

Research

Environmental and biological drivers of white plague disease on shallow and mesophotic coral reefs

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Outbreaks of coral white plague (WP) disease have caused significant regional declines of reef-building Caribbean corals. Due to a greater availability of epidemiological data, studies have primarily focused on shallow coral reefs (< 30 m). In the U.S. Virgin Islands, however, WP disease prevalence appears to be higher on upper mesophotic (30–40 m) coral reefs when compared with shallow reefs and may be inhibiting coral recovery after environmental disturbances. The aim of this study was to investigate the relationship of environmental drivers with spatio-temporal patterns of WP prevalence across shallow and mesophotic coral reefs in the U.S. Virgin Islands. We recorded WP prevalence at 13 reef sites (five shallow, three mid-depth and five upper-mesophotic reefs) across the south shelf of St. Thomas approximately monthly between 2012 and 2015 using a drop camera method. The influence of environmental factors on disease prevalence was investigated using Bayesian inference with generalized linear mixed-effect models. We found that WP tended to increase during the beginning of the rainy season (June), and when levels of water turbidity and temperature were higher, and levels of oxygen and salinity lower. The disease prevalence was higher on mesophotic than on shallow or mid-depth reefs, probably due to higher availability of corals (host), and a possible temperature threshold for WP occurrence that allows long-term persistence (year-round) of the disease on upper-mesophotic reefs. This is the first study to implement the drop camera method to survey a coral disease over several reef sites and depths. This method can be applied on surveys of other rapid tissue loss diseases, such as the newly emergent stony-coral-tissue-loss-disease (SCTLD).

Keywords: Bayesian inference, coral reefs, environmental factors, mesophotic corals, *Orbicella*, white plague disease

Introduction

Disease outbreaks have caused regional declines of living coral in recent times, affecting the diversity and structure of coral reefs around the world (Aronson and Precht 2001, Raymundo et al. 2005, Cróquer and Weil 2009b, Miller et al. 2009, Ruiz-Moreno et al. 2012, Muller et al. 2020). These outbreaks are associated with changes of environmental conditions, such as thermal anomalies (Harvell et al. 2001, Bruno et al. 2007, Muller et al. 2008, Brandt and McManus 2009, Ruiz-Moreno et al. 2012, Randall and van Woesik 2015, van Woesik and Randall 2017), increases in rainfall and related nutrient runoff, and dredging-associated sedimentation (Sutherland et al. 2004, Haapkylä et al. 2011, Pollock et al. 2014). Moreover, the synergistic effect of both warming temperatures and an increase in nutrients and water turbidity can generate physiological stress in corals, and simultaneously increase proliferation of opportunistic pathogens and their virulence expression, which compromise the health state of corals (Lesser et al. 2007, Mydlarz et al. 2009, Pollock et al. 2014).

To date, most of our understanding of coral diseases is based on studies from shallow coral reefs (1–20 m depth). Studies on coral diseases outbreaks on mesophotic reefs (> 30 m depth) are scarce. Hickerson et al. (2008) reported that 12–15% of the corals on the Flower Gardens Banks National Marine Sanctuary were affected by white syndromes; Calnan et al. (2008) and Smith et al. (2008) reported that between 0 and 15% of the surveyed corals on mesophotic reefs of the U.S. Virgin Islands showed signs of a variety of diseases, including black band, dark spot, white disease and yellow blotch/band. In the U.S. Virgin Islands, coral cover can be three times higher on mesophotic than on shallow coral reefs (Smith et al. 2008). Moreover, cover of the important reef building species of the genera *Orbicella* is four times higher on mesophotic than on shallow reefs. Between 2001 and 2005, WP did not show any trend associated with depth (Smith et al. 2008, 2016a). However, after the 2005 bleaching event the prevalence of WP on mesophotic reefs tripled in comparison to shallow reefs, and coral cover has declined at both depths (Smith et al. 2016a). In monitoring up to 2010, WP accounted for 24% of coral diseases recorded across all depths by the Virgin Islands Territorial Coral Reef Monitoring Program, but WP represented 59% of disease cases recorded on mesophotic reefs (2002–2013) (Smith et al. 2015). Knowing that mesophotic reefs are also the target of white plague that can kill corals in short periods of time (Bythell et al. 2004, Smith et al. 2008), it is important to understand what environmental factors trigger outbreaks and if patterns of disease are similar among reef habitats. This information will be valuable to evaluate and predict future trends of coral diseases under climate change (Maynard et al. 2015), and to develop regional solutions that allow us to conserve coral reefs and the benefits they bring through tourism, recreation and fisheries (Brander et al. 2007, Hoegh-Guldberg et al. 2007).

White diseases or syndromes cause rapid tissue loss on corals worldwide (Bythell et al. 2004), and among them, white plague (WP) affects at least 35 species of Caribbean corals (Sutherland et al. 2004). WP outbreaks have produced a significant decline in coral cover in specific regions of the Caribbean, including following a mass bleaching event in 2005 in the northeast Caribbean (Cróquer and Weil 2009b). Three distinct types of WP have been recognized (WP Types 1, 2 and 3); while they all present similar lesions that originate from the base or margin of a colony and expand rapidly leaving white areas of bare coral skeleton, they presumably differ in the rates of tissue loss (Dustan 1977, Richardson et al. 1998, 2001), and WP Type III exclusively affects large coral colonies (3–4 m in diameter) of *Colpophyllia natans* (Houttuyn 1772) and *Orbicella annularis* (Ellis and Solander 1786, Richardson et al. 2001). However, rates of progression can change through the lifespan of a lesion, and the distinction of types may be arbitrary (Clemens and Brandt 2015).

The etiological agent of WP is still in debate; initial studies suggested WP type I was originally associated with bacteria (Dustan 1977), but recent studies where tissue loss rates matched rates reported for type I by Dustan (1977) point to a viral origin (Soffer et al. 2013). WP type II was at first reported to be caused by a novel bacterium, *Aurantimonas corallicida* (Proteobacteria, Aurantimonadaceae) (Denner et al. 2003, Nugues et al. 2004), but other studies did not find *A. corallicida* in corals with similar signs (Pantos et al. 2003, Sunagawa et al. 2009). WP type III has not been associated with any pathogen. Due to the uncertainty of the WP etiology and its association with environmental stressors, it has also been hypothesized that WP signs are a host response to environmental stress and do not represent an infectious disease (Lesser et al. 2007). However, WP disease signs were demonstrated to be transmissible from one coral to another via water transport and a coral predator vector in aquarium experiments (Clemens and Brandt 2015), and by direct contact between colonies in their natural environment (Brandt et al. 2013). Clustered spatial distribution of the WP disease in the natural environment in some cases suggest infection, with diseased colonies being concentrated within the first meter of other diseased colonies (Brandt et al. 2013). Another study, however, found that WP does not always follow a spatially clustered pattern and, thus, there is controversy in relation to the transmissibility of all white diseases that follow a similar etiology (Muller and van Woesik 2012).

The microbiome of corals affected by WP disease, (denominated white syndromes in the Pacific Ocean, Willis et al. 2004) is distinctive across oceans and coral species (Roder et al. 2014). Changes in the environment can modify the microbiome of corals and help bacteria proliferate that are harmless to their host in low values, but can become a threat under elevated temperatures, (Lesser et al. 2007, Bourne and Webster 2013). The epidemiology of WP disease outbreaks on shallow Caribbean reefs (0–20 m) and white syndromes in the Pacific have been related to abiotic factors including thermal stress leading to bleaching, and

fragmentation due to storms (Bruno et al. 2007, Brandt and McManus 2009, Cróquer and Weil 2009b, Miller et al. 2009, Brandt et al. 2013, Precht et al. 2016, Smith et al. 2016a), as well as to sediment and turbidity associated with offshore dredging (Pollock et al. 2014). As a biotic factor affecting WP occurrence, coral overgrowth with the macroalgae *Halimeda opuntia* L. (Chlorophyta, Halimedaceae) has been linked to WP in shallow reefs, as this alga can also serve as reservoir of the possible WP type II pathogen *A. coralicida* (Nugues et al. 2004).

Habitat models or species distribution models (SDM) are commonly applied to understand disease dynamics in conservation or agricultural settings (Meentemeyer et al. 2004, Guisan and Thuiller 2005, Panassiti et al. 2017). By correlating presence/absence records with environmental data, SDM's can also help researchers understand the ecological drivers of disease occurrence. Different types of SDM's have been used to understand disease dynamics within coral populations, including epidemiological models (Sokolow et al. 2009, Zvuloni et al. 2015), and environmental models. The latter models unravel the relationship between biotic and abiotic factors and the prevalence of diseases (Bruno et al. 2007), and include the use of negative binomial models (Bruno et al. 2007), partial least square regression models (Haapkylä et al. 2011), generalized linear mixed models (Ruiz-Moreno et al. 2012), and, most commonly, multivariate models (Cróquer and Weil 2009a, Aeby et al. 2011, Pollock et al. 2014, Lamb et al. 2016). Bayesian inference has been recently integrated into linear model analyses of coral diseases, specifically to understand spatial-temporal dynamics of diseases in relation to biotic and abiotic factors (Muller and van Woesik 2014, Muller et al. 2020), and to create disease risk models at a regional scale (van Woesik and Randall 2017).

The objective of this study was to better understand the abiotic and biotic factors associated with WP disease in shallow and mesophotic coral reefs of St Thomas, U.S. Virgin Islands. We analyzed biophysical and WP prevalence data from the U.S. Virgin Islands for three years (2012–2015) and developed generalized linear mixed-effect models (in a Bayesian framework) of disease dynamics across shallow, mid-depth and mesophotic coral reef habitats. Specifically, we addressed two main questions: 1) are WP disease prevalence temporal patterns (monthly, seasonal and annual) similar among shallow (6–15 m), mid-depth (20–21 m) and mesophotic coral reefs (30–40 m)?, and 2) what environmental (biotic and abiotic) factors are most important in promoting an increase in WP disease across coral reef habitats?

Material and methods

Study area

The prevalence of WP was studied across five shallow (6–15 m), three mid-depth (20–21 m) and five mesophotic (30–40 m) coral reef sites in St Thomas, U.S. Virgin Islands (18.0°N,

65.0°W, Fig. 1). The U.S. Virgin Islands are the most northeastern of the Lesser Antilles, bounded by the tropical western Atlantic to the north and the Caribbean Sea to the south. This region is swept by near constant northeasterly trade winds that brings advective energy and moisture from the Atlantic (Watts 1990). As in most of the Caribbean region, the rainy season spans from May to November, and it is bimodal by nature with a brief drier period in July, dividing it into an early rainy season (May–July) and a late rainy season (August–November). The latter season is associated with greater rainfall, and coincides with the most active part of hurricane season as well as with elevated oceanic and atmospheric temperatures. Rainfall in the region is influenced by the El Niño/La Niña phenomenon (Taylor et al. 2002). The dry season spans from December to April, and also tends to be the windiest season.

Data collection

White plague (WP) prevalence

Prevalence of WP on corals was recorded monthly or every two to three months (contingent on weather and logistical considerations, Supporting information). Shallow and mid-depth reef sites were surveyed between February 2012 and December 2013, while mesophotic reefs were sampled between February 2012 and February 2015. During each sampling period, WP prevalence on corals was recorded using drop camera surveys performed at 13 reef sites within 1–2 days. We employed the drop camera method in place of traditional in situ surveys in order to assess a large number of corals at multiple coral reef sites across different depths (6–40 m) within one day each month. This would have been unachievable in such a short period of time with traditional SCUBA-based benthic survey methodologies. The method consisted of using two cameras; a weighted drop video camera (Sea-Drop 950, SeaViewer Cameras Inc.) monitored from the surface for guidance and a high definition GoPro camera (Hero3) in an underwater casing facing down for recording (Supporting information). At each site, two wide view down-facing video transects were recorded for 3–5 min with the GoPro camera approximately 2–5 m above the reefs. Each drop-camera survey at a site was started at the same coordinates using a boat-mounted GPS and though there was some variability in the reef area that was assessed due the boat drift being influenced by waves, currents and wind, we targeted calm days with similar conditions for sampling. Researchers on the boat who were familiar with the sites were observing the reef while the video was being collected, and transects were redone if it became apparent that the boat was not drifting over the same general reef area that was assessed each time. As day-to-day differences in water current and weather conditions made it impossible to maintain the camera at the same depth for a consistent time period, the length of surveys was determined by researchers on board based on a visual estimate that the video had recorded 100–150 corals (300 corals/reef site). At low coral cover reef sites, this required a longer length of time (5 min) than at high coral cover reef sites (3 min). At

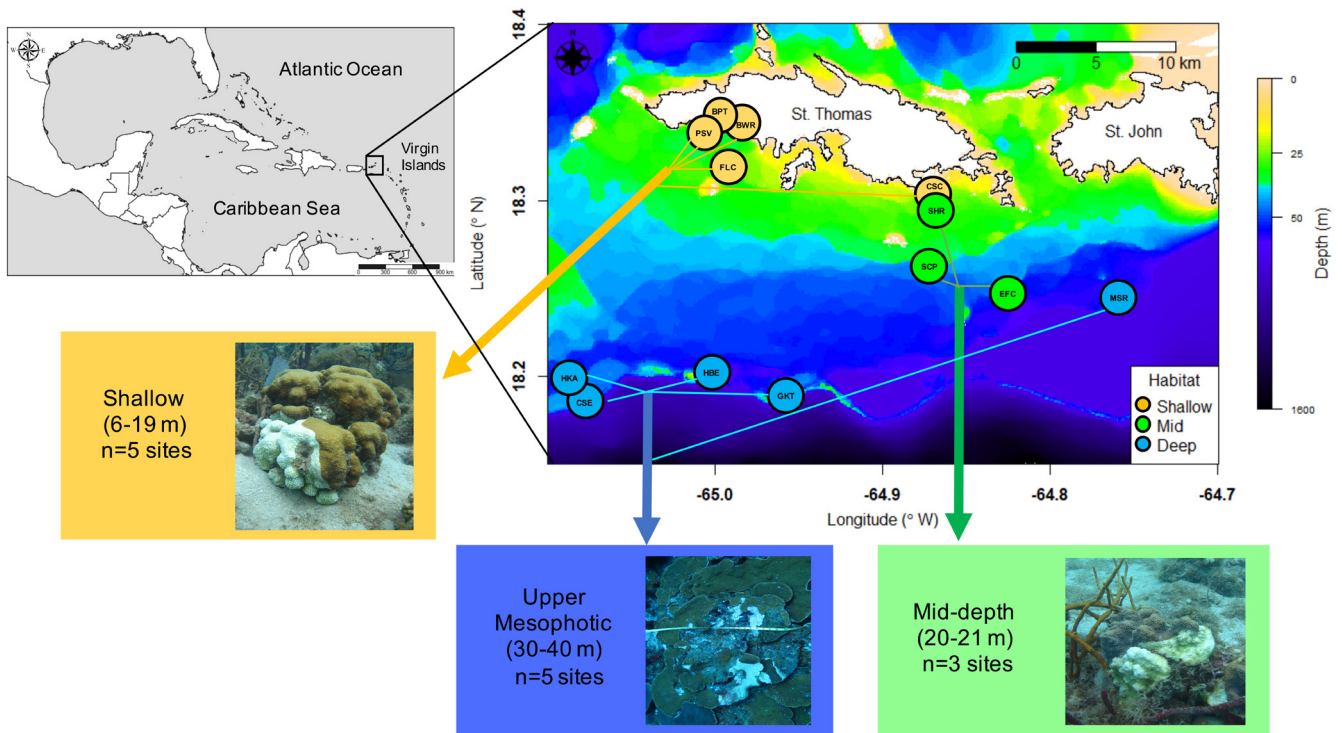


Figure 1. Map of the study area including 13 reef sites. In yellow are shallow sites (BPT: Black Point, BWR: Brewers Bay, CSC: Cas Cay Outer, FLC: Flat Cay, PSV: Perseverance Bay) in green mid-depth sites (EFC: East French Cap, SCP: South Capella, SHR: Seahorse), and in blue upper mesophotic reef sites (CSE: College Shoal, HBE: Hind Bank East, HKA: MCD S166, GRT: Grammanik Bank, MSR: Meri Shoal/South Fish). Photographs show cases of WP on *Orbicella faveolata* colonies at different depths.

some reef sites, the target of 300 corals was not reached due to logistical constraints, such as boat drift in a confined low coral abundance area that did not allow for non-overlapping transects (Supporting information). Overall, the same general reef area was assessed on each survey.

Videos were later analyzed by at least two observers. For each video transect, observers recorded and identified to species the number of corals that appeared completely within the frame of the video. The final count of the total coral colonies was estimated as a mean of the two observers. On average, counts differed by < 5% between observers. The videos were then reviewed again to identify the presence of WP lesions on the corals. WP lesions were defined following previous descriptions (Remily and Richardson 2006) as bright white areas of recently denuded skeleton defined from living tissue by a smooth, undulating margin. Lesions were only defined as WP lesions if they matched this description and occurred at the base or margin of the colony, which is a characteristic sign of WP (Bythell et al. 2004). The number of WP affected corals was confirmed by both observers.

All species of corals were counted and assessed for WP in the videos, but the most commonly affected species across all reef sites included *Orbicella annularis*, *O. franksi* and *O. faveolata*, which were also the most commonly found species across all sites (Supporting information). WP prevalence

data used in the analysis was therefore based only on disease found on the genus *Orbicella* (*Orbicella* spp. were combined in the data analysis), as lesions in other coral species were more difficult to identify. Other species identified with WP in the videos included *Agaricia agaricites*, *Dichocoenia stokesii*, *Siderastrea siderea*, *Porites astreoides*, *P. porites*, *Montastraea cavernosa* and *Colpophyllia natans*, but these species only accounted for 2% of the WP cases identified. Prevalence of disease was calculated as the number of WP affected *Orbicella* spp. corals divided by the total number of *Orbicella* spp., and expressed in figures as a percentage (disease *Orbicella* spp./total *Orbicella* spp. $\times$ 100). Other coral diseases were counted when they were observed but were rare and therefore not analyzed. These included black band disease (eight colonies affected), yellow band disease (eight colonies affected) and dark spot disease (seven colonies affected).

Environmental-abiotic variables

Conductivity-temperature-depth (CTD) profiles were taken immediately after or before video transects, except in the case of a few instances, when profiles were taken within 20 days of video transect sampling or not at all because of logistical difficulties. One CTD cast profile was obtained each sampling time at each station a Seabird SBE 25 sonde recording at 8 Hz and 16 Hz (Sea-Bird Electronics, Bellevue, WA,

USA) and secondary sensors, which also took measurements of oxygen concentrations, turbidity (fluorometric) and salinity of the seawater (Supporting information). Two different CTD units were used in different sampling periods. Two Wetlabs SN FLNTUB fluorometers – 618 and 401 affixed to one of the CTDs estimated water column chlorophyll concentrations (as a proxy for nutrients; Furnas et al. 2005). From each CTD cast profile, only the deepest 2 m of data registered over the reef was used and averaged for each reef site and sampling time point. Benthic temperature within each coral reef site was measured with a logging temperature probe (Hobo Water Temperature Pro V2, Onset Computer Corp., Bourne, MA, USA) and used to calculate monthly mean temperatures. Probes were set to measure with a frequency of 15–60 min and were swapped with different probes annually. Before and after usage, temperature probes were calibrated in a freshwater ice bath and ambient temperature bath, and probes were not deployed if their temperature deviated more than 0.3°C from that recorded with a bulb thermometer. Degree heating weeks (DHWs) were calculated using site-specific monthly maximum means as described for these sites in Smith et al. (2016a). DHWs represent the accumulation of coral temperature stress where temperatures have exceeded the monthly maximum mean for a given area; DHW values > 4.0°C-weeks are known to result in bleaching, and values > 8.0°C-weeks can result in mass bleaching and mortality (Liu et al. 2006, Kayanne 2017). However, these bleaching thermal thresholds can significantly vary with depth (Wyatt et al 2020), therefore we used site specific thresholds developed previously (Smith et al 2016a). Daily cumulative precipitation values (inches) were obtained from Charlotte Amalie, St Thomas, Cyril E. King International Airport weather archives. However, monthly average precipitation values had a moderate positive correlation with turbidity ($r=0.475$), therefore, only turbidity was included into analyses due to its ecological relevance for coral reefs (Supporting information). Turbidity not only reflects the effects of runoff due to precipitation, but also fine particle resuspension by wind and tides and other anthropogenic impacts (Fabricius et al. 2013).

Environmental–biotic variables

Benthic percent cover of all corals, *Orbicella* spp. (stony corals of the genus *Orbicella*, Dana 1846), sponges, macroalgae, cyanobacteria and sand were obtained annually from the U.S. Virgin Islands Territorial Coral Reef Monitoring Program (Smith et al. 2015). These data were estimated from high resolution video transects (captured by divers on SCUBA) using standardized methods on six 10 m² permanent transects at each site, and estimated visually from non-permanent transects in Perseverance Bay (one site with four transects by Henderson 2012) and MCD S166 site (two transects). Percent cover was averaged among transects per site. These transects were located at the same reef sites where WP prevalence was obtained monthly from drop-camera surveys. We adopted a combination of methods because it was not possible to standardize the estimate of benthic cover across

drop-camera surveys, therefore slightly different areas were covered on each survey. For instance, a three minute drop-camera video that allowed for the assessment of 100 corals at one site may have covered an area of 10 000 m² on one day, but because of variability of current, wind and waves, and the depth of the camera, the same length of video assessing 100 corals at the same site could have covered 12 000 m² on another day. Estimating changes in benthic cover accurately depends on knowing the total area assessed. Since this was not possible, we used data from TCRMP transects which consistently assess a known area (10 m × 10 cm per transect) each time. In addition, TCRMP surveys produce much higher resolution data for benthic cover than would be possible from the moving drop-camera videos, including the ability to distinguish among macroalgae types, cyanobacteria and sponges.

Statistical analyses

WP prevalence patterns (monthly, seasonal and annual)

To evaluate the effect of seasonality and account for thermal stress, four seasons were established combining the span of the rainfall season (Taylor et al. 2002) and the monthly seawater maximum temperatures (Smith et al. 2016a): 1) dry season (December–March), 2) transition to rainy season (April–May), 3) rainy season (June–August) and 4) rainy with heat stress (September–November). As is common for ecological studies, and disease studies in particular, our data did not meet the assumptions of normality and homogeneity of variances. Therefore, monthly, seasonal and annual fluctuations of WP prevalence on reef corals at different depths were analyzed by ranking WP prevalence data and running ANOVAs (as linear mixed effect models) on the ranked data. Statistical Analysis was performed in R (v3.5.0; <www.r-project.org>) using *lme4* ver. 1.1-26 (Bates et al. 2015) and *lsmeans* ver. 2.30-0 (Lenth 2016).

WP relation to the environment

To estimate the dependence of WP prevalence on environmental conditions, we developed WP disease distribution models with two sets of predictors, 1) water column or monthly – abiotic variables and 2) benthos or annual – biotic variables (Supporting information – white plague model) based on Bayesian inference and using R software (<www.r-project.org>) and Stan (<<http://mc-stan.org>>), according to Panassiti et al. (2015). Prior to model fitting, we ran pairwise Spearman's correlation tests on scaled variables to address the issue of multicollinearity (Graham 2003, Supporting information). In case of Spearman's $r > 0.7$, the selection among correlated variables was based on their ecological relevance (Guisan and Zimmermann 2000). The final set of explanatory abiotic and biotic environmental variables included in the analysis is shown in Table 1. The water-column model included only abiotic variables that did not correlate among each other (chlorophyll, depth, DHW, oxygen, salinity, temperature, turbidity), obtained at a monthly frequency and simultaneously with video-transects for WP prevalence (Table 1). The benthos model included all the benthos

variables: percent cover of coral, cyanobacteria, macroalgae, sponges and sand that were obtained annually (Table 1). To account for different scale units, all environmental and biotic variables were scaled and centered (i.e. mean subtracted and divided by the standard deviation).

To model WP prevalence monthly and annually, we built two logistic regression models, special cases of a generalized linear model, by specifying a binomial error distribution and a logit link function (Hosmer and Lemeshow 2000). The logistic regressions were evaluated in a Bayesian framework. Fixed parameter priors were chosen to be mildly informative: normally distributed, 0-centered and with an intermediate standard deviation (10). This prior specification causes a parameter shrinkage, similar to a ridge regression (Park and Casella 2008). Additionally, we accounted for extra variance in the response by specifying an observation-level random factor. This random effect prior was specified as a flat prior, normal distribution with a mean of zero. We introduced an additional level of uncertainty for the variance parameter of this random effect prior by using a flat inverse gamma hyper prior with shape α and scale β (0.001, 0.001). The posterior was estimated using the No-U-Turn Markov Chain Monte Carlo (MCMC) algorithm which is implemented in the Stan software (Hoffman and Gelman 2014, Stan Development Team 2017). For each model, we ran four separate MCMC chains with 1000, 4000 and 30 000 iterations (obtaining same results). Convergence of MCMC chains was checked visually and by assuring that the potential scale reduction factor R_{hat} was below 1.05 (Gelman et al. 2013). For each parameter, we provide posterior medians and 80% credible intervals to summarize marginal posterior distributions. Spatial and temporal autocorrelation was checked using the DHARMA package (Hartig 2017). The package performs a Moran's I and Durbin-Watson test for spatial and temporal autocorrelation, respectively. Given non-significant test results and no visual pattern in the residuals, we found no indication of either spatial or temporal autocorrelation. To assess model fit, standardized residuals (referred also as 'Bayes p-values', Gelman et al. 1996) were calculated using the DHARMA R package (Hartig 2017), and visualized on a map. Standardized residuals were created by comparing the expected values from the fitted model (posterior predictive simulations) to the observed values. A residual of 0.5 indicated that the observation was in the median of the posterior predictive simulation (perfect model fit), whereas residual values above 0.5 indicated that the model was over-fitting, and below 0.5 that the model was under-fitting.

Results

Monthly, seasonal and annual patterns of WP disease

WP was found at all sampled reef sites. Average WP prevalence varied significantly by depth and was higher at deeper reefs (upper mesophotic ≥ 30 m), than on shallow

and mid-depth reef sites ($F=106.18$, $df=1$, $p < 0.05$, Fig. 2, Supporting information). When evaluating the possible combined effects of depth and time (year, season and month), depth and all the interactions of time with depth were significant (Supporting information). WP prevalence was significantly higher in 2014 and 2015 ($p < 0.05$, Fig. 2b, Supporting information). Also, WP prevalence tended to be higher during the rainy and rainy+heat stress seasons (Fig. 2c, Supporting information), but was only significantly higher during the rainy season (specifically in June 2014) and lower during the dry season (specifically in February and March of 2012 and 2013, Fig. 2b, Supporting information).

Influence of environmental factors (biotic and abiotic) on WP disease

Water column abiotic factors (monthly level model)

The abiotic factors depth, turbidity, temperature, chlorophyll and DHW positively affected the prevalence of WP on corals (80% credible intervals deviate from zero, Fig. 3a). Among those, the model indicated that depth (interval median = 2.23), turbidity (interval median = 0.38) and temperature (interval median = 0.40) were the factors with the highest positive influence on WP disease occurrence on St Thomas reefs. Simultaneously, oxygen (interval median = -0.28) and salinity (interval median = -0.40) negatively affected WP prevalence (Fig. 3a). Monthly average values for each variable can be found in Supporting information.

Benthic biotic factors (annual level model)

Annual biotic factors that positively affected the prevalence of WP on corals, across all depths, included percentage cover of sand (interval median = 0.60), coral (interval median = 0.80), macroalgae (interval median = 0.31) and cyanobacteria (interval median = 0.39). Of these, coral cover (all species of corals, with 38–95% *Orbicella* spp. as the dominant group, Supporting information), had the highest positive influence on WP prevalence (Fig. 3b). In contrast, reduced percentage cover of sponges (interval median = -0.32) was associated with an increase of WP prevalence. Although we built this model including the environmental factors, we do not analyze the environmental factors at the annual level, as environmental effects on WP are explained at a finer resolution on the monthly model (better ecological scale).

Model fit

Comparisons of model residuals with simulated residuals showed an accurate fit of the water column (monthly model) with only slight over-fitting or under-fitting for most stations (0.45–0.55). We consider this model to be accurate. The benthic (annual model) fitted the biotic and abiotic factors well for most reef sites; only three sites (Grammanik Bank, Seahorse and South Capella) showed a higher average trend to under- or over-fitting (< 0.2 or > 0.8). Altogether, systematic autocorrelation and pseudo-replication over all stations were not found, suggesting both models fit simulated residuals properly (Fig. 3c–d, Supporting information).

Table 1. Description of response and environment (abiotic and biotic variables) used to fit white plague disease (WP) models. Percentage of sand was the only abiotic variable quantified annually, and included in the annual model (adding this variable to the monthly model would not be informative).

Data type	Unit	Sampling scale	Description	Source description	Reference
Response					
WP predictions	Prevalence	Monthly	no. disease corals / no. total corals	Estimated from video-transects	(Raymundo et al. 2008)
Abiotic explanatory					
Water column					
Chlorophyll	Mg m ⁻³	Monthly	Light emitted by chlorophyll, calculated as concentration	EcoFLNT fluorometer	http://www.seabird.com/sbe25plus-seallogger-ctd
Depth	m	Monthly	Calculated based on the pressure measured by transducer	Shearwater Petrel diving computer at each reef site	https://www.shearwater.com/products/petrel-2/#overview
Degree heating weeks (DHW)	°C	Monthly	Measure of thermal stress accumulation	Logging temperature probe	https://coralreefwatch.noaa.gov
Oxygen	mg ml ⁻¹	Monthly	Dissolved oxygen in the seawater	SBE 43 Dissolved Oxygen Sensor	http://www.seabird.com/sbe25plus-seallogger-ctd
Salinity	Practical Salinity Units (PSU)	Monthly	Concentration of dissolved salts in water	Seabird SBE 25	http://www.seabird.com/sbe25plus-seallogger-ctd
Temperature	°C	Monthly	Average thermal energy of the seawater	Logging temperature probe	http://www.onsetcomp.com/products/data-loggers/u22-001
Turbidity	Nephelometric Turbidity Units (NTU)	Monthly	Cloudiness or haziness of seawater caused by solid particles	EcoFLNT fluorometer	http://www.seabird.com/sbe25plus-seallogger-ctd
Benthos					
Sand	% cover	Annual	Average percent of sand	six 10 m TCRMP transects	(Smith et al. 2015)
Biotic explanatory					
Benthos					
Coral	% cover	Annual	Average percent of live coral	six 10 m TCRMP transects	(Smith et al. 2015)
Cyanobacteria	% cover	Annual	Average percent of cyanobacteria	six 10 m TCRMP transects	
Macroalgae	% cover	Annual	Average percent of macroalgae	six 10 m TCRMP transects	
Sponges	% cover	Annual	Average percent of sponges	six 10 m TCRMP transects	

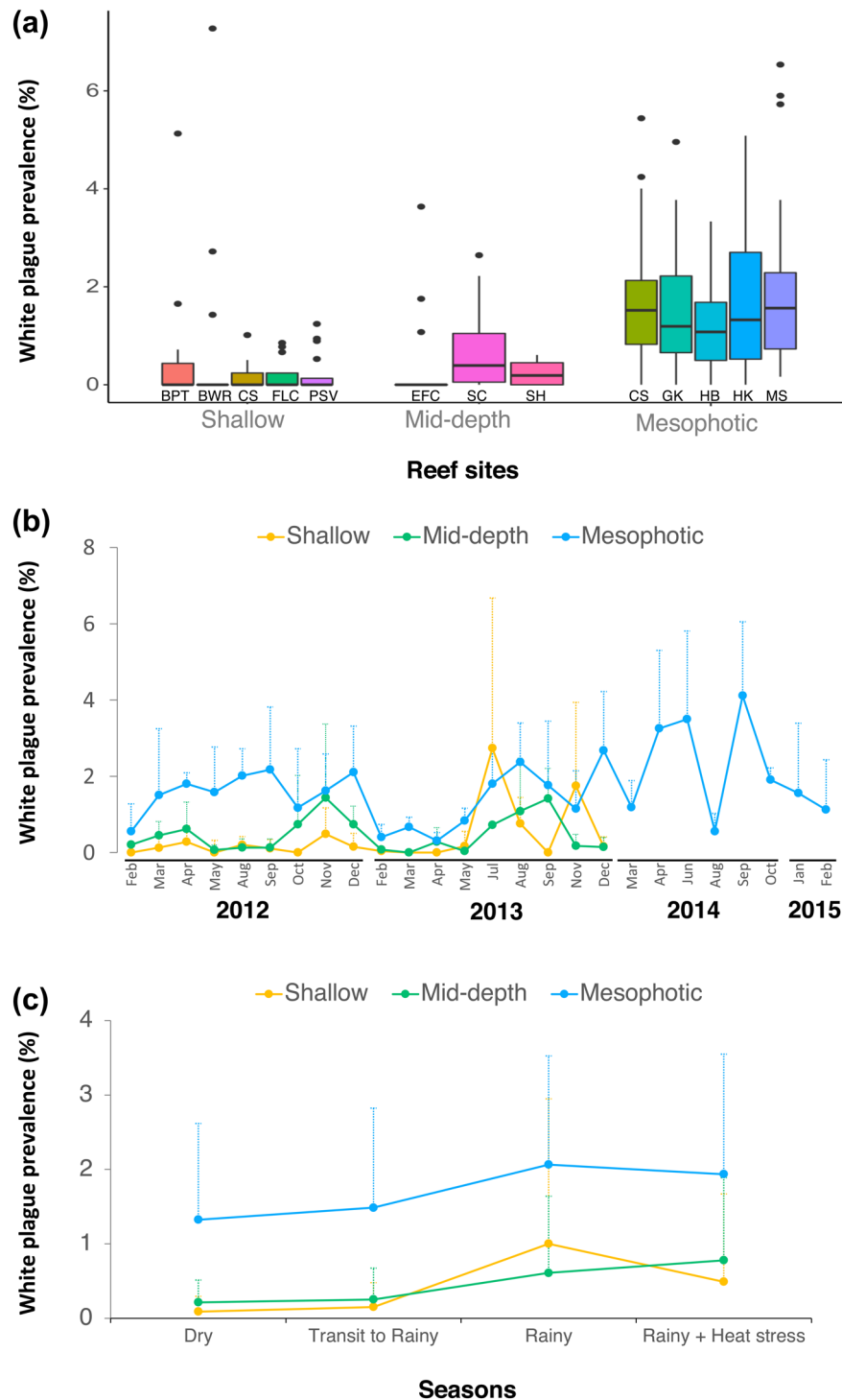


Figure 2. Average ($\pm$ SD) patterns of WP for each depth (shallow = 6–12 m, mid-depth = 20–21 m, mesophotic = 30–42 m). (a) Annual median with interquartile ranges per station and depth (outlier values are represented by black dots), (b) per month and year, (c) per season (all reef sites, months and years combined per depth). Site codes and names as in Fig. 1.

Discussion

Our results suggest consistent temporal patterns of WP disease prevalence that varied across depth and were associated with a suite of physical and biological variables. As was

suggested by previous research in the U.S. Virgin Islands (Calnan et al. 2008, Smith et al. 2008), WP prevalence was higher on mesophotic than on shallow or mid-depth reefs. WP prevalence reached its maximum across all reef depths during the rainy seasons (here referred as rainy season:

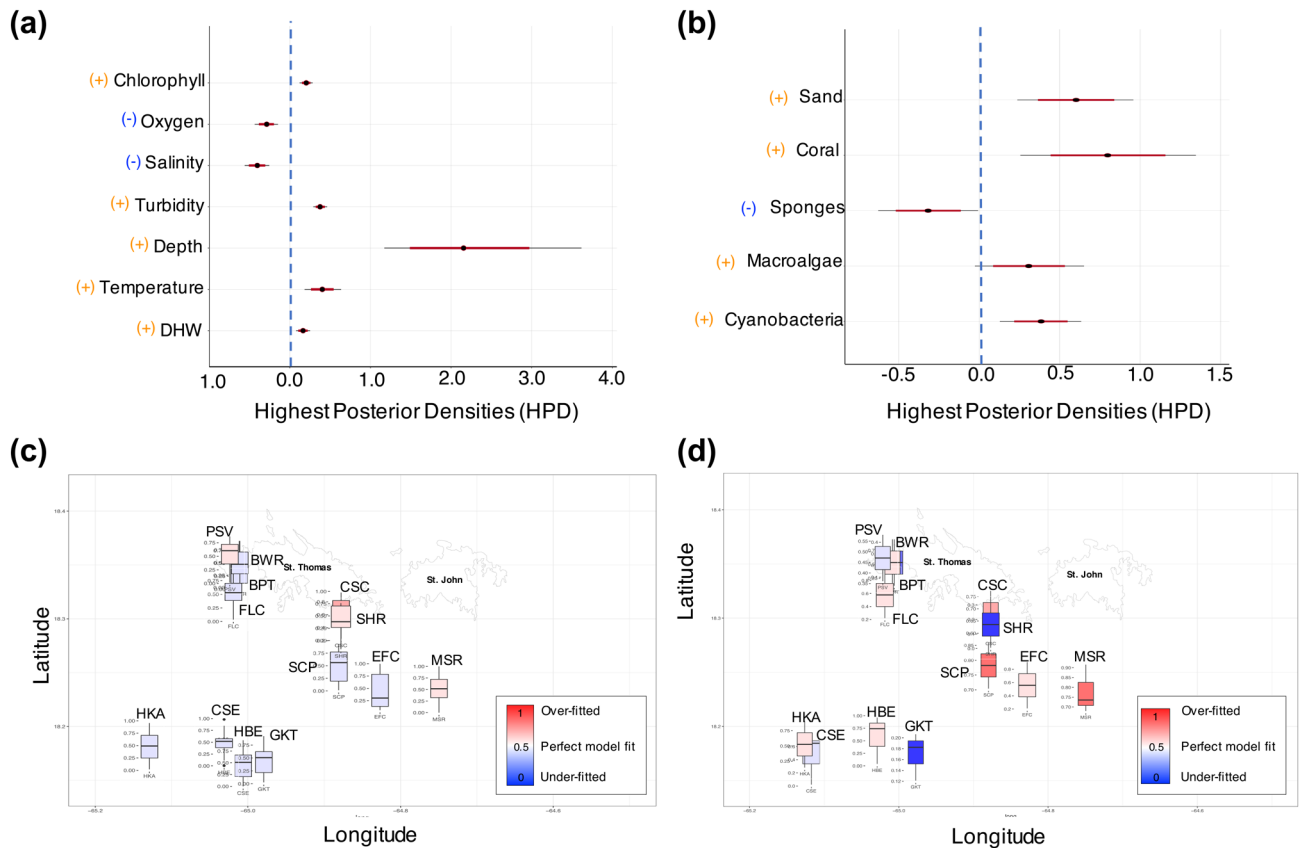


Figure 3. Marginal posterior distributions of (a) water column abiotic predictors (monthly interval), and (b) benthic biotic and abiotic predictors (annual interval). The length of the red line indicates the 80% credible interval, the length of the black lines indicates the 95% credible interval, and positive and negative values on the y-axis indicate a positive or negative effect for each variable. Standardized residuals (Bayesian p-values) for the two models c) monthly (water column abiotic) and (d) annual (benthic biotic and abiotic); values of 0.5 indicates that the observation is in the median of the posterior predictive simulation (perfect model fit). Reef site codes and names as in Fig. 1, specific variables units are in Table 1; (+): positively correlated with disease; (–) negatively correlated with the disease.

June–August, and rainy + heat stress: September–November). Our results suggested that higher turbidity and higher temperature stress in the rainy season were associated with the increased WP disease prevalence. In addition, higher coral cover of the highly susceptible species (*Orbicella annularis*, *O. faveolata* and *O. franksi*) with increasing depth was associated with higher WP prevalence on mesophotic reefs.

The monthly and annual Bayesian models allowed us to evaluate multiple environmental, biotic and abiotic drivers of WP across shallow and mesophotic coral reefs. The effect of these factors on the increase of WP prevalence is discussed here within the context of 1) host density and species susceptibility, 2) potential pathogen reservoirs and transmission mediums and 3) environmental drivers (Fig. 4).

Host density and species susceptibility

Why are mesophotic reefs being affected to a higher degree by WP disease? Our results showed a higher percent cover of *Orbicella* spp. was associated with higher levels of WP prevalence. The mesophotic reefs of the U.S. Virgin Islands are characterized by a higher coral cover, primarily of *Orbicella*

spp., than shallow and mid-depth reefs (Smith et al. 2008, 2016b). Corals of the *Orbicella* genus are more susceptible to white plague compared with other abundant species, as demonstrated in controlled laboratory experiments (Williams et al. 2020). Thus, it is possible that higher densities of *Orbicella* spp. on mesophotic reefs may be increasing the net transmission of the disease. Affected corals in a dense population are closer to each other (though rarely in direct contact), possibly facilitating the spread of WP, as reported for other diseases in dense animal populations (Scott 1988). It is also possible that WP prevalence is higher on mesophotic reefs because coral colony size (host size) tended to be larger with increasing depth (Supporting information). Larger colonies might provide a larger area for potential pathogens to encounter or larger colonies may be affected by the disease for a more extended period than small colonies (Caldwell et al 2018), allowing us (the observers) to register this disease more often on deeper sites. However, size distributions overlapped among sites within different depths (Supporting information). Also, the average sizes of corals within sites assessed at each time point likely did not differ from survey to survey (since the same area was surveyed each time and coral sizes

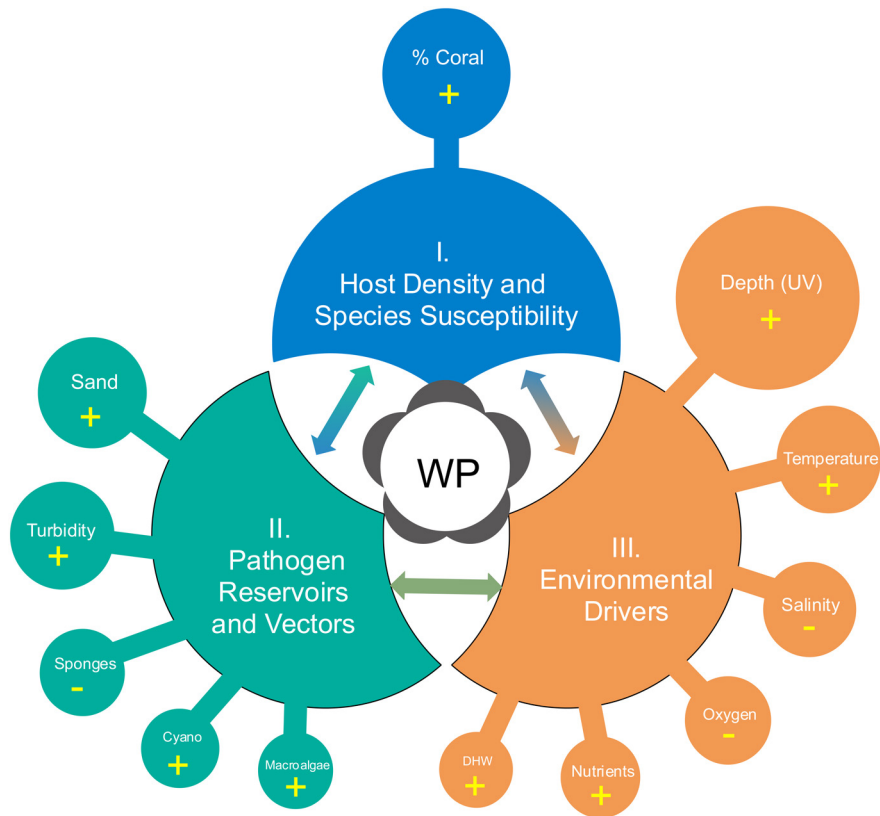


Figure 4. A conceptual model with multiple environmental and biological factors for WP disease to occur; (+): positively correlated with disease; (–) negatively correlated with disease. Factors are divided into three categories: 1) host density and species susceptibility, 2) potential pathogen reservoir and transmission mediums/vectors and 3) environmental drivers. Each bubble represents a factor, and bubble size indicates the relative influence of each factor on WP prevalence (based on the marginal posterior distributions from the Bayesian models).

had low variability from survey to survey within a site), and at least 300 corals were assessed upon nearly every survey. These observations support that temporal disease trends and disease differences among depths reflect true differences in disease and not just changes in the size distributions of corals assessed.

In addition to higher population densities, poor nutrition can also increase the risk of disease (Scott 1988). In the U.S. Virgin Islands, *O. faveolata* exhibits a lower calorimetric energy content (an indicator of total energy within the coral holobiont) on mesophotic versus shallow reefs during reproductive months (August or September), possibly do to intense reproductive activity over a short period of time (Brandtneris et al. 2016). Lower energy content may increase the susceptibility of this species to WP disease before or during reproductive months, and could modulate the higher WP prevalence found on mesophotic reefs during the rainy season (June–August) and rainy with heat stress (September–November) seasons.

Potential reservoirs and transmission mediums

Higher percent cover of sand, macroalgae and cyanobacteria were also related to an increase in WP prevalence. Sediment can act as a reservoir for possible pathogens (Goyal et al.

1977), and contact with sediment has been associated with WP during outbreaks (Brandt et al. 2013). Dramatic increases in the macroalgae *Dictyota* spp. have also been associated with outbreaks of WP in south Florida (Brandt et al. 2012). Macroalgae and cyanobacteria have the potential to trap sediment as well as be a source of microbes (Fabricius 2005, Charpy et al. 2012). In fact, shifts in the bacterial communities of corals have been associated with macroalgae contact (Morrow et al. 2013) and microbial diversity and activity on reefs has been linked to overall macroalgae abundance (Dinsdale et al. 2008, Haas et al. 2010). Also, although not abundant on reefs of the U.S. Virgin Islands, the macroalga *Halimeda opuntia* has been identified as a reservoir for the white plague type II pathogen, *A. corallicida* (Nugues et al. 2004). Therefore, sediment and macroalgae could be serving as reservoirs or mediums of transmission for a WP pathogen. Competitive interactions with macroalgae and cyanobacteria may also be weakening corals and making them more susceptible to diseases, a potential indirect linkage between algal abundance and WP (Brandt et al. 2012).

Conversely, higher percent cover of sponges was associated with less disease. The most obvious explanation for this relationship is that the presence and abundance of sponges is decreasing pathogen loads by removing pathogens through filter feeding, and possibly concentrating them in their

choanosome ('layer' of internal cells) (Negandhi et al. 2010) or phagocytizing them (Fu et al. 2006, Maldonado et al. 2010). Alternatively, sponges could be acting indirectly to affect coral susceptibility to disease or pathogen abundances through their effect on water quality or species interactions resulting in positive or negative outcomes for corals (López-Victoria et al. 2006, Chaves-Fonnegra and Zea 2011, González-Rivero et al. 2016). To our knowledge, the relationship between sponges and coral disease found here has not been identified, and should be investigated further.

Our results support turbidity as an important factor in WP prevalence. Similarly, increases in prevalence of white syndrome in Australia was associated with sediment plumes and turbidity from dredging (Pollock et al. 2014). Seawater turbidity can be the result of plankton and suspended solids, including silts, clays, sewage and industrial waste that originate from runoff driven by local rainfall (Fabricius 2005). Although a specific WP pathogen has not been determined for WP type I and III, or confirmed for WP type II, runoff initiated by local rainfall that adds particulates to the water column could provide additional surfaces for pathogen attachment and therefore contribute to greater pathogen dispersal and disease incidence (Goyal et al. 1977, Simon et al. 2002, Peduzzi and Luef 2008). This mechanism of pathogen dispersal has been previously suggested for some human gastrointestinal pathogens such as coliforms, fecal coliforms and *Salmonella* (Goyal et al. 1977). Simultaneously, suspended solids could act as shields and even protect pathogens from ultraviolet (UV) irradiation (Mamane 2008), allowing successful proliferation from shallow into mesophotic reefs. In our previous work in the U.S. Virgin Islands, we found that WP was associated with mechanical damage and direct contact with sediment initiated by storm effects, but that its spatial distribution was also clustered and indicative of potential secondary infectious spread (Brandt et al. 2013), possibly by water transport (Clemens and Brandt 2015). It is possible that solid particles in the water may play a role in dispersal of a WP pathogen.

Environmental drivers

Results of the analysis between disease and environmental factors showed that elevated seawater temperature was associated with increases in WP, and that this relationship was stronger on mid-depth and mesophotic than on shallow coral reefs. Thermal stress (as measured by DHW) was associated with increase in WP only on mid-depth reefs (Supporting information). These observations are in line with previous studies from the U.S. Virgin Islands that documented outbreaks of WP following a mass bleaching event in 2005 that was driven by thermal stress (Smith et al. 2016a), and other more recent but spatially limited outbreaks of WP that have also been associated with thermal stress (Brandt et al. 2013). However, WP has also been reported as affecting reefs of St John and Puerto Rico year round (Miller et al. 2003, Weil et al. 2009). While our results showed a year round prevalence of WP on mesophotic reefs, prevalence of WP on mid-depth and

shallow reefs occurred mainly during the rainier and warmer periods of the year. WP prevalence was maintained in mesophotic depths during the combined rainy and heat stress season (September–November) when maximum temperatures reached 28.4°C (0.0 DHW) and 29.4°C (6.7 DHW), respectively. However, on shallow reefs, WP declined to zero or near to zero during September and October, the peak of the rainy + heat stress season, and this occurred when maximum temperatures were higher than on mesophotic reefs (29.6°C), but accumulated thermal stress as measured by DHW was lower (2.9 DHW). Disease in mid-depth reefs was consistently elevated during the rainy and rainy + heat stress seasons, but maximum temperatures were lower (29.3°C), and thermal stress in this environment was similar to shallow reefs (2.7 DHW). The higher maximum temperature recorded in shallow reefs concurrent with a drop in white plague prevalence may indicate a possible upper temperature threshold for the development of WP disease. Specifically, temperatures reaching 29.6°C on shallow reefs might be inhibiting the development or persistence of WP disease on corals. In support of this hypothesis, white disease outbreaks (including white plague and other similar diseases), associated with the 2005 bleaching event on shallow and mesophotic reefs in the U.S. Virgin Islands started in the months after thermal stress abated and temperatures fell below 29.5°C, whereas the prevalence was near 0 during the event when temperatures were greater than 29.5°C (Smith et al. 2013, 2016a). The putative pathogen of white plague type II, *Aurantimonas corallicida*, has a temperature range that extends to 45°C, and an optimal growth temperature of 35°C (Denner et al. 2003, Remily and Richardson 2006), far above the temperatures that we observed. However, the WP we observed may not be associated with *A. corallicida*, as tissue loss rates of WP in St Thomas have been more closely aligned with those recorded for white plague type I (Brandt et al. 2013) and may be associated with a unique viral community (Soffer et al. 2013). Clearly, the relationship between WP disease and temperature is more complex than just a positive linear relationship, and should be explored in more detail. Generalized linear mixed models (GLMMs) also support this last statement, as temperature was the only variable that was not statistically significant, while all other estimates were similar in direction and magnitude to the ones obtained with the Stan Bayesian models (Supporting information).

Potentially intertwined with elevated turbidity and temperature, higher levels of nutrients (as chlorophyll), lower salinity and lower dissolved oxygen were also associated with higher WP prevalence. Low salinity can be indicative of freshwater inputs from local rainfall and coastal runoff that deliver additional nutrients and particulates that affect turbidity. This hyposaline (rainfall) environment may promote viral outbreaks on corals (Correa et al. 2016), and hence, supports the hypothesis that the WP pathogen might be viral (Soffer et al. 2013). In this study, disease tended to increase during the rainy and rainy + heat stress seasons, coinciding with periods of high local rainfall and also with the influence of waters from the Amazon river and the Orinoco river

plumes, respectively, which are carried to the north east Caribbean Sea (Corredor and Morell 2001). Simultaneously, a more turbid and nutrient rich environment may promote pathogen growth and/or decrease host resistance (Vega Thurber et al. 2014). Previous studies have found that seawater nutrient enrichment can both increase the prevalence of disease in coral populations (e.g. dark spot on *Siderastrea siderea*, Vega Thurber et al. 2014) and the severity of disease on individual corals (e.g. aspergillosis on *Gorgonia ventalina*, yellow band on *Orbicella*-previously *Montastraea annularis*, Bruno et al. 2003 and black band on *Siderastrea siderea*, Voss and Richardson 2006). In addition, higher levels of turbidity can decrease sunlight penetration, which can ultimately decrease the rate of photosynthesis and, therefore, levels of oxygen in seawater (Talke et al. 2009). Deeper corals could be more sensitive to shading caused by turbidity, which can affect the rate of photosynthesis by zooxanthellae, ultimately negatively affecting calcification and reproduction (Fabricius 2005). The *Orbicella* dominated mesophotic reefs studied here are near the maximum depth limits of the species in the U.S.V.I. (Smith et al. 2016a, 2016b), and exhibit periods of low caloric content as mentioned above (Brandtneris et al. 2016), and very low growth rates (Groves et al. 2018), suggesting these corals may be near their phototrophic compensation point and susceptible to any factor that decreases light. Thus, shading (due to turbidity) could also potentially negatively affect coral resistance to disease as indicated by our model (Fig. 4). Simultaneously, lower UV intensity may be playing a role in the increased abundance of WP on mesophotic reefs, where attenuated light intensity could shield pathogens from ultraviolet (UV) irradiation (see previous section). In our study, changes in turbidity, nutrients and dissolved oxygen caused by an input of freshwater either through runoff or the delivery of Amazon or Orinoco current water may ultimately have impacted both the coral's resistance to disease and/or the growth and virulence of the WP pathogen(s). More research is needed to prove this hypothesis and further disentangle these effects.

Drop camera method to assess WP and other coral reef diseases

Finally, the drop camera method applied in this study was simple, easy to use and did not require trained scientific divers, which was particularly useful for mesophotic sites which would have necessitated technical diving. The recording of video surveys of the approximately 300 corals at each of the thirteen study sites which were distributed over an area of approximately 66 km² was completed each month in a single day of field work. Normally, a one to two-hour dive would be needed to assess as many corals at one site. The drop-camera method therefore allows for the widespread assessment of large areas of coral reef in a fraction of the time. This is very advantageous for a disease like WP, which has been observed to increase to outbreak levels and then decline to near nothing in less than two months (Brandt et al. 2012, 2013). Equipment needed included a forward facing drop-camera that was used

to observe the reef in real time and a GoPro used to record videos of the benthos. At the time, this equipment combined totaled less than \$2000 USD. Drawbacks of the drop-camera method include the time needed for post-processing of videos, and issues typical of remote sensing. These included the potential for missing small coral colonies or small lesions, and the inability to identify coral diseases that may have characteristics difficult to see from more than several feet away, such as dark spot disease. However, the large white lesions appearing on the margins of corals which are characteristic of WP made this disease an ideal target for this method. We suggest that the drop camera method could also be useful for quantifying other visually dramatic conditions of corals, such as bleaching, or other rapid tissue loss diseases where large white lesions are characteristic, including the recently emergent stony coral tissue loss disease, or SCTLD (Muller et al. 2020). As coral reefs face increasingly stressful conditions, having fast and reliable methods to assess their condition are needed.

Conclusion

WP prevalence was higher on upper mesophotic than on mid-depth and shallow coral reefs, most likely due to high densities of highly susceptible species (*Orbicella* spp.) in mesophotic habitats (Fig. 4). The relationship between host abundance and disease prevalence observed in this study combined with our previous work demonstrating transmission of WP in laboratory experiments (Williams et al. 2020) supports that WP in the U.S. Virgin Islands is an infectious coral disease. However, WP disease prevalence was also associated with several environmental factors. Across habitats, disease prevalence tended to be higher during the rainy season and lower during the dry season, possibly due to changes in nutrients, salinity and turbidity affecting pathogen abundance and virulence or increasing host susceptibility (Fig. 4). Temperature was also positively associated with disease across habitats, as has been identified elsewhere, but our results suggested an upper thermal limit to WP, above which disease abated. The area of sand, cyanobacteria and macroalgae at a reef site positively influenced WP, possibly by acting as reservoirs or vectors for pathogens or pathogenic material. In contrast, the abundance of sponges was negatively associated with disease, possibly through positive influences on host susceptibility or through the removal of pathogens through filtration. These results combined suggest that WP is a dynamic disease driven by complex interacting relationships between diverse coral hosts, potential pathogen(s) and the heterogeneous reef habitats.

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Andia Chaves-Fonnegra: Conceptualization (equal); Data curation (lead); Formal analysis (lead); Methodology (equal); Validation (equal); Writing – original draft (lead); Writing – review and editing (equal). **Bernd Panassiti:** Formal analysis (supporting); Writing – review and editing (equal). **Tyler Smith:** Conceptualization (equal); Funding acquisition (equal); Investigation (equal); Methodology (equal); Writing – review and editing (equal). **Elizabeth Brown:** Data curation (supporting); Investigation (supporting); Methodology (supporting). **Elizabeth Clemens:** Data curation (supporting); Investigation (supporting); Methodology (supporting). **Moriah Sevier:** Data curation (supporting); Investigation (supporting); Methodology (supporting). **Marilyn Brandt:** Conceptualization (equal); Funding acquisition (equal); Investigation (equal); Methodology (equal); Project administration (lead); Resources (lead); Supervision (lead); Validation (equal); Writing – review and editing (equal).

Transparent Peer Review

The peer review history for this article is available at <<https://publons.com/publon/10.1111/ecog.05527>>.

Data availability statement

Data and code is available in the Supporting information (PDF file), the r-code is hosted on GitHub and the data is on figshare (<<https://figshare.com/>>).

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