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Twenty-year record of white pox disease in the Florida Keys: importance of environmental risk factors as drivers of coral health

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ABSTRACT: Declining coral populations worldwide place a special premium on identifying risks and drivers that precipitate these declines. Understanding the relationship between disease outbreaks and their drivers can help to anticipate when the risk of a disease pandemic is high. Populations of the iconic branching Caribbean elkhorn coral *Acropora palmata* have collapsed in recent decades, in part due to white pox disease (WPX). To assess the role that biotic and abiotic factors play in modulating coral disease, we present a predictive model for WPX in *A. palmata* using 20 yr of disease surveys from the Florida Keys plus environmental information collected simultaneously *in situ* and via satellite. We found that colony size was the most influential predictor for WPX occurrence, with larger colonies being at higher risk. Water quality parameters of dissolved oxygen saturation, total organic carbon, dissolved inorganic nitrogen, and salinity were implicated in WPX likelihood. Both low and high wind speeds were identified as important environmental drivers of WPX. While high temperature has been identified as an important cause of coral mortality in both bleaching and disease scenarios, our model indicates that the relative influence of HotSpot (positive summertime temperature anomaly) was low and actually inversely related to WPX risk. The predictive model developed here can contribute to enabling targeted strategic management actions and disease surveillance, enabling managers to treat the disease or mitigate disease drivers, thereby suppressing the disease and supporting the persistence of corals in an era of myriad threats.

KEY WORDS: Coral disease · *Acropora palmata* · White pox · Environmental drivers · Predictive model

1. INTRODUCTION

Coral reefs are declining globally with losses in the Caribbean and on the Great Barrier Reef (GBR) exceeding 60 and 50%, respectively, since the 1970s interspersed with periods of recovery for some corals (Gardner et al. 2003, De'ath et al. 2012, AIMS 2022). As of 2012, percent coral cover Caribbean-wide

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and GBR-wide were 16.3 and 13.8%, respectively (De'ath et al. 2012, Jackson 2014), and in the Florida Keys percent coral cover was just 4.9% in 2015 (Toth et al. 2019). These published coral cover values precede the 2014 emergence of stony coral tissue loss disease, a disease affecting multiple Caribbean coral species, with the notable exception of *Acropora* spp., that has accelerated coral cover loss in the Florida Keys and the Caribbean (Muller et al. 2020, Toth et al. 2022). As long-term coral cover declines and local species diversity decreases, the probability of extinction has increased for nearly every scleractinian coral species (Carpenter et al. 2008).

The elkhorn coral *A. palmata* (Fig. 1a), and its congener *A. cervicornis,* were once the most abundant shallow-water reef building corals in the Caribbean and the Florida Keys (Goreau 1959). Geological evidence suggests that these foundation species played a longstanding role in generating the habitat complexity of Caribbean reefs (Aronson & Precht 2001). Since the 1980s, however, *Acropora* populations have declined throughout the Caribbean region (Aronson & Precht 2001) including in the Florida Keys, where losses of *A. palmata* exceeded 70% (Baums et al. 2003, Sutherland et al. 2016). This high mortality has been followed by the bioerosion and collapse of calcium carbonate coral skeletons and the flattening of the 3-dimensional structure of Caribbean reefs (Toth et al. 2022). The consequence is altered ecosystem function that reduces biodiversity and biomass of organisms that rely on the coral reef for habitat, shelter, or food (Gratwicke & Speight 2005, Idjadi & Edmunds 2006). Due to the urgency characterized by the precipitous decline, in 2006 *A. palmata* and *A. cervicornis* became the first corals to be listed for protection under the United States Endangered Species Act (NOAA 2006).

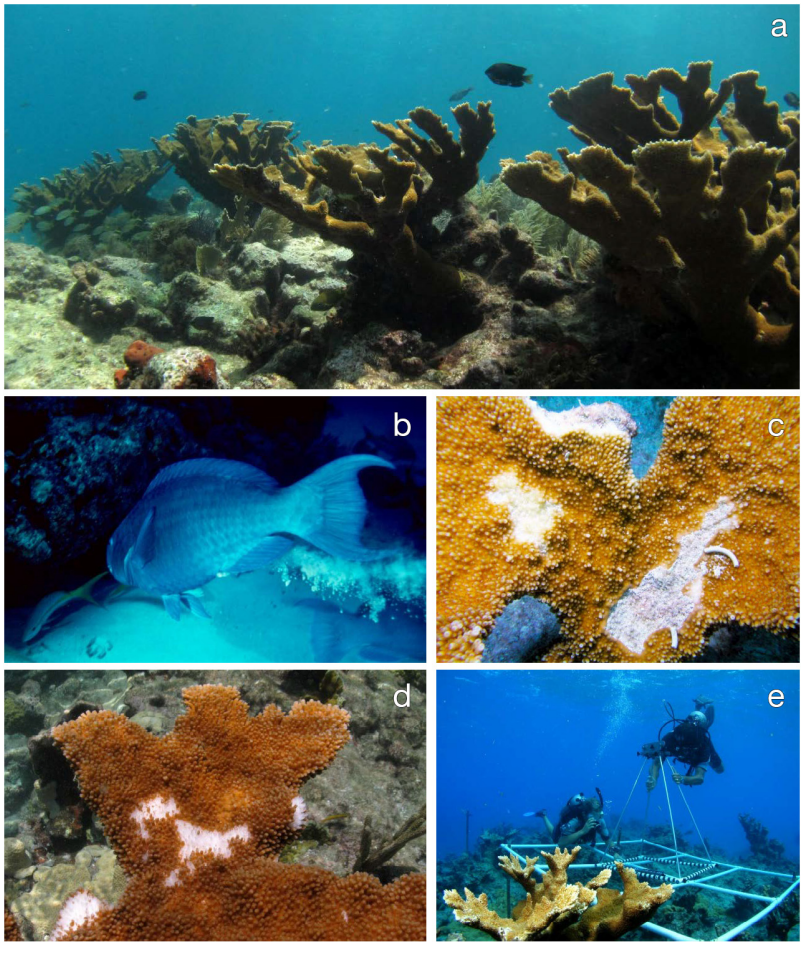

Fig. 1. (a) Apparently healthy *Acropora palmata* populations on Molasses Reef, Florida Keys. (b) Midnight parrotfish *Scarus coelestinus* passes calcareous debris in its feces after scraping coral substrates (Carysfort Reef, Florida Keys). (c) During doldrums, fish feces accumulate on *A. palmata* blades and branches (Rock Key, Florida Keys). (d) White pox disease-affected *A. palmata* (Looe Key, Florida Keys). (e) Fixed-station photography of coral reef in the Florida Keys (Eastern Dry Rocks, Florida Keys) (Photographs by James W. Porter)

Detectable stressors that have reduced population sizes and threaten recovery of *A. palmata* throughout the Caribbean region include disease, bleaching, predation, and hurricanes (Baums et al. 2003, Williams et al. 2008, Williams & Miller 2012, Bright et al. 2016). Disease of *A. palmata* most often manifests as white pox disease (WPX) or white-band disease (WBD) (Sutherland et al. 2004). WPX has affected populations of *A. palmata*, its exclusive host, for at least a quarter of a century (Sutherland et al. 2016) and has become the more prevalent of the 2 diseases on contemporary reefs (Mayor et al. 2006, Muller et al. 2008, 2014, Rogers & Muller 2012, Sutherland et al. 2016). WPX manifests as tissue loss that exposes irregularly shaped bright white patches of calcium carbonate coral skeleton (Fig. 1d). These lesions are focal to multifocal and cause partial or whole colony mortality (Patterson et al. 2002). While the earliest surveys of WPX affecting *A. palmata* populations in the Florida Keys indicated a severe

disease manifesting with large and numerous lesions that coalesce and cause whole colony mortality, recent assessments show that contemporary WPX manifests as smaller and fewer isolated lesions that result in partial, not whole, colony mortality (Sutherland et al. 2016). One pathogen, the bacterium *Serratia marcescens*, is known to cause WPX signs, especially in early epizootics; however, it is hypothesized that the same signs can also be caused by other, as yet unidentified, pathogens or abiotic stressors (Sutherland et al. 2016).

Globally, the number of coral disease outbreaks (Tracy et al. 2019) and the frequency of environmental stressors that drive these outbreaks are increasing (Hughes et al. 2018, Heron et al. 2016, Frölicher et al. 2018). Many drivers of coral disease are associated with human activities including elevated sea surface temperature (SST) (Bruno et al. 2007, Miller & Richardson 2015) and runoff of freshwater that often contains sediment, nutrients, and sewage into nearshore waters (Bruno et al. 2003, Voss & Richardson 2006b, Sutherland et al. 2011). Regions of the marine environment that are heavily influenced by anthropogenic stressors, including coral reefs of the Florida Keys, are at high risk for disease outbreaks (Maynard et al. 2015).

The increased availability of long-term environmental data (e.g. via satellites) has facilitated modeling investigations of the role of environmental factors in the occurrence and severity of coral disease. These studies have frequently demonstrated a relationship between the occurrence of disease events and specific temperature regimes (Heron et al. 2010, Maynard et al. 2015). Models created with *A. palmata* surveillance data have demonstrated a relationship between elevated seawater temperatures and outbreaks of both WPX (Muller & van Woesik 2014) and WBD (Randall & van Woesik 2015, van Woesik & Randall 2017), and these model results are supported by evidence from field investigations at many locations (Patterson et al. 2002, Muller et al. 2008, Rogers & Muller 2012, Bright et al. 2016). To date, little attention has been given to incorporating water quality parameters into predictive models for coral disease (Williams et al. 2010, Maynard et al. 2015, Sudek et al. 2015), which may be linked to issues of data consistency of satellite ocean color in near-shore environs (but see Geiger et al. 2021); however, field studies have shown that local nutrient conditions, for example, can enhance the severity and progression of disease lesions (Haapkylä et al. 2011, Kaczmarsky & Richardson 2011).

In this study, we use 20 yr (1995–2014) of WPX survey information (Sutherland et al. 2016) and site-level environmental information, including water quality data measured *in situ* and climate data measured remotely via satellite, to examine the drivers of WPX affecting *A. palmata* populations at 7 reefs throughout the Florida Keys National Marine Sanctuary (FKNMS; Fig. 2). We develop and test a predictive model that evaluates which biotic and abiotic parameters best explain WPX events. The model is first developed using subsets of data and then validated using data held back during development. Evaluating model performance on unseen data in this way lends further confidence in assessing the model's ability to predict future WPX outbreaks.

2. MATERIALS AND METHODS

2.1. *Acropora palmata* data

A. palmata data were collected in the FKNMS using 2 kinds of long-term photographic surveys. The *A. palmata* population at Eastern Dry Rocks (EDR) reef in the lower Florida Keys (Fig. 2) was monitored from 1995 to 2004 using a 13.5 m^2 grid consisting of 36 contiguous 0.75 × 0.5 m frames (Fig. 1e). Corners of the grid were demarcated by georeferenced stainless-steel survey stakes cemented into the reef (Patterson et al. 2002). This station was photographed with a Nikonos camera annually in 1995–1996, biannually in 1997 and 1998, and annually in 1999–2004 (Sutherland et al. 2016). Slides were digitally scanned at a resolution of 600 dots per inch. The health status of each colony was determined by concurrently evaluating field notes and scanned images, noting signs of WPX.

The *A. palmata* populations at 6 other reefs in the FKNMS were monitored in 40 m^2 belt transects from 2009 to 2014, 1 to 5 times per year (Sutherland et al. 2016). These 6 reefs were Carysfort and Molasses in the upper Florida Keys, Sombrero and Looe Key in the middle Keys, and Western Sambo and Rock Key in the lower Keys (Fig. 2). Each of the 6 surveyed areas had between 14 and 92 colonies, a number which remained constant throughout the 5 yr observational period. During each survey, colonies were photographed digitally with a scale in view. Individual colonies were relocated using the photograph from the previous survey and by measuring distance and bearing between the coral colony and a single survey stake implanted on each reef. Signs of WPX were recorded *in situ*.

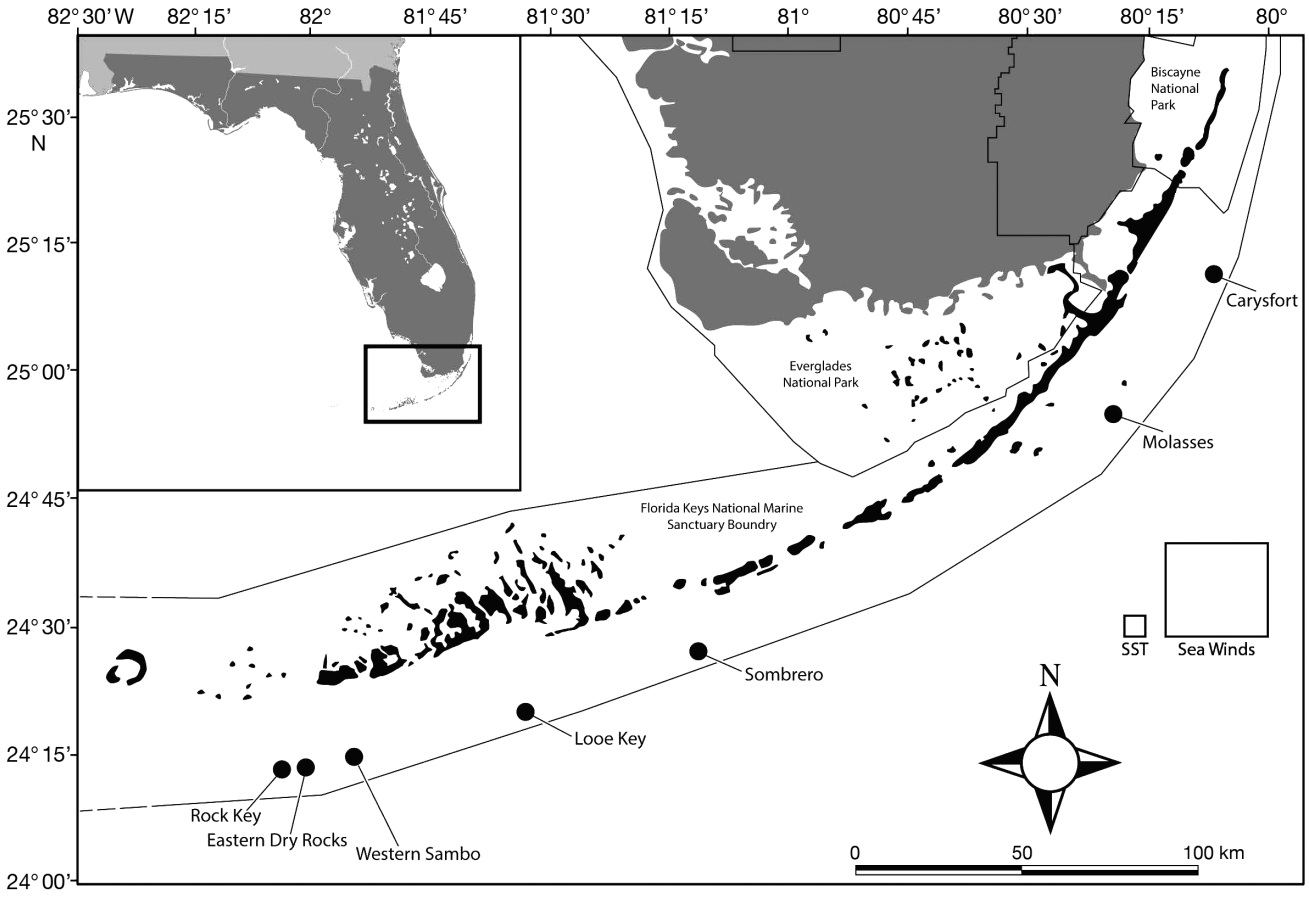

Fig. 2. The 7 *Acropora palmata* reef survey sites in the Florida Keys National Marine Sanctuary. Spatial scales of the CoralTemp sea surface temperature (SST) and Blended Sea Winds data included for reference

Digital images from the EDR (1995–2004) and FKNMS-wide (2009–2014) surveys were analyzed using ImageJ software (Schneider et al. 2012). The entire coral colony was traced, and the projected surface area of tissue (cm^2) was analyzed for disease status. For the EDR and the FKNMS-wide survey, respectively, the dimensions of each photostation frame (0.75 × 0.5 m) and the diameter of the within-image scale (3 cm) were used to calibrate the scale for each individual image analysis. For each reef site and survey, *A. palmata* data used in the model include colony size and occurrence of WPX, noted as signs present or absent (Sutherland et al. 2016, Griffin 2018).

2.2. Remotely sensed environmental data

Satellite SST data were sourced from CoralTemp v3.1, compiled by NOAA Coral Reef Watch program (Skirving et al. 2019, 2020). SST data at a spatial resolution of 0.05° (~5 km) were extracted for each of the *A. palmata* survey locations (Fig. 2) for the period 1985–2016. Temperature-based indices previously related to coral bleaching and disease included measures of summertime heat stress and conditions from the prior winter (Heron et al. 2010, Liu et al. 2014) (Table 1). HotSpot is the daily temperature anomaly above the maximum monthly mean (MMM) SST specific to each *A. palmata* survey location (Fig. 2) and degree heating weeks (DHW) accumulates HotSpot values that are 1°C or greater over a rolling 12 wk period (Liu et al. 2014). HotSpot and DHW reflect current and accumulated heat stress, respectively, associated with coral bleaching occurrences. The winter conditions metric, the accumulated SST anomaly about the winter average SST during winter months, was calculated to investigate its influence on WPX. This metric had been relevant to analysis of outbreaks of white syndromes coral diseases on the GBR (Heron et al. 2010). For comparison with *A. palmata* survey data, the maximum value from the

12 mo period prior to the survey was determined for summertime temperature metrics (HotSpot, DHW; Liu et al. 2014). For the winter conditions metric, the end-of-winter accumulation that preceded the most-recent summer was used (see Heron et al. 2010).

Measurements of wind speed and direction were extracted for each survey location from the NOAA Blended Sea Winds dataset (Zhang et al. 2006). Wind data at a spatial resolution of 0.25° (~25 km) (Fig. 2) were extracted for the disease survey locations for the period 1987–2016. Field observations by the authors (K.P.S., K.M.K., J.W.P.) have indicated that white pox responses have occurred during and immediately following periods of low wind (Kemp 2017). To test this hypothesis against survey data, we extracted the minimum wind speed from several periods (4, 7, 15, 30, 60 and 90 d windows up to each survey).

In situ temperature data were also acquired, together with the water quality parameters and are described in the following section.

2.3. Water quality data

Water quality data were compiled from the Southeast Environmental Research Center Water Quality Monitoring Project at Florida International University (SERC-FIU) (http://serc.fiu.edu/wqmnetwork/). SERC-FIU conducts quarterly surveys at 112 sites throughout the FKNMS and generates water quality data from both direct *in situ* measurements and from analyses of water samples taken at the sites (Caccia & Boyer 2005). Water quality data used in our model were collected quarterly between 1995 and 2014 and include 22 water quality parameters, most of which, due to differentials in current speed and direction, were sampled from both the surface and the bottom of each site (Table 1). For this study, water quality data were extracted from the acquired 20 yr data

Table 1. Remotely sensed environmental variables and water quality variables used to predict white pox disease (WPX) affecting *Acropora palmata* in the Florida Keys National Marine Sanctuary (FKNMS) and the relative influence (%) of each predictor variable on WPX risk. *A. palmata* data (disease presence and coral colony size) were collected with surveys at 7 reef sites in the FKNMS. Remotely sensed environmental data were extracted from the National Oceanic and Atmospheric Administration (NOAA) *CoralTemp* and *Blended Seawinds* datasets. Water quality data were compiled from the Southeast Environmental Research Center Water Quality Monitoring Project (SERC). Many water quality parameters were sampled from both the surface (S) and the bottom (B) of each site. DIN: dissolved inorganic nitrogen; TP: total phosphorous; DO: dissolved oxygen; Si: silica; TN: total nitrogen; TOC: total organic carbon; TON: total organic nitrogen;

Variable	Description	Source	Relative influence
	Disease presence	Survey	
coral.area	Colony size	Survey	44.13
wind_val_4	Wind speed 4 d window	NOAA	0.89
wind_val_7	Wind speed 7 d window	NOAA	2.42
wind_val_15	Wind speed 15 d window	NOAA	2.52
wind_val_30	Wind speed 30 d window	NOAA	0.90
wind_val_60	Wind speed 60 d window	NOAA	0.39
wind_val_90	Wind speed 90 d window	NOAA	0.17
dhw_12mt	Degree heating weeks 12 mo	NOAA	0.05
hs_12mt	HotSpot 12 mo	NOAA	1.15
cs_wint	Winter cold snap	NOAA	0.61
nh4.b	Ammonium – B	SERC	0.26
nh4.s	Ammonium – S	SERC	0.92
chla.s	Chlorphyll *a* – S	SERC	0.90
din.tp	DIN to TP	SERC	0.69
din.b	DIN – B	SERC	0.59
din.s	DIN – S	SERC	4.73
do.b	DO – B	SERC	0.48
do.s	DO – S	SERC	0.91
x.sat.b	DO saturation – B	SERC	0.47
x.sat.s	DO saturation – S	SERC	11.74
Kd	Light attenuation	SERC	1.35
x.Io	Light availability	SERC	0.33
no3.b	Nitrate – B	SERC	0.74
no3.s	Nitrate – S	SERC	0.95
nox.b	Nitric oxide – B	SERC	0.80
nox.s	Nitric oxide – S	SERC	0.18
no2.b	Nitrite – B	SERC	0.41
no2.s	Nitrite – S	SERC	0.26
sal.b	Salinity – B	SERC	0.57
sal.s	Salinity – S	SERC	3.31
Si.din	Si to DIN	SERC	1.36
siO2.b	Si – B	SERC	0.51
siO2.s	Si – S	SERC	1.31
srp.b	Soluble reactive P – B	SERC	1.04
srp.s	Soluble reactive P – S	SERC	1.29
temp.b	Temperature – B	SERC	0.25
temp.s	Temperature – S	SERC	0.32
tn.tp	TN to TP	SERC	0.22
tn.b	TN – B	SERC	0.47
tn.s	TN – S	SERC	0.21
toc.b	TOC – B	SERC	3.66
toc.s	TOC – S	SERC	0.37
ton.b	TON – B	SERC	0.23
ton.s	TON – S	SERC	0.02
tp.s	TP – S	SERC	1.59

Table 1 continued on next page

Table 1 (continued)

Variable	Description	Source	Relative influence
tp.b	TP – B	SERC	1.36
turb.b	Turbidity – B	SERC	0.61
turb.s	Turbidity – S	SERC	0.19
rCR	Reef Carysfort Reef		<0.01
rED	Reef Eastern Dry Rocks Reef		<0.01
rLK	Reef Looe Key Reef		<0.01
rMR	Reef Molasses Reef		<0.01
rRK	Reef Rock Key Reef		<0.01
rSR	Reef Sombrero Reef		0.01
rWS	Reef Western Sambo Reef		0.02
m1	Month 1 Jan		0
m2	Month 2 Feb		<0.01
m4	Month 4 Apr		0
m5	Month 5 May		0.05
m6	Month 6 Jun		0.19
m7	Month 7 Jul		0.06
m8	Month 8 Aug		0.13
m9	Month 9 Sep		0.47
m10	Month 10 Oct		0.08
m11	Month 11 Nov		0
m12	Month 12 Dec		0
y1995	Year 1995		<0.01
y1996	Year 1996		<0.01
y1997	Year 1997		<0.01
y1998	Year 1998		0
y1999	Year 1999		0
y2000	Year 2000		0
y2001	Year 2001		0
y2002	Year 2002		0
y2003	Year 2003		0
y2004	Year 2004		0
y2009	Year 2009		0.02
y2010	Year 2010		<0.01
y2011	Year 2011		<0.01
y2012	Year 2012		<0.01
y2013	Year 2013		<0.01
y2014	Year 2014		0.08

set (1995–2014) for each of the reef sites surveyed for WPX (Fig. 2). In one case, SERC-FIU did not have a monitoring station at our survey site (Rock Key). Because the statistical modeling approach we used can handle missing predictor data, we retained not available (NA) values for water quality for colonies in this reef site. The quarterly collection sampling frequency permits inclusion of chronic stressors, but not acute stressors, in the model.

2.4. Disease model

To train a model that can predict disease events from several variables, many of which are likely to be non-independent, we used a generalized boosted classification tree model (often referred to as a boosted regression tree or BRT). BRT models are well-suited to data with multiple predictors, do not require data transformations, can handle missing data, and can fit complex non-linear relationships including interaction effects between predictors (Elith et al. 2008). The BRT model quantifies and ranks predictor variables that are important in explaining the presence or absence of WPX by constructing trees to classify observations as present or absent with splits in tree branches representing splits in covariate values. Relative importance of each covariate is then quantified by how often a covariate is selected for splitting, weighted by improvement in fit to the data. From the BRT, we additionally present partial dependency plots to illustrate the relationship between a single target predictor and the response (WPX probability) while holding all other predictor variables at their average value.

To account for reef effects, we included dummy variables for each reef in the BRT analysis. This means that each observation included a binary predictor (0/1) to reflect membership of a given reef. This allows the BRT to identify particular reefs, if any, that contribute to the observed spatio–temporal pattern of WPX, rather than simply identifying 'reef' per se as an important effect. Because the distance between colonies within a reef is on the order of meters, whereas the distance between reefs is on the order of kilometers, reef membership also helps to detect spatial autocorrelation. Similarly, months and years were also included as dummy variables in case particular seasons or years were associated with WPX risk. The goal of the approach was therefore to identify abiotic and biotic predictors of WPX risk, over and above specific reef membership or time of study. This is necessary if predictive models are to be used in the future and on reefs not included in model training. We also calculated the relative strength of all pairwise combinations of the top 6 predictor variables (Hijmans et al. 2017) to evaluate potential greater-than-additive effects of predictors on WPX risk.

We trained a BRT model to classify observations for WPX as present or absent at the colony level using

biotic and abiotic information. Our full data set consisted of 1775 data points, of which 435 (24.5%) were positive for WPX. We used the *'gbm'* function from the R gbm package (Greenwell et al. 2019) to build and analyze our model, with the following specific BRT parameters: interaction depth = 5, shrinkage = 0.001, bag fraction = 0.7, train fraction = 0.8, cross-validation folds = 5, maximum number of trees = 50 000. To detect interactions between predictors, we calculated Friedman's *H*-statistic. *H* is on the scale of 0–1 with higher values indicating larger interaction effects.

Model predictions were evaluated by calculating the area under the curve (AUC), a metric used to assess how well the model correctly classifies true positives while minimizing false positives. AUC is a percentile measure for the area under a receiver operating characteristic (ROC) curve. ROC curves are drawn by varying the threshold for classification, the probability for classifying a data point into a binomial class and observing changes in the true positive rate (sensitivity) and false positive rate (1 – specificity). An AUC of 0.5 indicates that the predictive accuracy is no better than a coin toss, whereas a value of 1 or 0, respectively, indicates that the model makes the correct or incorrect prediction every time.

In our analysis, AUC is reported in 3 different ways. First, AUC was determined on the training data that is used to calibrate the model. This training-AUC is used for calibration and provides a measure on how well the model fits the provided data. Second, cross validation AUC (cv-AUC), the mean AUC value from *k*-fold cross validation during model training, was calculated to provide an estimate for how the model is expected to perform on data withheld during training. Finally, test-AUC was determined using testing data (a randomly selected 20% of initial data) that was withheld from the model prior to model training. The test-AUC verifies the estimate of cv-AUC. Both cv-AUC and test-AUC also help measure model overfitting during calibration. Model overfitting occurs when the model estimates too much of the provided training data and fails to generalize to previously unseen data.

3. RESULTS

Overall, our results indicate that a set of colony and environmental predictors provide sufficient information for predicting WPX occurrence in *Acropora palmata* colonies (training-AUC = 0.928). The AUC values generated by trained models on data withheld for testing purposes were similar to those obtained from *k*-fold cross validation (cv-AUV = 0.878 and test-AUC = 0.839). Low variation between cv-AUC and test-AUC values suggests that model over fitting was minimal.

Individual colony size was the single most important predictor of WPX events (relative importance = 44.1%, where the percentage score refers to improvement in model when the predictor is included, weighted by how often the covariate is included in the set of trees) (Fig. 3). The 5 next most important predictors were dissolved oxygen saturation (11.7%), dissolved inorganic nitrogen (DIN, 4.7%), total organic carbon (TOC, 3.6%), salinity (3.3%), and 15 d minimum wind speed (2.5%), with several other environmental predictors in the top 15 of over 80 predictor variables, including 7 d minimum wind speed (2.4%) and HotSpot (1.1%) (Fig. 3, Table 1). No particular reef or time of study predicted WPX

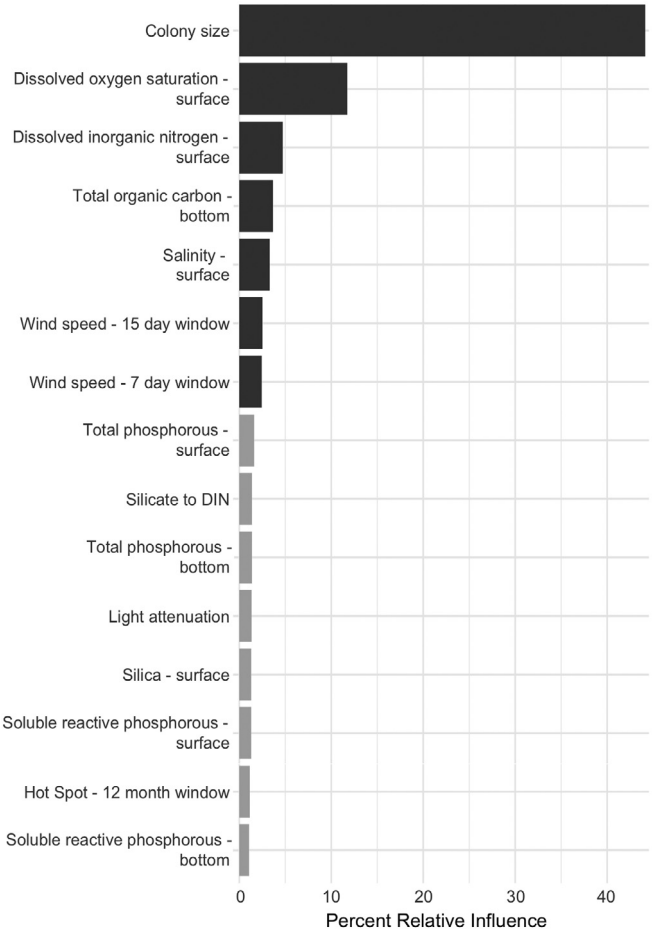

Fig. 3. Relative importance of biotic and abiotic predictors of white pox disease affecting *Acropora palmata* in the Florida Keys. Predictors shaded in black have an importance score >2.4%. Predictors with a score less than 1% are not shown

risk (Table 1). Because inter-reef distances are large (10s to 100s of km) relative to inter-colony distances within a reef (m), the fact that reef membership was unimportant in the model suggests that spatial autocorrelation is negligible. Pairwise combinations of top 6 predictor variables did not identify any strong interactive effects of predictors on WPX risk (Table 2). Model accuracy was relatively high (AUC = 0.91).

Colony size is positively associated with WPX risk (Figs. 4a & S1 in the Supplement; www.int-res.com/articles/suppl/d154p015_supp.pdf), with small colonies (<500 cm^2) experiencing a low probability of WPX (0–0.1) and larger colonies a high probability (up to 0.45). Of the top abiotic predictors dissolved oxygen saturation, DIN (the combination of nitrate, nitrite, and ammonium) (Caccia & Boyer 2005), and salinity are negatively associated with WPX risk, whereas TOC is positively associated with the disease (Fig. 4b–e). Minimum wind speed over 15 d is positively associated with WPX risk (Fig. 4f), but the minimum wind speed over shorter (7 d) and longer (30 d) windows is non-monotonically associated with

Table 2. Pairwise comparisons of top 6 predictors (see Table 1) of white pox disease (WPX) risk did not identify any greater-than-additive drivers of WPX risk. Friedman's *H*-statistic is on the scale of 0–1 with higher values indicating larger interaction effects. Water quality parameters were sampled from the surface (S) or the bottom (B) of each site. DIN: dissolved inorganic nitrogen; DO: dissolved oxygen; TOC: total organic carbon

Predictor 1	Predictor 2	Friedman's *H*-statistic
coral.area	do.s	0.20
coral.area	din.s	0.09
coral.area	toc.b	0.15
coral.area	sal.s	0.13
coral.area	wind_val_15	0.06
do.s	din.s	0.09
do.s	toc.b	0.04
do.s	sal.s	0.11
do.s	wind_val_15	0.07
din.s	toc.b	0.04
din.s	sal.s	0.02
din.s	wind_val_15	0.02
toc.b	sal.s	0.01
toc.b	wind_val_15	0.02
sal.s	wind_val_15	0.10

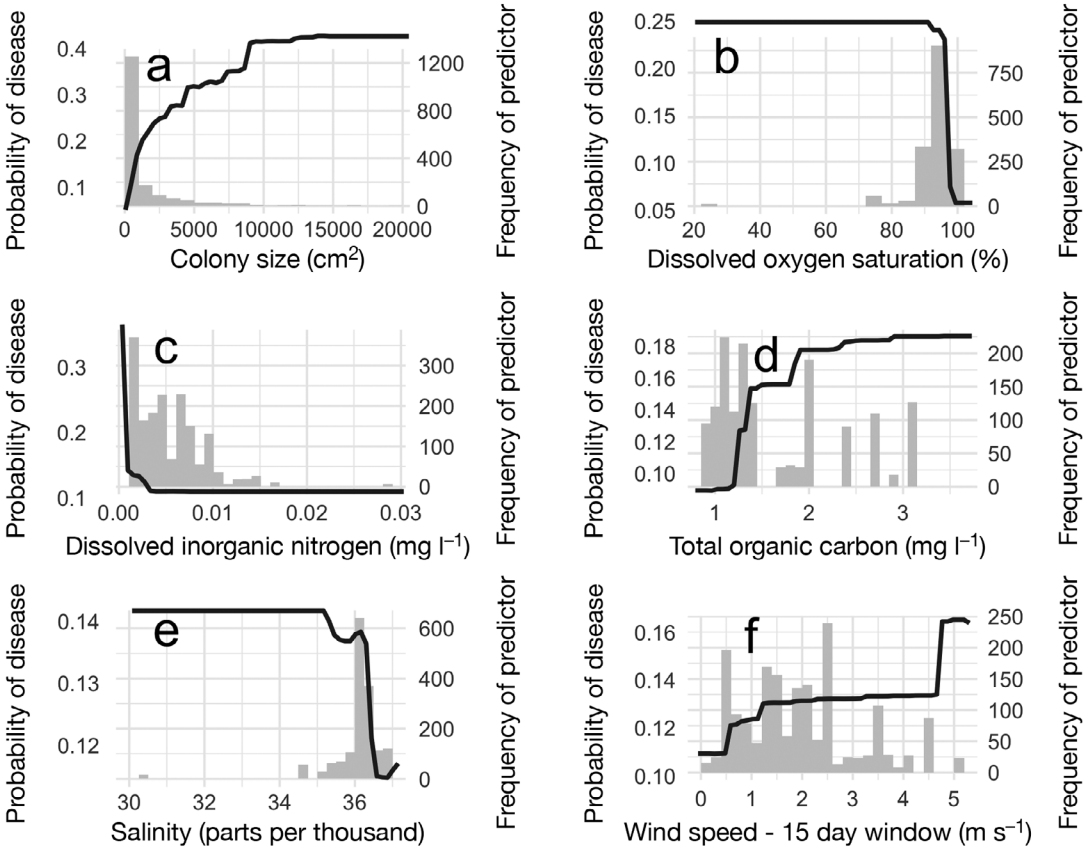

Fig. 4. Partial dependence plots of the 6 most important predictors of white pox disease affecting *Acropora palmata* in the Florida Keys. In each subplot, line shows the probability of disease (left *y*-axis) as a function of the predictor value (with other predictors held constant at their average value); histogram (right *y*-axis) shows the frequency of the predictor value in the dataset

WPX risk; specifically, with risk being highest at low and high wind speeds, and reduced WPX risk at moderate wind speeds (Fig. S2).

Colonies were not typically affected many times, and the frequency of repeated disease signs on a colony diminished with the number of disease events. Of the 123 colonies that were ever diseased during the study period, 48 were affected only once, 26 only twice, and 10 only 3 times, whilst the remaining colonies each had a higher instance of disease. Collectively, colonies affected rarely (1–3 times) represent over 68% of all colonies, and approximately 72% of consecutive observations of a colony were heterotypic regarding disease status. Average number of disease observations per colony, conditional on being affected at least once, was low (mode = 1, median = 2, mean = 3.5). Given that colonies were surveyed 7.3 times on average, these data indicate that temporal autocorrelation of disease at the colony level was low.

4. DISCUSSION

4.1. *Acropora palmata* data

Our model shows that *A. palmata* colony size was the most informative predictor for WPX in the FKNMS (Fig. 3). A positive relationship for colony size and WPX has also been reported for the *A. palmata* population in St. John, US Virgin Islands (Muller et al. 2014, Muller & van Woesik 2014). Host colonies in our study ranged in area from 5 cm^2 to 20 015 cm^2. WPX risk increased with colony size and was greatest (0.3–0.35) for colonies exceeding 4500 cm^2 (Fig. 4a). *A. palmata* colonies of this size are in the largest size class (class 4, >4000 cm^2) for contemporary populations of this species in the FKNMS and are the size class most likely to reproduce sexually (Vardi et al. 2012).

Colony size is an important factor for other white diseases of corals including white plague disease (WP) in the Caribbean and white syndromes (WS) in the Indo-Pacific. Host size and disease risk are positively correlated for WP affecting *Orbicella faveolata* and *Colpophyllia natans* (Nugues 2002) and for WS affecting multiple genera (Caldwell et al. 2020, Greene et al. 2020). Caribbean cases of black-band disease (BBD) affecting multiple host species and aspergillosis affecting *Gorgonia* spp. sea fans (Kim & Harvell 2002, Voss & Richardson 2006a) are also positively correlated with host size, as are growth anomalies affecting *A. cytherea* (Irikawa et al. 2011) and multiple species of *Porites* and *Montipora* (Caldwell et al. 2020) in the Indo-Pacific.

The link between host size and signs of host stress conforms to the understanding of coral life histories. Partial mortality is inherent to the growth of corals (Mercado-Molina et al. 2018), and many drivers of mortality are size-selective (Madin et al. 2014). Thus, large coral colonies are more prone to partial mortality than small coral colonies, and small colonies are more likely to die than are large colonies (Hughes & Jackson 1985, Babcock 1991, Vardi et al. 2012). This disproportionate partial and whole colony mortality occurs in both the absence of and in the presence of detectable stressors, including disease.

Our results for the FKNMS and those of Muller and colleagues for the US Virgin Islands (Muller et al. 2014, Muller & van Woesik 2014) indicate that large *A. palmata* colonies are more likely to show partial tissue loss caused by WPX than are small colonies (Fig. 4a). Partial tissue loss reduces coral colony size and often leads to colony fission, increasing the number of smaller individuals in a population. Small individuals may have lower growth (Mercado-Molina et al. 2018) and lower or no reproduction (Hughes & Jackson 1985, Bright et al. 2013) than large colonies but may be as old as or older.

Colony age and senescence are hypothesized to influence the size-dependent nature of signs of reduced coral health, including WPX (Kim & Harvell 2002, Irikawa et al. 2011, Muller et al. 2014, Muller & van Woesik 2014). In *A. palmata* the base of a colony is older than the branches and the age of the individual polyps decreases toward the distal branch tips (Meesters & Bak 1995). Thus *A. palmata* shows the age-size correlation documented for other coral species (Hughes & Jackson 1985), namely that larger branching colonies are of a greater age than some of the smallest colonies. Other small colonies, however, are older individuals that have been reduced in size by fragmentation or partial mortality (Babcock 1991, Miller et al. 2007, Vardi et al. 2012).

The smallest *A. palmata* colonies in the FKNMS (<100 cm^2, size class 1) are unlikely to be young, as sexual recruits are rare (Williams et al. 2008, van Woesik et al. 2014). Small *A. palmata* colonies in the FKNMS are often remnants or fragments of large colonies (Williams et al. 2014, Vardi et al. 2012). Distinguishing older remnants and fragments from young recruit colonies is difficult (Bright et al. 2013) without genotyping or long-term monitoring of individuals (Miller et al. 2007). *A. palmata* populations in the FKNMS have very low genotypic and genetic diversity (Baums et al. 2006, Williams et al. 2014), indicating

that asexual fission is the dominant mode of reproduction for populations in this region. Sexual recruitment in *A. palmata* is reduced under elevated seawater temperature (Randall & Szmant 2009), increasing the likelihood that as climate warms, the small size classes in *A. palmata* populations will be dominated even further by remnants, not recruits. These populations of older, clonal individuals may be considered as having elevated risk for WPX. If age is the factor that controls the correlation between WPX risk and colony size, then we would expect to see all size classes similarly affected by WPX; instead, our model shows an increased risk of disease for large colonies (Fig. 4a).

It is important to consider that the simultaneous distal and proximal onset of WPX signs may signal that this disease is not correlated with colony age. WPX is one of 2 white diseases that have contributed to the Caribbean-wide collapse of *A. palmata* populations; WBD is the other (Sutherland et al. 2004). WBD begins as a proximal band of tissue loss that progresses upward toward the branch tips (Gladfelter 1982). This proximal initiation of tissue loss associated with WBD indicates that polyp age and senescence may play a role in this disease (Meesters & Bak 1995). WPX differs from WBD in that the disease manifests as focal to multifocal lesions that occur all over the coral colony from colony base to branch tips, affecting both older and younger regions of the colony simultaneously. This pattern of tissue loss combined with decreased risk for the disease for colonies in the smaller size classes (size classes 1–3) (Vardi et al. 2012) hints that the greater WPX risk for large colonies may be surface area, not age, dependent.

Large colonies provide more surface area over which partial mortality can occur and specifically for coral diseases, may provide a larger area of living tissue for a waterborne pathogen (Kim & Harvell 2002, Muller et al. 2014, Muller & van Woesik 2014) or a vector to encounter. Large colonies concentrated in high density on the reef would be especially vulerable to infection from waterborne pathogenic microorganisms. The bacterial pathogen *Serratia marcescens* causes acroporid serratiosis, the one form of WPX for which a pathogen has been identified, and it is hypothesized that this pathogen may be waterborne or vector-borne (Patterson et al. 2002, Sutherland et al. 2011). The coral-eating snail *Coralliophila abbreviata* may be a vector of the bacterium causing acroporid serratiosis (Sutherland et al. 2010) and other white diseases affecting Caribbean acroporids (Williams & Miller 2005). Alternatively, sediment may be a mechanical vector for WPX. Reef sediments that originate from the feces of parrotfishes (Adam et al. 2018) may act as a reservoir (Carlos et al. 2013, Ezzat et al. 2019) of coral disease pathogens (Fig. 1b). Sediments derived from feces accumulate on the upper surfaces of *A. palmata* colonies and can cause tissue loss where it collects (Williams & Miller 2012) (Fig. 1c). *A. palmata* is inefficient at removing sediment from tissue surfaces and requires water motion to accomplish the task (Bak & Elgershuizen 1976). The correlation between WPX and reduced wind speed shown by our model (Fig. S2) may be driven by accumulation of biologically spread sediment on *A. palmata* under conditions of low wind and reduced water flow. At other times, correlation between WPX and high wind speed (Figs. 4f & S2) may suggest storm-driven sediment mobilization is also an important transmission route.

4.2. Environmental predictors

4.2.1. Dissolved oxygen saturation

Dissolved oxygen saturation in surface waters had the strongest influence of all the environmental factors in the model (Fig. 3), with a negative relationship between oxygen saturation and WPX probability (Fig. 4b). To our knowledge, there are no other studies linking elevated coral disease to reduced environmental oxygen saturation levels. Dissolved oxygen may not have a direct biological influence on disease occurrence but rather may serve as a bioindicator for localized environmental stress (Altieri et al. 2017). Low wind speed and elevated temperatures, for example, would contribute to low dissolved oxygen saturation in surface waters.

4.2.2. Wind speed

The minimum wind speed value during the 15 d preceding *A. palmata* surveys was a top predictor of WPX risk, closely followed by the same variable for only the preceding 7 d (Fig. 3). In both cases, the probability of disease was greatest at the highest wind speeds (Figs. 4f & S2). For the 7 d window, WPX was predicted under conditions of low (1–3 m s^{-1}) and high (>7 m s^{-1}), but less so at moderate wind speeds (Fig. S2). NOAA defines doldrum-like wind conditions at a threshold of <3 m s^{-1} (Liu et al. 2012). In doldrum conditions, a lack of sufficient downwelling can lead to increased stratification in the water column and reduced heat and gas exchange (Fordyce et al. 2019). Additionally, flat seas minimize

light scattering through surface turbulence leading to increased light intensity experienced by corals and their symbionts. Calm seas also favor pathogen settlement on susceptible hosts, with or without a mechanical vector (sediment or fish feces). High wind speeds increase wave action, possibly facilitating waterborne pathogen dispersal between colonies (Barott & Rohwer 2012), or agitate the water column and suspended sediment particles, promoting scouring of coral colonies by sediment (Fabricius 2005), and thereby promoting disease.

While our study is the first to explicitly examine wind speed and disease occurrence, other studies have noted an association between disease signs and low wind. Low wind conditions have coincided with WPX (Rodríguez-Martínez et al. 2001) and with WBD affecting *A. cervicornis* (Kline & Vollmer 2011), and with several other coral diseases in the Indian Ocean (Onton et al. 2011). Further, high local temperatures and coral bleaching are widely reported to be associated with doldrum conditions (Hendee et al. 2001, Maina et al. 2008, Barnes et al. 2015, Raymundo et al. 2017).

Low wind speed reduces water turbulence, thereby promoting greater water clarity and therefore also increasing solar and UV penetration. Low turbulence can also allow the persistence of elevated SST in shallow water and decrease oxygen exchange from corals to the surrounding seawater (Hendee et al. 2001, Maina et al. 2008). Doldrums thus cause a combination of stressors that have been documented to increase the incidence and severity of bleaching (Nakamura & van Woesik 2001, Finelli et al. 2006, Raymundo et al. 2017) and are also likely to facilitate or exacerbate disease. Solar irradiation, for example, increases the rate of progression of WP affecting *C. natans* in the British Virgin Islands (Muller & van Woesik 2009) and of BBD affecting *Montipora hispida* in Australia (Sato et al. 2011). Our model found that light attenuation and light availability had a low relative influence on WPX risk (Fig. 3, Table 1). Similarly, Muller & van Woesik (2014) found no correlation between light intensity and WPX in the US Virgin Islands. The correlation between wind speed and WPX revealed by our model may instead be driven by water flow, elevated temperature, or a combination of the two. Periods of elevated SST are often associated with periods of lower wind speeds within the FKNMS (Hendee et al. 2001, Barnes et al. 2015).

Evidence from multiple studies and many locations, including the FKNMS, has firmly established the association between elevated temperatures and prevalence of WPX (Patterson et al. 2002, Muller et al. 2008, Muller & van Woesik 2014, Bright et al. 2016), and temperature has emerged as an important factor in the seasonal occurrence of this disease (Rogers & Muller 2012, Sutherland et al. 2016). In our study, however, maximum temperature anomaly (HotSpot) and the measure of accumulated heat stress (DHW) ranked low in importance as direct risk factors for WPX (Fig. 3, Table 1). Furthermore, WPX was actually negatively associated with HotSpot, indicating that the absence of anomalously high temperature is associated with this disease in the Florida Keys rather than elevated temperature. This potentially counterintuitive outcome is, however, consistent with recent analyses of longitudinal coral disease observations from other global reef locations (Caldwell et al. 2020, Greene et al. 2020) and warrants further research to consider alternative underlying mechanistic explanations.

4.2.3. Total organic carbon

TOC in bottom waters aided in discriminating apparently healthy and diseased coral status (Fig. 4d). WPX probability shows a positive association with TOC that supports a correlation between elevated carbon and WPX. TOC levels in the FKNMS are typically low (Fig. 4d) with median values of 1.41 mg l^{-1} (Briceño et al. 2013). Our model shows that WPX risk increases when TOC in bottom waters exceeds this average condition and that disease probability is greatest (0.185) when TOC exceeds 2.0 mg l^{-1} (Fig. 4d). Two experimental studies investigated the effects of enrichment with organic carbon on the health of fragments of 3 species of corals collected from Caribbean Panama (Kuntz et al. 2005, Kline et al. 2006). *Orbicella annularis* (Kline et al. 2006), *Agaricia tenuifolia,* and *Porites furcata* (Kuntz et al. 2005) demonstrated WP-like patterns of tissue loss and whole coral fragment mortality following carbon addition. Our results for WPX agree with these controlled studies (Looney et al. 2010) and show that WPX signs are present in the FKNMS at TOC levels (<4 mg l^{-1}; Fig. 4d) lower than those shown to trigger WP-like signs in the laboratory (5 and 25 mg l^{-1}) (Kuntz et al. 2005, Kline et al. 2006).

4.2.4. Dissolved inorganic nitrogen

DIN in surface waters was implicated as a correlate of WPX (Fig. 3) and showed a negative association with disease risk (Fig. 4c). Like TOC, DIN is typically

low in the FKNMS (Fig. 4c), with median values less than 0.007 mg l^{-1} (Briceño et al. 2013). Our model shows that WPX probability is highest (>0.3) at these ambient values (<0.003 mg l^{-1}) for DIN (Fig. 4c). This finding suggests a correlation between WPX risk and low DIN. The DIN values used in our model only rarely exceeded the 0.75 µM (0.01 mg l^{-1}) US EPA target for healthy coral reefs in the FKNMS and even less frequently exceeded the 1.0 µM (0.014 mg l^{-1}) threshold for reef eutrophication (Lapointe et al. 2019) (Fig. 4c). Our study, thus, does not address the role of elevated DIN in WPX risk. Under laboratory conditions, elevated concentrations of ammonium increase the survival in seawater of *S. marcescens* PDL100, a confirmed causal agent of WPX (Patterson et al. 2002), but at levels greater than those encountered environmentally in our study (Looney et al. 2010). In fact, no experimental studies to date have investigated the impact of nutrient enrichment on WPX prevalence or severity.

Field assessments that address the contribution of nitrogen to coral disease risk are limited (Kim & Harvell 2002, Kuta & Richardson 2002, Voss & Richardson 2006b) but include a recent long-term study (1984–2014) conducted at Looe Key, one of the 7 reefs included in our study (Lapointe et al. 2019). This study overlaps with our study both temporally and spatially and identifies a positive correlation between DIN and living coral cover at Looe Key. Lapointe et al. (2019) hypothesize that DIN enrichment contributes to risk of coral diseases at this reef. While the average values for DIN measured by Lapointe et al. (2019) for each decade of their study (0.51 ± 0.02 µM in the 1980s to 1.21 ± 0.08 µM in the 2010s) fall within the range of DIN values found in our study (0.08–2.06 µM), their maximum measured values for DIN (2.68 µM in the 1980s to 7.28 µM in the 2010s) were much higher than any DIN value included in our model (Fig. 4c). These acute DIN stressors are proof of nutrient enrichment at Looe Key and may account for the link between DIN enrichment and the 30 yr decline in coral cover reported for this reef (Lapointe et al. 2019).

Other field investigations indicate variable responses between higher DIN and several disease parameters. For instance, Kim & Harvell (2002) investigated the effect of water quality in the prevalence and severity of aspergillosis affecting *Gorgonia ventalina* at 5 reefs in the FKNMS using field surveys and a subset of the same water quality data (June 1997) used in our model (Boyer & Jones 2001). For aspergillosis, higher DIN values (0.70–1.0 µM), exceeding the 0.75 µM target for healthy reefs in the FKNMS, did increase the severity but not the risk (prevalence) of the disease (Kim & Harvell 2002).

4.2.5. Salinity

Below average salinity (<36 ppt) in surface waters was a top predictor of WPX (Fig. 3). Reduced salinity has been reported as a driver for just one other coral disease, atramentous necrosis (AtN) affecting *Montipora* spp. in the GBR (Haapkylä et al. 2011). AtN risk increases during the rainy season and is correlated with both reduced salinity and elevated organic carbon associated with terrestrial runoff (Haapkylä et al. 2011). WPX risk, too, is correlated negatively with salinity and positively with TOC (Fig. 4d–e). Our model did not, however, identify a strong interactive effect between salinity and either of 2 top nutrient predictors (TOC and DIN) (Table 2).

In both Florida Bay and the Florida Keys, salinity is controlled by terrestrial runoff. This freshwater input is a combination of surficial and subterranean aquifer flows of water moving southward from below Lake Okeechobee (Lee et al. 2002). Within both Florida Bay (Brand 2002) and the Florida Keys (Lapointe et al. 2002) nitrogen content is tightly correlated with patterns of rainfall and terrestrial runoff. Even after the terrestrial salinity signal disappears in the full oceanic salinity of offshore coral-reef waters, the terrestrial nitrogen signal can remain (Brand 2002, Lapointe et al. 2019). On Looe Key, altered stoichiometry involving DIN:SRP ratios correlated strongly with disease and bleaching events, including within *A. palmata* populations, even in the absence of abnormally elevated temperatures (Lapointe et al. 2019). This is consistent with our findings for physio/chemical drivers of elkhorn disease in the FKNMS. It seems likely that it is not lowered salinity per se that is a driver of WPX, but rather either the pollutants or the terrestrial microbes that this terrestrial-influenced water brings with it.

4.3. Biotic and abiotic parameters affect the microbial community of *A. palmata*

The key to understanding WPX risk may lie largely in the composition of the microbial community resident in *A. palmata* host surface mucus layer (SML) or tissues. Our model implicates 2 environmental drivers that have been directly (elevated organic carbon) or indirectly (low or high wind speed) linked to alterations of the host microbiome. Several studies

hypothesize that coral-associated microbes are nutrient limited (Bruno et al. 2003, Voss & Richardson 2006b, Kaczmarsky & Richardson 2011). Investigations with a confirmed WPX pathogen, *S. marcescens* PDL100 (Krediet et al. 2009, Looney et al. 2010) and other coral pathogens (Kline et al. 2006, Smith et al. 2006, Haapkylä et al. 2011) indicate that pathogen fitness is benefited by enrichment with organic carbon, lending experimental support to our model results linking elevated organic carbon to WPX risk. These microbial blooms in the host SML can lead to hypoxia at the surface of coral tissue, triggering tissue loss that leads to partial or whole colony mortality (Smith et al. 2006). The *S. marcescens* pathogen and organic carbon may originate from a common source, sewage contamination from land (Kline et al. 2006, Sutherland et al. 2011). Alternatively, organic carbon may be released from macroalgae resident on the reef (Smith et al. 2006, Dinsdale et al. 2008). Like sewage, macroalgae may serve as a source of dissolved organic carbon and also as a reservoir of coral pathogens (Nugues et al. 2004, Barott & Rohwer 2012).

Elevated organic carbon and variations in wind speed (Ritchie 2006, Looney et al. 2010, Lee et al. 2017) are both correlated with increased abundance of candidate WPX pathogens within the genus *Vibrio* (Kemp et al. 2018). A commensal-to-*Vibrio* microbial community change has been documented from SML collected from WPX lesions (Kemp et al. 2018). No single *Vibrio* species dominates the communities isolated from lesions, and thus it is likely that the *Vibrio* spp. bloom is opportunistic and secondary to infection by a primary pathogen(s) (Kemp et al. 2018). Low wind speed may play an indirect role in this switch from a beneficial microbial community to one dominated by potentially pathogenic *Vibrio* spp. because the change occurs under conditions of elevated seawater temperature (31°C) in combination with low water flow (Lee et al. 2017). In contrast, when seawater temperature is high and water flow is also high, microbial communities are stable, suggesting that high water flow prevents proliferation of pathogenic microbes (Lee et al. 2017). Water flow is in part determined by wind, with low wind reducing water flow on the reef, and our model shows an increased risk of WPX with persistent low wind (Fig. S2). The increased risk of WPX associated with high wind speed (Figs. 4f & S2) may be driven by high water flow that facilitates *A. palmata* host exposure to a pathogen directly from the water flowing over the coral or indirectly via scour by a sediment vector (Caldwell et al. 2020).

The primary predictor of WPX risk, colony size, may also be explained by the microbial community resident in the SML or tissues of a host colony. Examination of the diversity of microbial communities based on colony age, irrespective of colony size, is limited to one study with one coral species, *Porites lutea,* and shows no evidence for age-associated changes in the microbial community (Wainwright et al. 2020). This study further supports our reasoning that the positive correlation between colony size and WPX identified by our model is not a consequence of colony age, but rather of colony surface area. Disease signs caused by a commensal-to-pathogenic bacteria shift would require a higher colony surface area, not a greater colony age. The potential for a surface area-dependent disease state is supported by evidence, from multiple Indo-Pacific coral species, of microbial community shifts associated with increased coral colony size (Williams et al. 2015, Pollock et al. 2018).

5. CONCLUSIONS

Our model shows that colony surface area is the most important predictor for WPX in the FKNMS and that large colony size and environmental factors of dissolved oxygen, DIN, TOC, salinity, and wind speed drive this disease. Our model can be used to forecast WPX outbreaks and to make predictions to assist with management of *Acropora palmata* populations in the FKNMS and may be useful regionally. For our model to be most effective, population demographics are needed. Annual monitoring is sufficient for determining size class of individuals in a population, but more frequent surveys are necessary to ground-truth the model predictions and to track the age and health status of individual colonies (and confirm that WPX is dependent on the surface area, not the age, of the host colony). If instead, colony age drives the size dependence in WPX, then large colonies and remnant small colonies will show WPX signs more frequently than small sexual recruits. A long-term monitoring program that follows populations to capture recruitment and fission, and thus colony age, is necessary to determine if WPX susceptibility increases with colony age. Such a program should include investigations of select SML from WPX-affected and unaffected tissues and colonies to elucidate environmental drivers of WPX at the microbial level.

Management strategies for *A. palmata* should be informed by data on environmental drivers that are

both predictable and diagnostic of threats to this species. Wind speed, for example, is somewhat predictable as it can be forecast over short time scales of days or weeks (e.g. Windfinder.com) and can also be tracked in real-time. Monitoring efforts immediately preceding, during, and after periods of low or high wind (especially during high temperature) will help to clarify the role of wind, and the correlated dissolved saturated oxygen levels at the surface, in disease events. Additionally, elevated TOC may indicate the presence of macroalgae-rich reefs and signal a wide-spread coral die-off in the vicinity. (Kline et al. 2006, Smith et al. 2006, Dinsdale et al. 2008). Though most abiotic factors identified by our model as drivers of WPX are beyond the immediate control of managers, poor water quality, including introduction of organic carbon from land, is a local stress factor that can, and should, be managed and controlled (Kruczynski & McManus 2002).

The continuing global decline of coral reefs has led to the call for new perspectives to help define realistic expectations for managing and mitigating coral loss under climate change (Anthony 2016). One novel approach calls for identifying reefs that have 'escaped' the negative consequences of climate change (Cinner et al. 2016). These outlier reefs may be defined, for example, as those with exceptionally high biomass following a common stressor event, such as bleaching (Cinner et al. 2016). These resilient reefs can then be further studied to identify biotic and abiotic features that differentiate coral survivorship on them from survivorship patterns on reefs that suffered higher losses during stressor events (Guest et al. 2018). We recommend adding disease prevalence to the definition of resilient reefs. By gathering information on disease prevalence, severity, and lethality, our predictive framework will generate baseline expectations for disease occurrence at particular locations and times as a function of population structure and environmental conditions. Additionally, this approach can help to identify at-risk reefs that can then be targeted for strategic, pre-emptive field studies that permit the acquisition of pre-outbreak microbiome samples (Kemp et al. 2015), captured at the onset, not just in the aftermath, of an epizootic (Burge et al. 2016), and identify reefs for application of beneficial microorganisms or probiotics (Peixoto et al. 2021).

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LITERATURE CITED

Adam TC, Duran A, Fuchs CE, Roycroft MV, Rojas MC, Ruttenberg BI, Burkepile DE (2018) Comparative analysis of foraging behavior and bite mechanics reveals complex functional diversity among Caribbean parrotfishes. Mar Ecol Prog Ser 597:207–220

AIMS (Australian Institute of Marine Science) (2022) Annual summary report of coral reef condition 2021/2022. AIMS, Townsville

Altieri AH, Harrison SB, Seemann J, Collin R, Diaz RJ, Knowlton N (2017) Tropical dead zones and mass mortalities on coral reefs. Proc Natl Acad Sci USA 114: 3660–3665

Anthony KRN (2016) Coral reefs under climate change and ocean acidification: challenges and opportunities for management and policy. Annu Rev Environ Resour 41:59–81

Aronson RB, Precht WF (2001) White-band disease and the changing face of Caribbean coral reefs. Hydrobiologia 460:25–38

Babcock RC (1991) Comparative demography of three species of scleractinian corals using age- and size-dependent classifications. Ecol Monogr 61:225–244

Bak RPM, Elgershuizen JHBW (1976) Patterns of oil-sediment rejection in corals. Mar Biol 37:105–113

Barnes BB, Hallock P, Hu C, Muller-Karger F, Palandro D, Walter C, Zepp R (2015) Prediction of coral bleaching in the Florida Keys using remotely sensed data. Coral Reefs 34:491–503

Barott KL, Rohwer FL (2012) Unseen players shape benthic competition on coral reefs. Trends Microbiol 20:621–628

Baums IB, Miller MW, Szmant AM (2003) Ecology of a coral-livorous gastropod, *Coralliophila abbreviata*, on two scleractinian hosts. I. Population structure of snails and corals. Mar Biol 142:1083–1091

Baums IB, Miller MW, Hellberg ME (2006) Geographic variation in clonal structure in a reef-building Caribbean coral, *Acropora palmata*. Ecol Monogr 76:503–519

Boyer JN, Jones RD (2001) A view from the bridge: external and internal forces affecting the ambient water quality of the Florida Keys National Marine Sanctuary (FKNMS). In: Porter JW, Porter KG (eds) The Everglades, Florida Bay, and Coral Reefs of the Florida Keys. CRC Press, Boca Raton, FL, p 609–628

Brand LE (2002) The transport of terrestrial nutrients to south Florida coastal waters. In: Porter JW, Porter KG (eds) The Everglades, Florida Bay, and Coral Reefs of the Florida Keys. CRC Press, Boca Raton, FL, p 361–414

Briceño HO, Boyer JN, Castro J, Harlem P (2013) Biogeochemical classification of South Florida's estuarine and coastal waters. Mar Pollut Bull 75:187–204

Bright AJ, Williams DE, Kramer KL, Miller MW (2013) Recovery of *Acropora palmata* in Curaçao: a comparison with the Florida Keys. Bull Mar Sci 89:747–757

Bright AJ, Rogers CS, Brandt ME, Muller E, Smith TB (2016) Disease prevalence and snail predation associated with swell-generated damage on the threatened coral, *Acropora palmata* (Lamarck). Front Mar Sci 3:77

Bruno JF, Petes LE, Drew Harvell C, Hettinger A (2003) Nutrient enrichment can increase the severity of coral diseases: effect of nutrients on coral disease severity. Ecol Lett 6:1056–1061

Bruno JF, Selig ER, Casey KS, Page CA and others (2007) Thermal stress and coral cover as drivers of coral disease outbreaks. PLOS Biol 5:e124

Burge CA, Friedman CS, Getchell R, House M and others (2016) Complementary approaches to diagnosing marine diseases: a union of the modern and the classic. Phil Trans R Soc B 371:20150207

Caccia VG, Boyer JN (2005) Spatial patterning of water quality in Biscayne Bay, Florida as a function of land use and water management. Mar Pollut Bull 50:1416–1429

Caldwell JM, Aeby G, Heron SF, Donahue MJ (2020) Case-control design identifies ecological drivers of endemic coral diseases. Sci Rep 10:2831

Carlos C, Torres TT, Ottoboni LMM (2013) Bacterial communities and species-specific associations with the mucus of Brazilian coral species. Sci Rep 3:1624

Carpenter KE, Abrar M, Aeby G, Aronson RB and others (2008) One-third of reef-building corals face elevated extinction risk from climate change and local impacts. Science 321:560–563

Cinner JE, Huchery C, MacNeil MA, Graham NAJ and others (2016) Bright spots among the world's coral reefs. Nature 535:416–419

De'ath G, Fabricius KE, Sweatman H, Puotinen M (2012) The 27-year decline of coral cover on the Great Barrier Reef and its causes. Proc Natl Acad Sci USA 109: 17995–17999

Dinsdale EA, Pantos O, Smriga S, Edwards RA and others (2008) Microbial ecology of four coral atolls in the Northern Line Islands. PLOS ONE 3:e1584

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

Ezzat L, Lamy T, Maher RL, Munsterman KS and others (2019) Surgeonfish feces increase microbial opportunism in reef-building corals. Mar Ecol Prog Ser 631:81–97

Fabricius KE (2005) Effects of terrestrial runoff on the ecology of corals and coral reefs: review and synthesis. Mar Pollut Bull 50:125–146

Finelli CM, Helmuth BST, Pentcheff ND, Wethey DS (2006) Water flow influences oxygen transport and photosynthetic efficiency in corals. Coral Reefs 25:47–57

Fordyce AJ, Ainsworth TD, Heron SF, Leggat W (2019) Marine heatwave hotspots in coral reef environments: physical drivers, ecophysiological outcomes, and impact upon structural complexity. Front Mar Sci 6:498

Frölicher TL, Fischer EM, Gruber N (2018) Marine heatwaves under global warming. Nature 560:360–364

Gardner TA, Côté IM, Gill JA, Grant A, Watkinson AR (2003) Long-term region-wide declines in Caribbean corals. Science 301:958–960

Geiger EF, Heron SF, Hernández WJ, Caldwell JM and others (2021) Optimal spatiotemporal scales to aggregate satellite ocean color data for nearshore reefs and tropical coastal waters: two case studies. Front Mar Sci 8:643302

Gladfelter WB (1982) White-band disease in *Acropora palmata*: implications for the structure and growth of shallow reefs. Bull Mar Sci 32:639–643

Goreau TF (1959) The ecology of Jamaican coral reefs I. Species composition and zonation. Ecology 40:67–90

Gratwicke B, Speight MR (2005) Effects of habitat complexity on Caribbean marine fish assemblages. Mar Ecol Prog Ser 292:301–310

Greene A, Donahue MJ, Caldwell JM, Heron SF, Geiger E, Raymundo LJ (2020) Coral disease time series highlight size-dependent risk and other drivers of white syndrome in a multi-species model. Front Mar Sci 7: 601469

Greenwell B, Boehmke B, Cunningham J, Developers G (2019) Gbm: generalized boosted regression models. R package version 2.1.5. https://CRAN.R-project.org/package=gbm

Griffin AP (2018) Understanding and predicting coral disease across scales: white pox disease and the critically-endangered elkhorn coral, *Acropora palmata*. PhD dissertation, University of Georgia, Athens, GA

Guest JR, Edmunds PJ, Gates RD, Kuffner IB and others (2018) A framework for identifying and characterising coral reef 'oases' against a backdrop of degradation. J Appl Ecol 55:2865–2875

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

Hendee JC, Mueller E, Humphrey C, Moore T (2001) A data-driven expert system for producing coral bleaching alerts at Sombrero Reef in the Florida Keys, USA. Bull Mar Sci 69:673–684

Heron SF, Willis BL, Skirving WJ, Eakin CM, Page CA, Miller IR (2010) Summer hot snaps and winter conditions: modelling white syndrome outbreaks on Great Barrier Reef corals. PLOS ONE 5:e12210

Heron SF, Maynard JA, van Hooidonk R, Eakin CM (2016) Warming trends and bleaching stress of the world's coral reefs 1985–2012. Sci Rep 6:38402

Hijmans RJ, Phillips S, Leathwick J, Elith J (2017) Dismo: species distribution modeling. R package version 1 1-4. https://CRAN.R-project.org/package=dismo

Hughes TP, Jackson JBC (1985) Population dynamics and life histories of foliaceous corals. Ecol Monogr 55:141–166

Hughes TP, Kerry JT, Baird AH, Connolly SR and others (2018) Global warming transforms coral reef assemblages. Nature 556:492–496

Idjadi JA, Edmunds PJ (2006) Scleractinian corals as facilitators for other invertebrates on a Caribbean reef. Mar Ecol Prog Ser 319:117–127

Irikawa A, Casareto BE, Suzuki Y, Agostini S, Hidaka M, van Woesik R (2011) Growth anomalies on *Acropora cytherea* corals. Mar Pollut Bull 62:1702–1707

Jackson J (2014) Status and trends of Caribbean coral reefs: 1970–2012. Global Coral Reef Monitoring Network, Washington, DC

Kaczmarsky L, Richardson LL (2011) Do elevated nutrients and organic carbon on Philippine reefs increase the prevalence of coral disease? Coral Reefs 30:253–257

Kemp KM (2017) The role of *Vibrio* bacteria in disease and dysbiosis of the threatened Caribbean elkhorn coral, *Acropora palmata*. PhD dissertation, University of Georgia, Athens, GA

Kemp DW, Rivers AR, Kemp KM, Lipp EK, Porter JW, Wares JP (2015) Spatial homogeneity of bacterial communities associated with the surface mucus layer of the reef-building coral *Acropora palmata.* PLOS ONE 10:e0143790

Kemp KM, Westrich JR, Alabady MS, Edwards ML, Lipp EK (2018) Abundance and multilocus sequence analysis of *Vibrio* bacteria associated with diseased elkhorn coral (*Acropora palmata*) of the Florida Keys. Appl Environ Microbiol 84:e01035–17

Kim K, Harvell C (2002) Aspergillosis of sea fan corals: disease dynamics in the Florida Keys. In: Porter JW, Porter KG (eds) The Everglades, Florida Bay, and Coral Reefs of the Florida Keys. CRC Press, Boca Raton, FL, p 813–824.

Kline DI, Vollmer SV (2011) White band disease (type I) of endangered Caribbean acroporid corals is caused by pathogenic bacteria. Sci Rep 1:7

Kline DI, Kuntz NM, Breitbart M, Knowlton N, Rohwer F (2006) Role of elevated organic carbon levels and microbial activity in coral mortality. Mar Ecol Prog Ser 314:119–125

Krediet CJ, Ritchie KB, Cohen M, Lipp EK, Sutherland KP, Teplitski M (2009) Utilization of mucus from the coral *Acropora palmata* by the pathogen *Serratia marcescens* and by environmental and coral commensal bacteria. Appl Environ Microbiol 75:3851–3858

Kruczynski WL, McManus F (2002) Water quality concerns in the Florida Keys: sources, effects, and solutions. In: Porter JW, Porter KG (eds) The Everglades, Florida Bay, and Coral Reefs of the Florida Keys. CRC Press, Boca Raton, FL, p 827–881

Kuntz NM, Kline DI, Sandin SA, Rohwer F (2005) Pathologies and mortality rates caused by organic carbon and nutrient stressors in three Caribbean coral species. Mar Ecol Prog Ser 294:173–180

Kuta K, Richardson L (2002) Ecological aspects of black band disease of corals: relationships between disease incidence and environmental factors. Coral Reefs 21:393–398

Lapointe BE, Matzie WR, Barile PJ (2002) Biotic phase-shifts in Florida Bay and fore reef communities of the Florida Keys: linkages with historical freshwater flows and nitrogen loading from Everglades runoff. In: Porter JW, Porter KG (eds) The Everglades, Florida Bay, and Coral Reefs of the Florida Keys. CRC Press, Boca Raton, FL, p 629–648

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

Lee TN, Williams E, Johns E, Wilson D, Smith NP (2002) Transport processes linking south Florida coastal ecosystems. In: Porter JW, Porter KG (eds) The Everglades, Florida Bay, and Coral Reefs of the Florida Keys. CRC Press, Boca Raton, FL, p 309–342

Lee STM, Davy SK, Tang SL, Kench PS (2017) Water flow buffers shifts in bacterial community structure in heat-stressed *Acropora muricata.* Sci Rep 7:43600

Liu G, Eakin CM, Rauenzahn JL, Christensen TRL and others (2012) NOAA coral reef watch's decision support system for coral reef management. Proc 12th Int Coral Reef Symp, Cairns

Liu G, Heron SF, Eakin CM, Muller-Karger FE and others (2014) Reef-scale thermal stress monitoring of coral ecosystems: new 5-km global products from NOAA coral reef watch. Remote Sens 6:11579–11606

Looney EE, Sutherland KP, Lipp EK (2010) Effects of temperature, nutrients, organic matter and coral mucus on the survival of the coral pathogen, *Serratia marcescens* PDL100. Environ Microbiol 12:2479–2485

Madin JS, Baird AH, Dornelas M, Connolly SR (2014) Mechanical vulnerability explains size-dependent mortality of reef corals. Ecol Lett 17:1008–1015

Maina J, Venus V, McClanahan TR, Ateweberhan M (2008) Modelling susceptibility of coral reefs to environmental stress using remote sensing data and GIS models. Ecol Model 212:180–199

Maynard J, van Hooidonk R, Eakin CM, Puotinen M and others (2015) Projections of climate conditions that increase coral disease susceptibility and pathogen abundance and virulence. Nat Clim Chang 5: 688

Mayor PA, Rogers CS, Hillis-Starr ZM (2006) Distribution and abundance of elkhorn coral, *Acropora palmata*, and prevalence of white-band disease at Buck Island Reef National Monument, St. Croix, US Virgin Islands. Coral Reefs 25:239–242

Meesters EH, Bak RPM (1995) Age-related deterioration of a physiological function in the branching coral *Acropora palmata.* Mar Ecol Prog Ser 121:203–209

Mercado-Molina AE, Ruiz-Diaz CP, Sabat AM (2018) Tissue loss rather than colony size determines the demographic fate of the branching coral *Acropora cervicornis.* Mar Ecol Prog Ser 597:147–159

Miller AW, Richardson LL (2015) Emerging coral diseases: a temperature-driven process? PSZNI: Mar Ecol 36: 278–291

Miller MW, Baums IB, Williams DE (2007) Visual discernment of sexual recruits is not feasible for *Acropora palmata.* Mar Ecol Prog Ser 335:227–231

Muller EM, van Woesik R (2009) Shading reduces coral-disease progression. Coral Reefs 28:757–760

Muller EM, van Woesik R (2014) Genetic susceptibility, colony size, and water temperature drive white-pox disease on the coral *Acropora palmata.* PLOS ONE 9: e110759

Muller EM, Rogers CS, Spitzack AS, van Woesik R (2008) Bleaching increases likelihood of disease on *Acropora palmata* (Lamarck) in Hawksnest Bay, St John, US Virgin Islands. Coral Reefs 27:191–195

Muller EM, Rogers CS, van Woesik R (2014) Early signs of recovery of *Acropora palmata* in St. John, US Virgin Islands. Mar Biol 161:359–365

Muller EM, Sartor C, Alcaraz NI, van Woesik R (2020) Spatial epidemiology of the stony-coral-tissue-loss disease in Florida. Front Mar Sci 7:163

Nakamura T, van Woesik R (2001) Water-flow rates and passive diffusion partially explain differential survival of corals during the 1998 bleaching event. Mar Ecol Prog Ser 212:301–304

NOAA (National Oceanic and Atmospheric Administration) (2006) Endangered and threatened species: final listing determinations for elkhorn coral and staghorn coral. Fed Regist 71:26852–26872

Nugues MM (2002) Impact of a coral disease outbreak on coral communities in St. Lucia: What and how much has been lost? Mar Ecol Prog Ser 229:61–71

Nugues MM, Smith GW, Hooidonk RJ, Seabra MI, Bak RPM (2004) Algal contact as a trigger for coral disease. Ecol Lett 7:919–923

Onton K, Page CA, Wilson SK, Neale S, Armstrong S (2011) Distribution and drivers of coral disease at Ningaloo reef, Indian Ocean. Mar Ecol Prog Ser 433:75–84

Patterson KL, Porter JW, Ritchie KB, Polson SW and others (2002) The etiology of white pox, a lethal disease of the Caribbean elkhorn coral, *Acropora palmata.* Proc Natl Acad Sci USA 99:8725–8730

Peixoto RS, Sweet M, Villela HDM, Cardoso P and others (2021) Coral probiotics: premise, promise, prospects. Annu Rev Anim Biosci 9:265–288

Pollock FJ, McMinds R, Smith S, Bourne DG and others (2018) Coral-associated bacteria demonstrate phylosymbiosis and cophylogeny. Nat Commun 9:4921

Randall CJ, Szmant AM (2009) Elevated temperature affects development, survivorship, and settlement of the elkhorn coral, *Acropora palmata* (Lamarck 1816). Biol Bull 217:269–282

Randall CJ, van Woesik R (2015) Contemporary white-band disease in Caribbean corals driven by climate change. Nat Clim Chang 5:375

Raymundo LJ, Burdick D, Lapacek VA, Miller R, Brown V (2017) Anomalous temperatures and extreme tides: Guam staghorn *Acropora* succumb to a double threat. Mar Ecol Prog Ser 564:47–55

Ritchie KB (2006) Regulation of microbial populations by coral surface mucus and mucus-associated bacteria. Mar Ecol Prog Ser 322:1–14

Rodríguez-Martínez RE, Banaszak AT, Jordán-Dahlgren E (2001) Necrotic patches affect *Acropora palmata* (Scleractinia: Acroporidae) in the Mexican Caribbean. Dis Aquat Org 47:229–234

Rogers CS, Muller EM (2012) Bleaching, disease and recovery in the threatened scleractinian coral *Acropora palmata* in St. John, US Virgin Islands: 2003–2010. Coral Reefs 31:807–819

Sato Y, Bourne DG, Willis BL (2011) Effects of temperature and light on the progression of black band disease on the reef coral, *Montipora hispi*da. Coral Reefs 30:330

Schneider CA, Rasband WS, Eliceiri KW (2012) NIH Image to ImageJ: 25 years of image analysis. Nat Methods 9: 671–675

Skirving WJ, Heron SF, Marsh BL, Liu G, De La Cour JL, Geiger EF, Eakin CM (2019) The relentless march of mass coral bleaching: a global perspective of changing heat stress. Coral Reefs 38:547–557

Skirving W, Marsh B, De La Cour J, Liu G and others (2020) CoralTemp and the Coral Reef Watch Coral Bleaching Heat Stress Product suite version 3.1. Remote Sens 12: 3856

Smith JE, Shaw M, Edwards RA, Obura D and others (2006) Indirect effects of algae on coral: algae-mediated, microbe-induced coral mortality. Ecol Lett 9: 835–845

Sudek M, Williams GJ, Runyon C, Aeby GS, Davy SK (2015) Disease dynamics of *Porites* bleaching with tissue loss: prevalence, virulence, transmission, and environmental drivers. Dis Aquat Org 113:59–68

Sutherland KP, Porter JW, Torres C (2004) Disease and immunity in Caribbean and Indo-Pacific zooxanthellate corals. Mar Ecol Prog Ser 266:273–302

Sutherland KP, Porter JW, Turner JW, Thomas BJ and others (2010) Human sewage identified as likely source of white pox disease of the threatened Caribbean elkhorn coral, *Acropora palmata.* Environ Microbiol 12:1122–1131

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

Sutherland KP, Berry B, Park A, Kemp DW, Kemp KM, Lipp EK, Porter JW (2016) Shifting white pox aetiologies affecting *Acropora palmata* in the Florida Keys, 1994–2014. Phil Trans R Soc B 371:20150205

Toth LT, Stathakopoulos A, Kuffner IB, Ruzicka RR, Colella MA, Shinn EA (2019) The unprecedented loss of Florida's reef-building corals and the emergence of a novel coral-reef assemblage. Ecology 100:e02781

Toth LT, Courtney TA, Colella MA, Kupfner Johnson SA, Ruzicka RR (2022) The past, present, and future of coral reef growth in the Florida Keys. Glob Chang Biol 28: 5294–5309

Tracy AM, Pielmeier ML, Yoshioka RM, Heron SF, Harvell CD (2019) Increases and decreases in marine disease reports in an era of global change. Proc R Soc B 286: 20191718

van Woesik R, Randall CJ (2017) Coral disease hotspots in the Caribbean. Ecosphere 8:e01814

van Woesik R, Scott WJ IV, Aronson RB (2014) Lost opportunities: coral recruitment does not translate to reef recovery in the Florida Keys. Mar Pollut Bull 88:110–117

Vardi T, Williams DE, Sandin SA (2012) Population dynamics of threatened elkhorn coral in the northern Florida Keys, USA. Endang Species Res 19:157–169

Voss JD, Richardson LL (2006a) Coral diseases near Lee Stocking Island, Bahamas: patterns and potential drivers. Dis Aquat Org 69:33–40

Voss JD, Richardson LL (2006b) Nutrient enrichment enhances black band disease progression in corals. Coral Reefs 25:569–576

Wainwright BJ, Zahn GL, Afiq-Rosli L, Tanzil JTI, Huang D (2020) Host age is not a consistent predictor of microbial diversity in the coral *Porites lutea.* Sci Rep 10:14376

Williams DE, Miller MW (2005) Coral disease outbreak: pattern, prevalence and transmission in *Acropora cervicornis.* Mar Ecol Prog Ser 301:119–128

Williams DE, Miller MW (2012) Attributing mortality among drivers of population decline in *Acropora palmata* in the Florida Keys (USA). Coral Reefs 31:369–382

Williams DE, Miller MW, Kramer KL (2008) Recruitment failure in Florida Keys *Acropora palmata*, a threatened Caribbean coral. Coral Reefs 27:697–705

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

Williams DE, Miller MW, Baums IB (2014) Cryptic changes in the genetic structure of a highly clonal coral population and the relationship with ecological performance. Coral Reefs 33:595–606

Williams AD, Brown BE, Putchim L, Sweet MJ (2015) Age-related shifts in bacterial diversity in a reef coral. PLOS ONE 10:e0144902

Zhang HM, Bates JJ, Reynolds RW (2006) Assessment of composite global sampling: sea surface wind speed. Geophys Res Lett 33:L17714

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