L)	0.060	13.24	
	Chlorophyll-a (ug/L)	0.169	68.87	
Human population (impacts to coastal areas)	Human population count (total number within 1, 2, 3, 5, 8, 13, 21 km radius)	0	1,564,696	https://www.census.gov/geographies/mapping-files/2010/geo/tiger-data.html
	Housing units (total number within 1, 2, 3, 5, 8, 13, 21 km radius)	0	709,263	

Table 2. Relative influence (%) of the 18 significant variables that together explained 64.4% of the variation in the number of DSD cases (cross-validated percentage deviance explained = 47.1%). See Table 1 for a more detailed description of the predictor variables.

Predictor	Influence (%)
mean silica (um/L) in surface waters within 13 km	18.4
total number of *S. siderea*	16.9
septic area (km^2) within 8 km	10.2
habitat characterization (URM class Lv4)	9.4
depth	8.6
year	7.1
reef zone	5.2
total organic carbon (um/L) in surface waters within 13 km	4
Chlorophyll-a (um/L) in surface waters within 21 km	3.9
total organic carbon (um/L) in surface waters within 8 km	3.8
mean total phosphorous (um/L) in bottom waters within 21 km	2.7
mean spending within 1 km	2.4
septic areas (km^2) within 21 km	1.5
total organic carbon (um/L) in bottom waters within 21 km	1.3
degree of urbanization (m^2) within 8 km	1.3
mean spending within 5 km	1.2
number of septic systems within 8 km	1.1
mean total phosphorous (um/L) in surface waters within 8 km	1.1

Figure legends

Figure 1. Example of dark spot disease (DSD) in *Siderastrea siderea*. Dark reddish-brown areas indicate disease.

Figure 2. Benthic survey locations (black dots) from 2005 to 2019 within ecoregions across Florida's Reef Tract. The table summarizes the number of sites surveyed per year.

Figure 3. Overall DSD prevalence (all sites combined for each year) through time on reefs across the Florida Reef Tract. Dotted lines indicate mean prevalence for the two time periods 2005-2012 and 2013-2019.

Figure 4. Overall frequency of occurrence of DSD (all sites combined for each year) through time. Dotted lines indicate the mean FOC for the two time periods 2005-2012 and 2013-2019.

Figure 5. Predicted relationship from the BRT model between *Siderastrea siderea* abundance and (A) surface silica concentration within a 13 km radius and (B) septic area within 8 km radius, on the number of DSD cases (fitted values). See Table 1 for a more detailed description of the predictor variables.

Figure 6. Cumulative DSD prevalence within different ecoregions of the Florida Reef Tract. Number above each bar indicates total number of surveys conducted in each region between 2005 and 2019 (excluding 2017).

Figure 7. Map of the Florida Reef Tract showing the association between DSD prevalence in *S. siderea* and the top three predictor variables among different ecoregions along the Florida Reef Tract. Colored dots indicate sites exceeding the threshold levels for the model's three top predictors of DSD occurrence. Blue dots indicate sites where the threshold levels of *S. siderea* densities are found, yellow dots indicate the threshold for silica concentrations and red dots indicate the threshold for septic areas within 8 km.

Figure 1

Year	total # sites surveyed	Depth ranges (m)	total area surveyed (m2)
2005	84	2.3-19	1680
2006	110	1.2-18.9	2200
2007	134	1.6-19.8	2680
2008	183	1.6-21.6	3660
2009	231	1.1-20.7	4620
2010	187	1.5-21.3	3740
2011	215	1.6-19.5	4300
2012	213	1.3-19.3	4260
2013	95	1.8-20.6	1900
2014	191	1.5-18.4	3820
2015	225	1.9-21.4	4500
2016	149	1.5-18.9	2980
2018	188	1.8-20.4	3760
2019	237	2.1-19.8	4740
TOTAL	**2442**	**1.1-21.6**	**48,840**

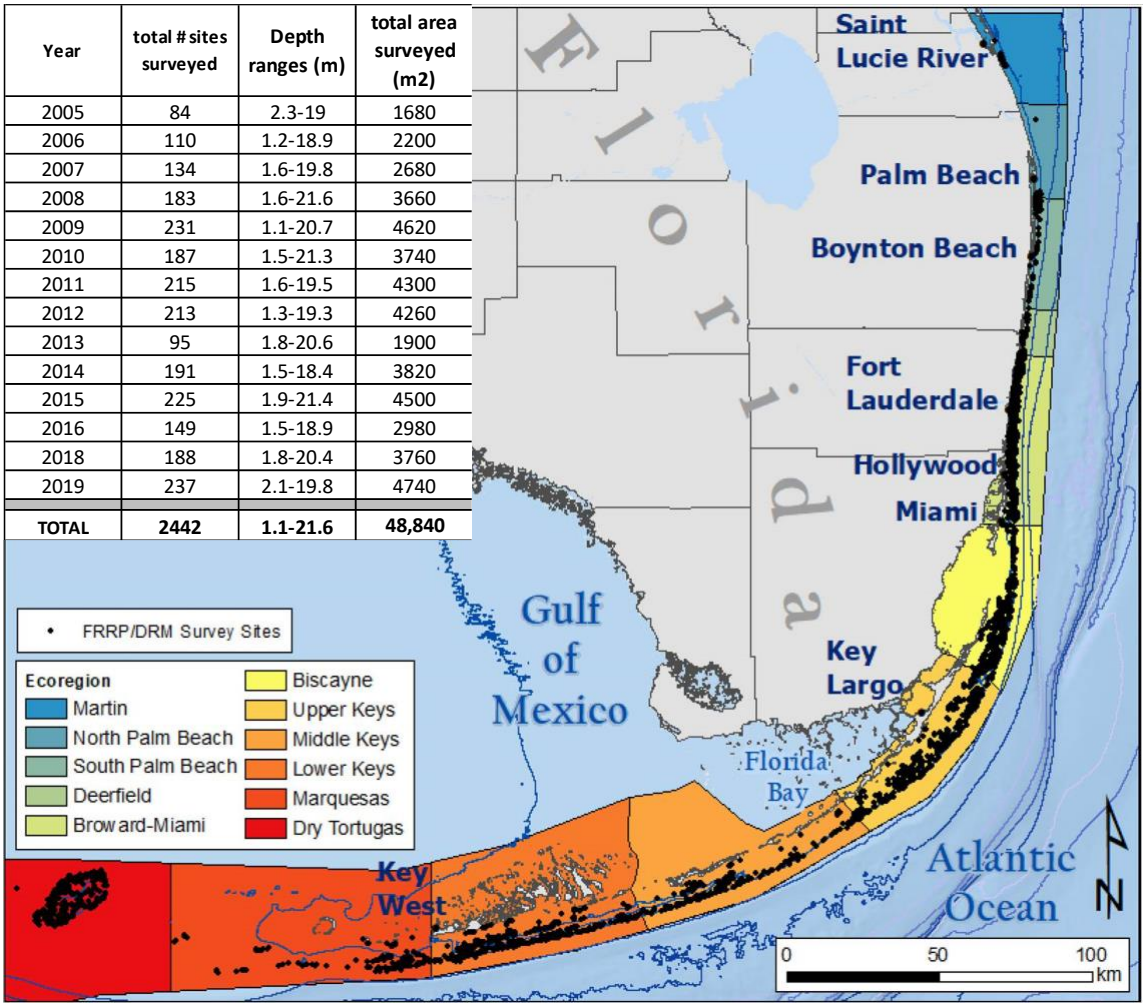

Figure 2

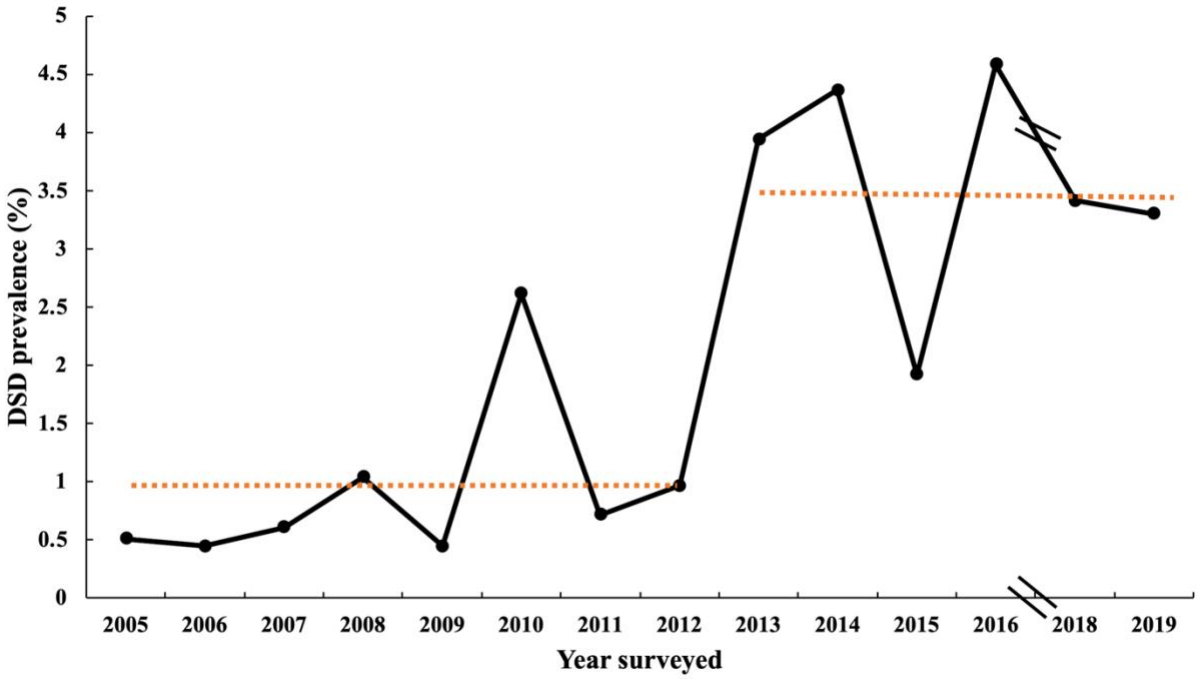
DSD prevalence (%)
5
4.5
4
3.5
3
2.5
2
1.5
1
0.5
0
2005
2006
2007
2008
2009
2010
2011
2012
2013
2014
2015
2016
2018
2019
Year surveyed

Figure 3

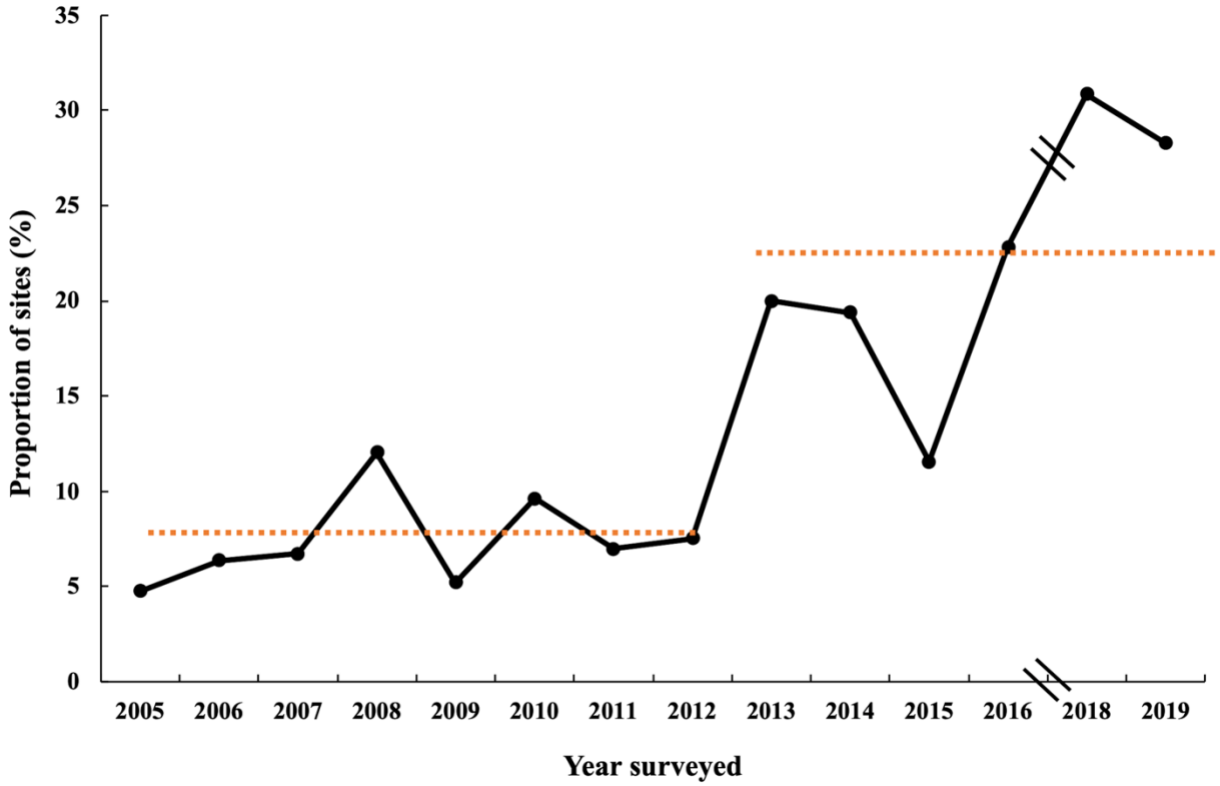
35
30
25
20
15
10
5
0
Proportion of sites (%)
2005
2006
2007
2008
2009
2010
2011
2012
2013
2014
2015
2016
2018
2019
Year surveyed

Figure 4

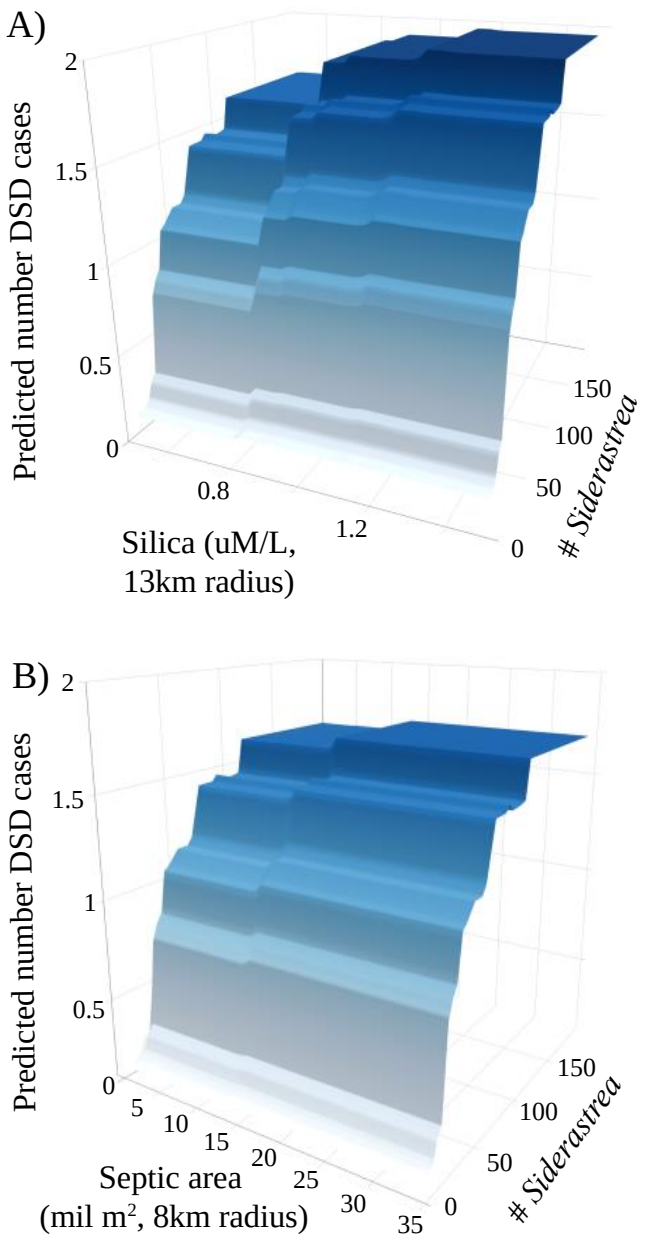
A)
Predicted number DSD cases
2
1.5
1
0.5
0
0.8
1.2
Silica (uM/L, 13km radius)
150
100
50
0
Siderastrea
B)
Predicted number DSD cases
2
1.5
1
0.5
0
5
10
15
20
25
30
35
Septic area (mil m², 8km radius)
150
100
50
0
Siderastrea

Figure 5. A & B.

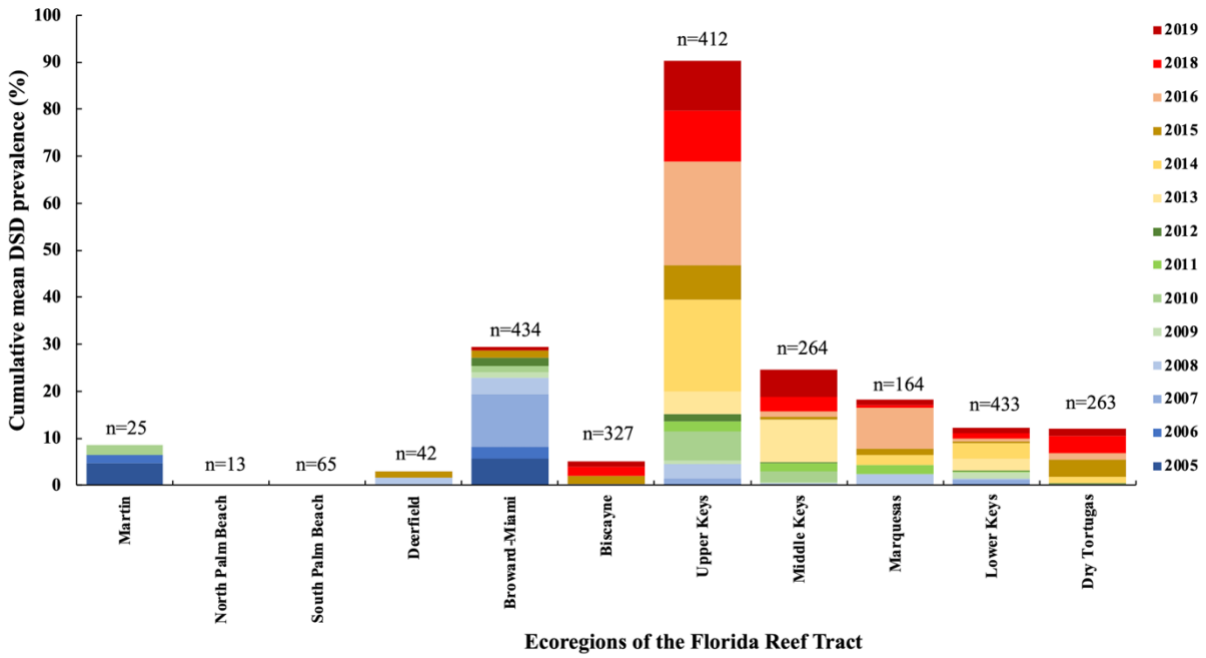
Cumulative mean DSD prevalence (%)
100
90
80
70
60
50
40
30
20
10
0
n=25
n=13
n=65
n=42
n=434
n=327
n=412
n=264
n=164
n=433
n=263
Martin
North Palm Beach
South Palm Beach
Deerfield
Broward-Miami
Biscayne
Upper Keys
Middle Keys
Marquesas
Lower Keys
Dry Tortugas
Ecoregions of the Florida Reef Tract
2019
2018
2016
2015
2014
2013
2012
2011
2010
2009
2008
2007
2006
2005

Figure 6

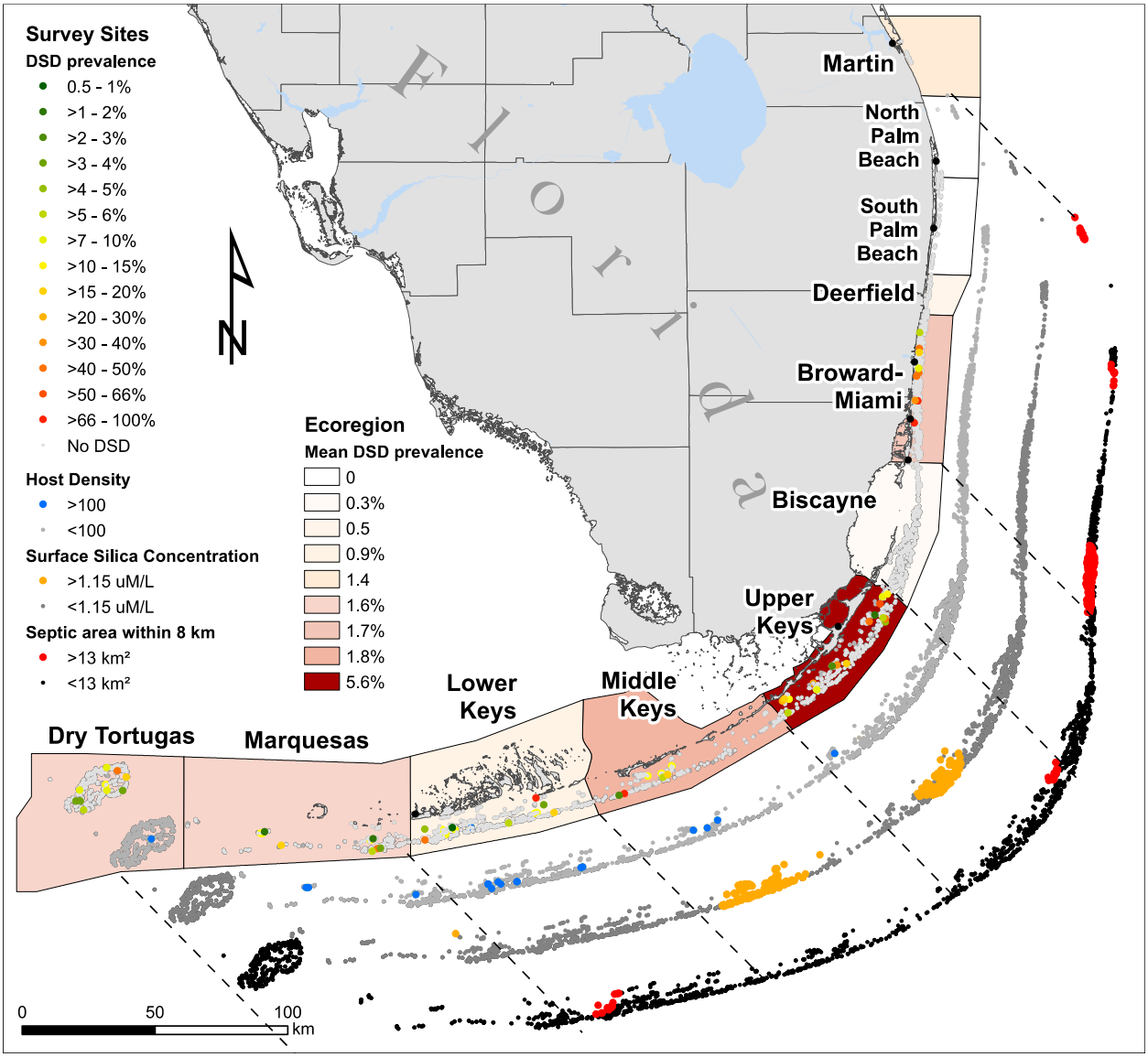
Survey Sites
DSD prevalence
0.5 - 1%
>1 - 2%
>2 - 3%
>3 - 4%
>4 - 5%
>5 - 6%
>7 - 10%
>10 - 15%
>15 - 20%
>20 - 30%
>30 - 40%
>40 - 50%
>50 - 66%
>66 - 100%
No DSD
Host Density
>100
<100
Surface Silica Concentration
>1.15 uM/L
<1.15 uM/L
Septic area within 8 km
>13 km²
<13 km²
Ecoregion
Mean DSD prevalence
0
0.3%
0.5
0.9%
1.4
1.6%
1.7%
1.8%
5.6%
Florida
Martin
North Palm Beach
South Palm Beach
Deerfield
Broward-Miami
Biscayne
Upper Keys
Middle Keys
Lower Keys
Marquesas
Dry Tortugas
N
0
50
100
km

Figure 7