

Disease dynamics of *Porites* bleaching with tissue loss: prevalence, virulence, transmission, and environmental drivers

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ABSTRACT: The prevalence, number of species affected, and geographical extent of coral diseases have been increasing worldwide. We present ecological data on the coral disease *Porites* bleaching with tissue loss (PBTL) from Kaneohe Bay, Oahu (Hawaii, USA), affecting *P. compressa*. This disease is prevalent throughout the year, although it shows spatio-temporal variability with peak prevalence during the warmer summer months. Temporal variability in disease prevalence showed a strong positive relationship with elevated water temperature. Spatially, PBTL prevalence peaked in clearer waters (lower turbidity) with higher water flow and higher densities of parrotfish, together explaining approximately 26% of the spatial variability in PBTL prevalence. However, the relatively poor performance of the spatial model suggests that other, unmeasured factors may be more important in driving spatial prevalence. PBTL was not transmissible through direct contact or the water column in controlled aquaria experiments, suggesting that this disease may not be caused by a pathogen, is not highly infectious, or perhaps requires a vector for transmission. In general, PBTL results in partial tissue mortality of affected colonies; on average, one-third of the tissue is lost. This disease can affect the same colonies repeatedly, suggesting a potential for progressive damage which could cause increased tissue loss over time. *P. compressa* is the main framework-building species in Kaneohe Bay; PBTL therefore has the potential to negatively impact the structure of the reefs at this location.

KEY WORDS: *Porites compressa* · Tissue mortality · Branching coral · Framework species · Coral reef ecology · Etiology · Coral disease · Water temperature

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INTRODUCTION

Diseases have the potential to severely alter the structure and function of marine ecosystems (Ward & Lafferty 2004). In some species, such as the long-spined sea urchin in the Caribbean (Lessios 1988) and the abalone in California, USA (Lafferty & Kuris 1993), infectious diseases have reduced population densities to such an extent that recovery is uncertain (Lafferty et al. 2004). The same is true for coral dis-

eases. Coral disease outbreaks have caused declines in coral cover in many reef systems (Nugues 2002, Croquer et al. 2005, Bruckner & Hill 2009). For example, disease outbreaks in the Caribbean have caused a severe reduction in the abundance of the 2 previously most abundant hard corals, *Acropora palmata* and *A. cervicornis*, causing a shift in the reef community structure (Gladfelter 1982, Aronson & Precht 2001, Patterson et al. 2002). Even though the Indo-Pacific appears to be less affected by coral diseases

than the Caribbean, an increasing amount of evidence suggests that coral diseases are common (Sutherland et al. 2004, Willis et al. 2004, Raymundo et al. 2005), even at remote, uninhabited islands (Williams et al. 2008, 2011b, Vargas-Angel 2009), with the types of diseases and their prevalence varying across multiple spatial scales (Aeby et al. 2011a,b). In fact, the geographical extent, number of species affected, and incidence of new diseases are increasing globally (Harvell et al. 1999, Ward & Lafferty 2004, Sokolow 2009). Environmental stress, shifts in virulence of existing pathogens, introduction of novel pathogens from anthropogenic activities, and global climate change are associated with this increase (Harvell et al. 1999, 2004, Sokolow 2009).

Coral disease prevalence can be expected to show intricate interactions with a variety of driving factors (Williams et al. 2010, 2014). For example, an increase in temperature can lead to an increase in pathogen virulence or cause stress to the host, which can increase its susceptibility to disease (Harvell et al. 2002). Coral disease outbreaks and increases in disease prevalence and progression have been linked to various environmental factors (e.g. Bruno et al. 2003, 2007, Sato et al. 2009, Williams et al. 2010, Aeby et al. 2011b). It is likely that several environmental factors simultaneously influence disease dynamics within a system, with the relative importance of each factor varying among regions, spatial scales, and species (Aeby et al. 2011a,b). We have just begun to understand the complex web of interactions between environmental factors and disease prevalence exemplified by studies that applied a multi-factor approach to studying coral disease dynamics (e.g. Bruno et al. 2007, Haapkylä et al. 2007, McClanahan et al. 2009, Williams et al. 2010, 2014, Aeby et al. 2011a,b).

Additionally, different coral diseases can show varying levels of ecological impact. For example, black band disease (BBD) and white syndromes often cause severe colony mortality (Edmunds 1991, Bruckner et al. 1997, Roff et al. 2006, Aeby et al. 2010, Williams et al. 2011a), whereas corals with *Porites* ulcerative white spot syndrome (PUWS) in the Philippines often show complete recovery after infection (Kaczmarek 2006). In addition, different coral species/taxa appear to vary in their susceptibility to disease infection. For example, on the Great Barrier Reef (GBR), BBD affects 25 out of approximately 350 hard coral species, with branching *Acropora* spp. being most affected (Page & Willis 2006). The degree of damage to the ecosystem therefore depends on the suite of coral species and diseases that occur on the reef. If we are to successfully manage our reef sys-

tems, it is vital to understand the often intricate disease–environment interactions that lead to complex temporal and spatial disease dynamics, as well as the nature and causes of the different diseases that affect a system.

Kaneohe Bay in Oahu, Hawaii (USA), contains many fringing and patch reefs with high coral cover. *Porites compressa* and *Montipora capitata* are the 2 dominant framework-building corals on these reefs, with *P. compressa* accounting for up to 80% of the hard coral cover at some sites (Williams et al. 2010). Several coral diseases have been reported from Kaneohe Bay, of which *Porites* trematodiasis (Aeby 2007, Williams et al. 2010), *Porites* growth anomalies (Domart-Coulon et al. 2006, Williams et al. 2010, Stimson 2011), and *Montipora* white syndrome (MWS) (Aeby et al. 2010, Williams et al. 2010) are the most studied. In the present study, we describe the dynamics of another disease in Kaneohe Bay, *Porites* bleaching with tissue loss (PBTL), which affects *P. compressa*. This disease manifests as bleaching of the coenenchyme with the polyps remaining brown, giving the coral a ‘speckled’ appearance (Fig. 1B) which is distinct from the uniform color loss associated with thermal bleaching. PBTL causes tissue loss due to necrosis and tissue fragmentation (Sudek et al. 2012b), and a significant reduction in gamete development (Sudek et al. 2012a). Preliminary observations of an apparent increase of PBTL prevalence during the summer months suggested a potential link to temperature (M. Sudek unpubl. data), but overall little is known about the ecology of this disease. The objectives of the present study were therefore to (1) examine the variability in disease prevalence (proportion of individuals affected) over the course of 1 yr (temporal variability), and determine the spatial distribution of PBTL within Kaneohe Bay and Hanauma Bay (another reef with high *P. compressa* cover located on the windward side of Oahu); (2) examine virulence (degree of harm to the host); (3) investigate disease transmissibility; and (4) determine the environmental correlates of variations in disease prevalence.

MATERIALS AND METHODS

Prevalence and spatial distribution

Prevalence surveys were conducted at 8 permanent sites (A–D and G–J) around Coconut Island Marine Reserve (CIMR), Kaneohe Bay, Oahu, Hawaii, USA (21° 26' N, 157° 47' W; Fig. 2), on an ap-

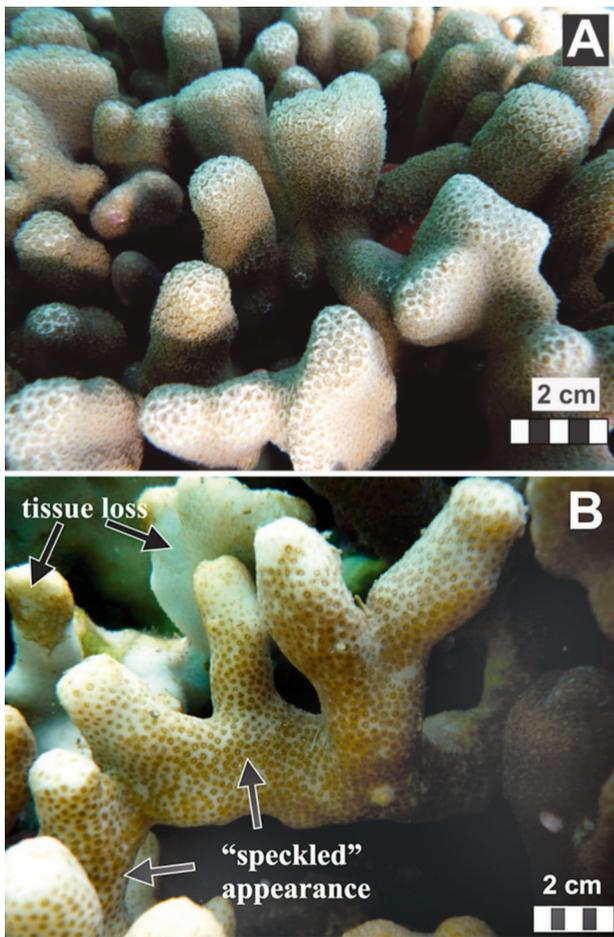


Fig. 1. (A) Healthy *Porites compressa*. Note regular brown coloration. (B) *Porites* bleaching with tissue loss (PBTL)-affected *P. compressa*. Note pigmented polyps and bleached coenenchyme ('speckled appearance') with onset of tissue loss

proximately monthly basis during 2011. Five 10×2 m belt transects were deployed at each site in which every *Porites compressa* colony was counted and examined for signs of PBTL.

To investigate the larger spatial extent of PBTL, rapid visual surveys were conducted on 9 patch reefs within Kaneohe Bay and on the reef in Hanauma Bay Marine Reserve (Fig. 2). Within Kaneohe Bay, a snorkeler swam for 10 min at a speed of approximately 10 m min^{-1} along a haphazardly selected patch reef and recorded every PBTL-affected colony observed. In Hanauma Bay, 2 divers swam across the reef at approximately the same speed and recorded the number of PBTL-affected colonies encountered within 30 min. This rapid survey method allows a larger spatial coverage of the reefs and provides a semi-quantitative measure of disease abundance. All rapid surveys were conducted in October 2011.

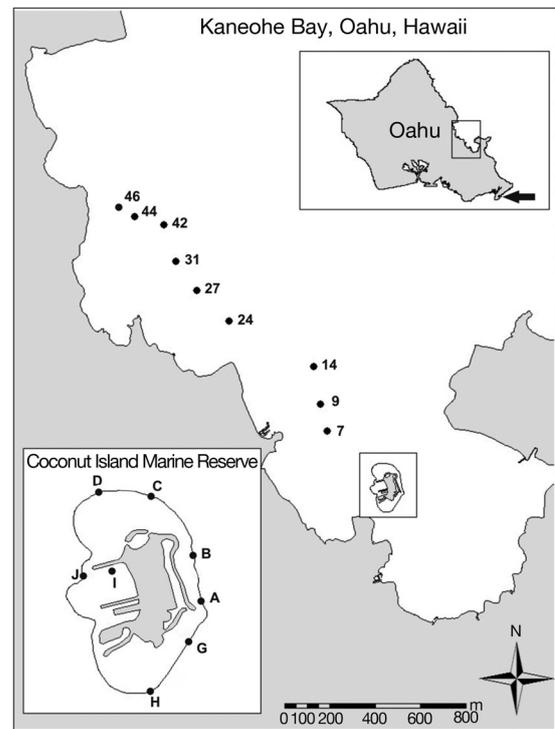


Fig. 2. Kaneohe Bay, Hawaii (USA), showing the 9 rapid survey sites (black dots marked 7 to 46) numbered after Roy (1970), with an inset of Oahu showing the location of Kaneohe Bay and Hanauma Bay (arrow) and another inset of Coconut Island Marine Reserve (CIMR) showing the 8 permanent sites (A–J)

Disease virulence

To determine disease virulence, 42 individual PBTL-affected colonies were tagged in 2010 (Sudek et al. 2012a), and an additional 36 PBTL-affected colonies were tagged in 2011 and followed through time (approximately monthly examinations). The colonies that were tagged in 2010 were resurveyed and checked for new PBTL signs and/or signs of tissue recovery. Due to the 3-dimensional structure of *Porites compressa* and the often poor visibility in Kaneohe Bay, we could not rely on photographic surveys with post hoc image analysis. Instead, the percentage of healthy, dead, and affected tissue was estimated visually *in situ* in addition to photo documentation.

Transmission

To determine whether PBTL is transmissible through the water column or via direct contact, healthy as well as PBTL-affected coral samples (approximately

3 cm² each) were collected from the reef crest around CIMR using a bone cutter. Samples were transported to the lab in individual plastic bags to avoid any cross contamination. Manipulative experiments were run in aquaria (8 l) under closed conditions. To maintain water quality, a bubbler was placed in each aquarium to ensure water movement, and partial water changes, using 0.2 µm filtered seawater, were carried out every 5 d. Aquaria were kept outside under natural light.

To conduct the transmission experiment, a coral fragment showing signs of PBTL was placed in an aquarium touching a healthy fragment, with another fragment from the same healthy colony placed about 10 cm away from the PBTL-affected fragment (n = 10). As a control for possible effects of intraspecific competition, the same setup was used with fragments from the same healthy colony as those used in the transmission treatment, but the diseased fragment was replaced by a healthy fragment from a different colony (n = 10). The healthy fragments were monitored daily for signs of PBTL over the course of at least 3 wk or until the affected fragment died (max. 5 wk). The transmission experiment was carried out at ambient (25°C) and increased (28°C) water temperatures to determine whether transmission would occur more readily at higher temperatures (n = 10 treatment⁻¹). Thermal bleaching of Hawaiian corals occurs with prolonged exposure to 29–30°C (Jokiel & Coles 1990), so by choosing a temperature of 28°C, the experimental setup stayed below the range in which temperature-induced bleaching would usually occur. Additionally, the 'speckled' appearance of PBTL is not observed during thermal bleaching or bleaching due to competition; therefore only the typical 'speckled' bleaching appearance was considered a sign of PBTL.

Environmental drivers

All measurements of environmental variables were conducted at the depths of the transects. Temperature data were collected at each site using HOBO® Pro data loggers (www.onsetcomp.com) with an accuracy of ±0.2°C. The loggers recorded continuously every 30 min from late February to late December 2011. Turbidity, chlorophyll *a* (chl *a*), and salinity were measured at each site using an RBR® XR-420 data logger (www.rbr-global.com) recording every minute over a 36 to 48 h period on 4 to 6 different occasions per site in 2010 and 2011. The logger was moved randomly between sites to maximize spatial

coverage over time. Water motion was estimated using the clod card technique (Jokiel & Morrissey 1993). Two clod cards were placed at the beginning of each survey site and left overnight (21 to 23 h). In addition, 2 clod cards were placed into a large bucket containing seawater (ca. 60 l) to serve as a diffusion control. The exact time that the clod cards were immersed in water was recorded, and the diffusion factor (DF, a dimensionless index of water motion) was calculated for each site (Jokiel & Morrissey 1993). Clod cards were deployed 4 times over the course of 6 mo in 2011, and the average DF for each site was used in subsequent data analyses.

Corallivorous fish can be potential vectors of disease (Aeby & Santavy 2006) or a source of injury which can promote the spread of certain diseases (Page & Willis 2008, Raymundo et al. 2009). The densities of all corallivorous butterflyfish (facultative and obligate) and parrotfish were recorded over an area of 50 × 4 m at all 8 sites. The observer swam at a speed of approximately 10 m min⁻¹ and recorded all butterflyfish to species level (*Chaetodon auriga*, *C. ephippium*, *C. lineolatus*, *C. lunulatus*, *C. multicinctus*, *C. ornatissimus*, *C. unimaculatus*). Due to difficulties with species-level identification, all parrotfish (adults and juveniles) were grouped. Fish counts were carried out during 4 different months in 2011 (July, August, September, December), and all sites were surveyed on the same day within 2 to 3 h of each other. Total numbers of fish were used in the subsequent data analyses.

Statistical analyses

Prevalence and spatial distribution

Prevalence data by transect did not display a normal distribution, even after transformation. We therefore used a repeated measures permutational analysis of variance based on a binomial deviance matrix (the technique does not assume normality) in PERMANOVA+ (Anderson et al. 2008), to test the effect of 2 fixed factors (site, month) and their interaction with disease prevalence.

Environmental drivers

To investigate temporal variations in disease prevalence, the relationship of temperature and prevalence was explored over a period of 10 mo using a general linear model (GLM) performed with SPSS

(PASW 18). Prevalence was averaged for each site (over the 5 individual transects), and the data displayed a normal distribution. Temperature data were averaged over the 10 d period before each survey for every site.

To examine spatial variations in disease prevalence (differences in PBTL prevalence across sites regardless of month), 8 environmental predictor variables were modeled against spatial variations in prevalence across sites. Measurements for predictor variables were not continuous through time and were therefore averaged for each site. To achieve the same resolution for temperature and prevalence data, all temperature data (10 d before each survey) and prevalence values (February to December) were averaged for each site. Predictor variables were: host cover, turbidity, water temperature, chl *a*, water motion, salinity, parrotfish density, and butterflyfish density (Table 1). Because most butterflyfish species showed low abundances on the reef, all butterflyfish counts were grouped. The mean and 1 SD of all predictor variables were initially examined to also account for the variability of factors at the individual sites. Inter-correlation of predictor variables was tested using Pearson's correlation, with predictors exceeding a correlation value of >0.75 considered for removal and further examined using principal coordinates analysis (PCO) plots (Anderson et al. 2008). Variables chosen for inclusion in the model were mean values for host cover, temperature, chl *a*, and water motion, and the variability (SD) in turbidity, salinity, butterflyfish density, and parrotfish density. A permutational distance-based linear model (DISTLM) was used (McArdle & Anderson 2001) to analyze the data. DISTLM is a multivariate multiple regression technique that quantifies the proportion of the variation in the response variable (in this case

PBTL prevalence) explained by the predictor variables. Environmental data were normalized and the DISTLM routine was run using the 'best' selection procedure, based on 9999 permutations. Akaike's information criterion (Akaike 1973) with a second-order bias correction applied (AICc) (Hurvich & Tsai 1989, Burnham & Anderson 2004) was used for model selection. The most parsimonious model with the lowest AICc and highest R^2 value was selected. Modeling analyses were based on 0-adjusted Bray-Curtis similarity matrices (Clarke et al. 2006) and carried out using PRIMER v6 (Clarke & Gorley 2006) and PERMANOVA+ (Anderson et al. 2008).

RESULTS

Prevalence and spatial distribution

Overall, average PBTL prevalence at CIMR was 1.5% ($\pm 0.2\%$ SE). PBTL-affected colonies were found at all 8 survey sites, but prevalence differed significantly between sites ($df = 7$, pseudo- $F = 9.5969$, $p < 0.001$; Fig. 3A) and between months ($df = 9$, pseudo- $F = 8.5552$, $p < 0.001$; Fig. 3B), but no significant interaction between the two was detected ($df = 63$, pseudo- $F = 0.83471$, $p = 0.7473$). PBTL-affected colonies were observed throughout the year, with a peak during the summer months (Fig. 3B) and the highest average prevalence observed in June ($2.5 \pm 0.3\%$ SE). Rapid visual surveys showed that PBTL was present in all of Kaneohe Bay but that it was absent from Hanauma Bay (Table 2).

Disease virulence and transmission

Most colonies affected by PBTL showed the typical 'speckled' bleaching for a period of approximately 2 to 3 mo and then the disease regressed in most cases (no more signs of bleaching). Within this time, the majority of colonies (85%) showed tissue loss ranging from 5 to 100% of the colony, with a case fatality rate (total mortality) of 3%. On average, a colony lost a third (30%) of its tissue within 2 mo. Of the 42 colonies tagged in 2010, 55% showed no signs of recovery in 2011, and 24% showed partial tissue regrowth. In addition, 31% of these colonies became affected again by PBTL during 2011.

No disease transmission occurred between individuals via the water column or direct contact in either the ambient or the increased temperature treatments ($n = 10$ treatment $^{-1}$).

Table 1. Predictor variables used in model analyses with their units and minimum and maximum values

| Variable | Description and units | Min. | Max. |
|-----------------------|----------------------------------|------|------|
| Water temp. | °C | 22.1 | 28.3 |
| Host cover | % <i>Porites compressa</i> cover | 30.0 | 75.6 |
| Turbidity | Formazin turbidity unit (FTU) | 0.3 | 22.6 |
| Chl <i>a</i> | $\mu\text{g l}^{-1}$ | 0.02 | 2.1 |
| Water motion | Diffusion factor (DF) | 1.26 | 7.95 |
| Salinity | ppt | 33.0 | 35.7 |
| Parrotfish density | Number per 200 m ² | 0 | 68 |
| Butterflyfish density | Number per 200 m ² | 0 | 13 |

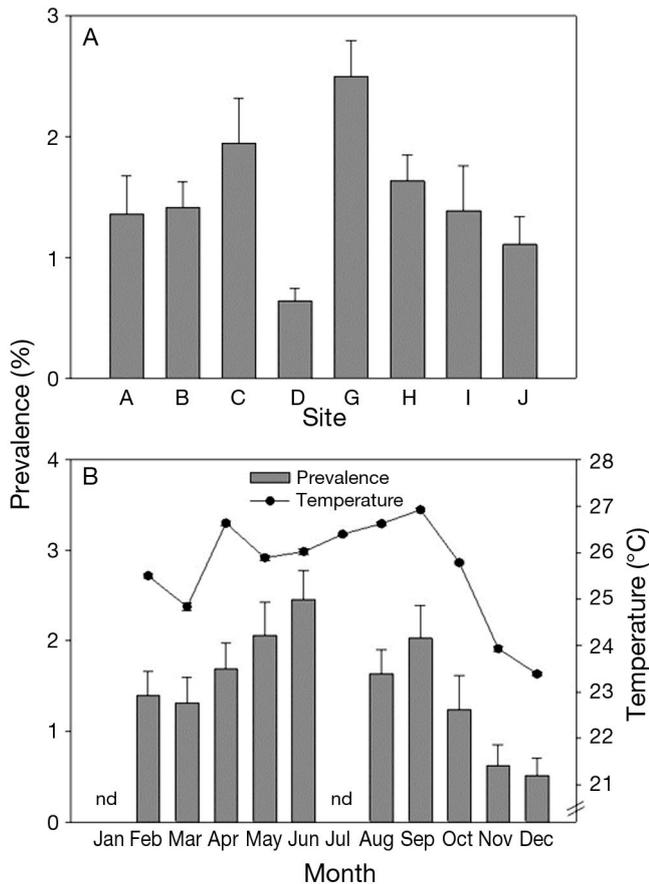


Fig. 3. *Porites* bleaching with tissue loss (PBT) prevalence at Coconut Island Marine Reserve (CIMR). (A) Average prevalence (\pm SE) for each permanent site around CIMR. (B) Average prevalence over sites (\pm SE) for each month in 2011 with corresponding average temperature (the average over 10 d prior to surveying); nd: no prevalence data obtained

Table 2. Number of *Porites* bleaching with tissue loss (PBT)-affected *Porites compressa* colonies observed (see Fig. 1 for reef locations) during rapid surveys within Kaneohe Bay and Hanauma Bay, Hawaii, USA

| Area | Reef no. | No. of colonies min^{-1} of search time |
|-------------|-------------|--|
| Kaneohe Bay | North Bay | 46 |
| | | 44 |
| | | 43 |
| Central Bay | | 31 |
| | | 27 |
| | | 24 |
| South Bay | | 14 |
| | | 9 |
| | | 7 |
| SE Oahu | Hanauma Bay | 0 |

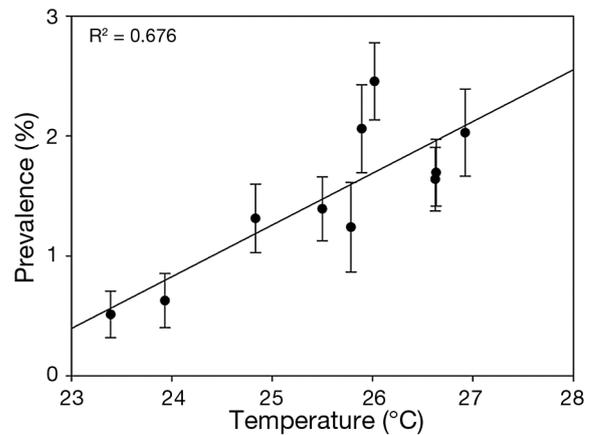


Fig. 4. Relationship of average *Porites* bleaching with tissue loss (PBT) prevalence (\pm SE) for each month (February to December) with corresponding temperature (the average over 10 d prior to surveying)

Environmental drivers

A significant positive linear relationship was found between temporal water temperature and disease prevalence (GLM: Wald $\chi^2 = 38.128$, $df = 1$, $p < 0.001$; Fig. 4). Based on this relationship, each degree increase in temperature can be expected to result in an increase of 0.29 to 0.56 % in disease prevalence.

Modeling of the spatial variation in PBT prevalence across sites identified water motion and the variability in turbidity and parrotfish density as the strongest predictors, with 26.2% of the total variability in PBT prevalence across sites explained (Table 3). Water motion and parrotfish density showed a positive correlation to variations in PBT prevalence, whereas turbidity showed a weak negative correlation. Butterflyfish density, chl *a*, spatial temperature (difference between sites), salinity, and host cover were not found to be important spatial predictors of PBT prevalence across sites.

DISCUSSION

Prevalence and distribution

PBT was found to be widely distributed on reefs within Kaneohe Bay but was absent from Hanauma Bay which also has a high abundance of the affected coral species (*Porites compressa*). PBT has also not been reported from other reefs within the Main or Northwestern Hawaiian Islands (Aeby et al. 2011a),

Table 3. Summary results of a distance-based linear model (DISTLM) analysis showing the 'best' model with the lowest Akaike's information criterion with a second-order bias correction (AICc) value and highest amount of variability explained

| AICc | Predictor | Pseudo- <i>F</i> | <i>p</i> | % variability explained | Relationship with prevalence |
|--------|-----------------------|------------------|----------|-------------------------|------------------------------|
| 274.94 | Turbidity SD | 6.0364 | 0.0026 | 12.8 | Negative |
| | Water motion | 3.7671 | 0.0281 | 9.0 | Positive |
| | Parrotfish density SD | 2.1351 | 0.1148 | 4.4 | Positive |
| | Total | | | 26.2 | |

suggesting that PBTL may be restricted to Kaneohe Bay.

In Hawaii, average coral disease prevalence (excluding *Porites* trematodiasis) is less than 1% (Aeby et al. 2011a), which is lower than what was documented for PBTL (average prevalence: $1.5 \pm 0.2\%$ SE; range: 0 to 3.7%). Compared to other diseases within Kaneohe Bay, PBTL prevalence was higher than that of MWS (average prevalence: $0.23 \pm 0.09\%$ SE; Aeby et al. 2010) but lower than *Porites* growth anomalies (Por GAs) (average prevalence: $21.7 \pm 8.3\%$ SE at a particular site; Domart-Coulon et al. 2006). However, both MWS and Por GAs have a wider range in prevalence across sites within Kaneohe Bay (0 to 29 and 1 to 56%, respectively) (Williams et al. 2010).

The ecological damage from disease on a host population depends on a combination of the spatial distribution, prevalence, and virulence of the disease. For example, MWS has a much lower prevalence than Por GAs, but MWS can cause extensive tissue loss and high colony mortality (Aeby et al. 2010), whereas Por GAs only result in colony morbidity (reduced growth) (Stimson 2011). PBTL has a relatively low prevalence, but it can cause extensive tissue loss, and recovery rates (tissue re-growth) appear to be very slow. It was found to be an ephemeral disease with disease signs (speckled bleaching) disappearing in most cases within a couple of months, and a small proportion of colonies showed disease regression (i.e. repigmentation) without any signs of tissue loss. However, a third of the colonies showed signs of PBTL again after complete cessation of the disease. A number of other coral diseases have also been found to reoccur (e.g. Kuta & Richardson 1996, Sato et al. 2009, Aeby et al. 2010), and it has been suggested that recurrent infections can cause cumulative tissue loss leading to colony mortality and resulting in increased damage to the reef system over time (Borger & Steiner 2005). Even though PBTL prevalence is relatively low, a cumulative effect of periodic tissue loss could have a negative impact on *Porites compressa*-dominated reefs.

Transmission

No disease transmission was observed between healthy and PBTL-affected fragments, suggesting that PBTL does not easily transmit via direct contact or the water column (at least over a period of approximately 1 to 2 mo). It may be that the environmental conditions needed for successful transmission were not replicated by our experimental treatment. However, direct transmission between touching colonies was also not observed in the field. In contrast, other manipulative experiments have successfully shown disease transmission in aquaria. For example, MWS was shown to be transmissible through direct contact in aquarium conditions, with direct transmission also observed in the field (Aeby et al. 2010). In our model, we found that host abundance was not an important factor in predicting PBTL prevalence. The relationship between disease prevalence and host abundance is a central element in the theory of infectious disease ecology (Lloyd-Smith et al. 2005) because transmission is a key process in host–pathogen interactions, and increased host density can increase the probability of horizontal transmission of an infectious disease (Altizer & Augustin 1997). As such, we suggest that PBTL is either not caused by a pathogen, is not highly infectious, or that some other variable, such as a vector, may be needed for disease transmission.

Environmental drivers

Variations in turbidity were identified as the overall strongest predictor of spatial variation in disease prevalence (higher PBTL prevalence) across sites. Turbidity showed a weak negative relationship with PBTL prevalence, indicating that clearer waters are associated with higher disease prevalence. We also found that PBTL prevalence, across all sites, was highest during the summer months, strongly correlated with water temperature. Increased disease prevalence on coral reefs often correlates with ele-

vated seawater temperature, for example as reported for BBD (Boyett et al. 2007, Rodriguez & Croquer 2008), some white syndromes (Selig et al. 2006, Bruno et al. 2007, Williams et al. 2010, 2011a), and a fungal disease affecting tropical crustose coralline algae (Williams et al. 2014). Increased temperatures can lead to an increase in pathogen virulence and/or cause stress to the host, making it more susceptible to disease (Harvell et al. 2007). However, water temperature is not the only abiotic factor that varies seasonally on reefs. For example, Sato et al. (2011) found that high light and elevated seawater temperature drive the occurrence of BBD on the GBR. They proposed that seasonally increased light levels may be even more important for inducing new infections than increased water temperature. A link to increased light could explain the spotty appearance of PBTL (bleached coenenchyme and pigmented polyps), as *Symbiodinium* cells may be more shielded in the polyps because they can retract into the skeleton. However, manipulative experiments are needed to clarify the link between light, temperature, and PBTL.

PBTL prevalence was also correlated with higher water motion and higher parrotfish densities, although the link appeared rather weak and is therefore not discussed further. Overall, only a quarter of the variability in PBTL prevalence could be explained by the measured factors, suggesting that other unmeasured abiotic or biotic factors could be more important drivers of PBTL prevalence. Alternatively, our predictor variables may not have been captured at an appropriate temporal scale, with seasonal variations in these factors missed; this may have caused a reduction of the predictive power of our model. Cause of disease is dependent on the intricate interactions between the host, environment, and pathogen (Work et al. 2008). One can therefore expect coral disease spatio-temporal dynamics to be highly complex and to be correlated with multiple, and possibly co-interacting, environmental drivers (Williams et al. 2010).

CONCLUSION

This is the first study examining the disease dynamics of PBTL in Kaneohe Bay, Hawaii. PBTL causes partial colony mortality in the host coral *Porites compressa*, appears to be non-infectious, and was found to have the highest prevalence occurring in the warmer summer months, indicating possible seasonal dynamics. Spatial variation in disease

prevalence (higher PBTL prevalence) across sites was correlated with higher water motion, lower turbidity, and higher parrotfish densities, but the model did not sufficiently explain the spatial variability. This highlights the complex nature of host–pathogen–environment interactions and the need for investigating and understanding coral disease ecology. Further research into the causative agent and links to environmental drivers, specifically at a finer temporal scale, are needed to better understand the dynamics of this disease. *Porites compressa* is among the main framework-building corals in Kaneohe Bay, and so chronic, recurrent diseases, such as PBTL, could have a negative impact on the health and structure of these reefs.

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