Coral Reefs
DOI 10.1007/s00338-014-1174-x

REPORT

Spatial and temporal patterns of coral health and disease along leeward Hawai'i Island

C. S. Couch · J. D. Garriques · C. Barnett · L. Preskitt · S. Cotton · J. Giddens · W. Walsh

Received: 5 October 2013 / Accepted: 19 May 2014

Abstract Ecological processes including disease, competition for space, and predation strongly influence coral reef health from the colony to reef level. The leeward/west coast of the island of Hawai'i consists of the largest expanse of intact reefs in the Main Hawaiian Islands (MHI), yet little is known about the health of its coral communities. We measured prevalence of coral diseases and non-disease conditions at nine regions across two depths in the summer and winter months between 2010 and 2011. We also assessed long-term changes in coral cover (2003–2011). Mean prevalence of chronic diseases was 5–21 times greater than previously reported for the MHI. Coral health varied minimally across survey months with mild seasonality only detected in algal overgrowth (ALOG). Coral health varied considerably by depth and site, and was primarily driven by the most prevalent and common conditions: *Porites* growth anomalies (13.7 ± 0.82 %), *Porites* trematodiasis (9.5 ± 0.90 %), discoloration (5.6 ± 0.33 %), ALOG (9.9 ± 0.54 %), and gastropod predation (2.4 ± 0.23). While several conditions were significantly elevated in shallow zones, unique site × depth interactions suggest that specific site-level factors are driving prevalence. At the coast-wide level, percentage of coral cover did not change significantly between 2003 and 2011, but decreased significantly at two sites and increased at one site. Based on coral cover decline and high prevalence of certain coral health conditions, we identified four regions of concern (Puakō, Mauna Lani, Ka'ūpūlehu, and Hōnaunau). The high spatial variation in coral health not only advances our understanding of coral disease ecology, but also supports reef resilience planning by identifying vulnerable areas that would benefit most from targeted conservation and management efforts.

Keywords Coral disease patterns · Hawai'i · *Porites* · Growth anomaly · Trematodiasis · Predation · Algal overgrowth

Communicated by Biology Editor Dr. Hugh Sweatman

Electronic supplementary material The online version of this article (doi:10.1007/s00338-014-1174-x) contains supplementary material, which is available to authorized users.

C. S. Couch (✉)
Department of Ecology and Evolutionary Biology, Cornell University, Ithaca, NY 14853, USA
e-mail: courtneyscouch@gmail.com

C. S. Couch
Hawai'i Institute of Marine Biology, University of Hawai'i at Mānoa, Kaneohe, HI 96734, USA

J. D. Garriques
NOAA Pacific Islands Fisheries Science Center, Joint Institute for Marine Atmospheric Research, 1125B Ala Moana Blvd, Honolulu, HI 96814, USA

C. Barnett · L. Preskitt · S. Cotton · W. Walsh
Hawai'i Division of Aquatic Resources, Kailua-Kona, HI 96740, USA

J. Giddens
Fishery Ecology Research Lab, Department of Biology, University of Hawai'i at Mānoa, Honolulu, HI 96822, USA

Introduction

Ecological processes including disease, competition for space, and predation play important roles in regulating coral populations (reviewed by Birkeland 1997; Harvell et al. 2007). Although coral disease is a natural component of healthy ecosystems, it can have detrimental effects on ecosystem structure and function during outbreaks (Aronson

Published online: 05 June 2014

and Precht 2001; Kim and Harvell 2004). Disease has become a major contributor to global coral mortality since the 1980s, especially in the Caribbean where disease and other local stressors have led to widespread phase shifts to communities dominated by other coral genera or macroalgae (Aronson and Precht 2001). Although the effects of coral disease have been most pronounced throughout the Caribbean (e.g., Aronson and Precht 2001; Ruiz-Moreno et al. 2012), impacts to Pacific reefs remained largely undetected until the early 2000s (Willis et al. 2004).

While the causative agents of most coral diseases remain unknown, an estimated 25 diseases/syndromes affect Pacific corals ranging from chronic such as growth anomalies to acute infectious diseases such as white syndrome and black band disease (Vargas-Angel 2009). Preliminary evidence suggests that Pacific coral diseases are increasing both temporally (Willis et al. 2004; Ruiz-Moreno et al. 2012) and spatially, now reaching even remote reefs such as the US Pacific Remote Island Area (Vargas-Angel 2009), Palmyra Atoll (Williams et al. 2010b) and the Northwestern Hawaiian Islands (Aeby et al. 2011a). While the causes of this increase are not well understood, coral disease patterns have been linked to a combination of global and local factors. Most notably, tissue loss diseases have been correlated with seasonal increases in sea surface temperature and light (Boyett et al. 2007; Sato et al. 2009) and thermal stress associated with climate change (Bruno et al. 2007; Heron et al. 2010). On the local scale, disease is also affected by local environmental disturbances (e.g., Kaczmarsky and Richardson 2010; Haapkylä et al. 2011), as well as coral abundance (Bruno et al. 2007). Only through repeated coral health assessments at the same sites over time can we understand the broader role of disease in reef health, as well as the underlying mechanisms driving these patterns.

In addition to disease, other biological interactions and compromised health states such as competition and predation strongly influence reef ecosystems both directly and indirectly. Coral–macroalgal competition is a well known contributor to colony- and reef-level mortality. Under eutrophic conditions, accelerated algal growth can overgrow and kill coral tissue, inhibit coral recruitment, enhance microbial activity, or result in ecosystem phase shifts (e.g., Done 1992; Hughes 1994; Smith et al. 2006). Invertebrate corallivores such as the gastropod *Drupella* spp. and the crown-of-thorns starfish (COTS), *Acanthaster planci*, have become major contributors to coral cover decline (e.g., Turner 1994; De'ath et al. 2012) and have been linked to disease onset by spreading pathogenic microorganisms and wounding tissue (Nugues and Bak 2009; Nicolet et al. 2013). To date, very few coral health monitoring programs have quantitatively assessed the role of these ecological processes.

In Hawai'i, coral disease epizootics, macroalgal blooms, and a variety of natural and anthropogenic disturbances have dramatically shaped coral health and reef resilience. Human population growth and a burgeoning tourism industry in Hawai'i have resulted in considerable land use change, mass sedimentation events, and eutrophication (Jokiel et al. 2004; Kittinger et al. 2011). These changes have been most pronounced in regions such as Kāne'ohe Bay, O'ahu, where environmental disturbances have resulted in precipitous coral cover decline and widespread macroalgal blooms (reviewed by Hunter and Evans 1995). Of the 12 diseases affecting Hawaiian corals (Aeby et al. 2011a), outbreaks of *Montipora* white syndrome in Kāne'ohe Bay (Aeby et al. 2010) and *Acropora* white syndrome in French Frigate Shoals (Aeby et al. 2011a), as well as anomalously high *Montipora* growth anomaly levels in southeastern Hawaii Island (Burns et al. 2011) have dramatically affected one of the region's dominant reef builders. To date, coral disease research has been focused on impacted reefs with ongoing disease outbreaks, with relatively less effort in describing disease dynamics of the most intact reefs such as those surrounding the Island of Hawai'i (but see Takabayashi et al. 2008; Burns et al. 2011).

As the largest and youngest of the Hawaiian Islands, the Island of Hawai'i has the highest coral cover in the state (Battista et al. 2007; Franklin et al. 2013) and is also of particular concern due to its rapid human population growth rate (US Census Bureau 2007) and highly porous basaltic rock, which renders coastal ecosystems susceptible to coastal pollution (Street et al. 2008). The leeward/west coast (WHI) has the largest expanse of intact and actively growing reef in Hawai'i (Jokiel et al. 2004) and has not experienced the widespread coral cover loss observed on other islands but is demonstrating localized reef decline (Minton et al. 2012; Walsh et al. 2013). Coral disease assessments have been conducted along WHI as part of archipelago-wide studies (Vargas-Ángel and Wheeler 2009; Aeby et al. 2011a), but less is known about the spatiotemporal dynamics of WHI coral health. To characterize coral health, determine whether coral cover is changing over time and identify sites of specific concern for management action along WHI, we tested the following hypotheses: (1) coral health varies temporally and is correlated with temperature fluctuation, (2) coral health varies as a function of depth and site, and (3) percentage of coral cover varied temporally during the last 8 yrs.

Methods

Study sites

Surveys were conducted at nine regions along West Hawai'i and were chosen based on proximity to West Hawai'i Aquarium Project sites (Tissot et al. 2004),

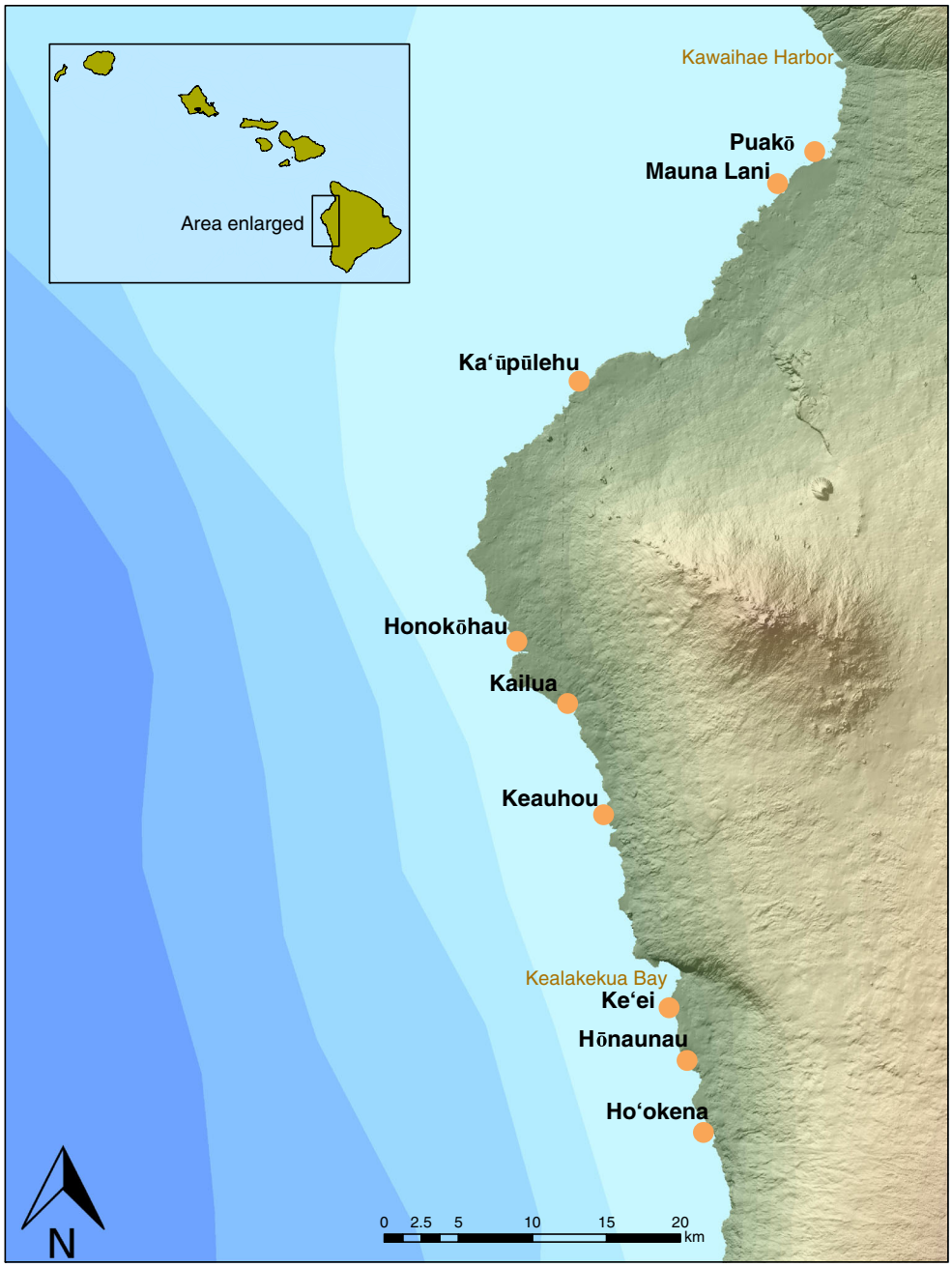

Fig. 1 West Hawai'i study site locations, Main Hawaiian Islands, USA

shoreline access, and expressed interest from the Hawai'i Division of Aquatic Resources (Fig. 1). At each location, a shallow site was established by haphazardly choosing four coordinates within a 5,000 m^2 area (3–6 m depth). At each coordinate, a 10-m transect was extended at a haphazard bearing parallel to shore and marked with GPS coordinates to relocate in future seasons. At each site, the deep habitat was also surveyed by conducting coral health assessments along the first 10 m of four established West Hawai'i Aquarium Project permanent transects (10–15 m depth).

Coral health assessments

Coral health surveys were conducted on four 10 × 2-m belt transects (80 m^2), along which all colonies were counted, identified to species, and inspected for lesions causing any morphological change to the tissue (Work and Aeby 2006). Lesions were characterized based on macroscopic similarities to lesions described by Beeden et al. (2008) and Aeby et al. (2011a). The following categories were used (Electronic Supplemental Material, ESM Fig.

S1; ESM Table 1): growth anomalies (protuberant growths of skeleton accompanied by aberrant calyx formation), *Porites* trematodiasis [multiple small (~5 mm) swollen pink to white nodules], tissue loss syndrome (distinct areas of subacute tissue loss revealing intact white skeleton progressing basally to an algal patina), multifocal tissue loss (multiple variably sized areas of acute to subacute tissue loss), *Pavona* dark spot (diffuse purple or reddish lesions macroscopically similar to hypermycosis (Work et al. 2008b), algal overgrowth (areas where macroalgae actively overgrows, abrades, and/or kills underlying coral tissue), discoloration (areas of discolored and/or swollen tissue not associated with other lesion categories), gastropod predation, crown-of-thorns predation, and bleaching.

Shallow sites were surveyed between January and February 2010, July–August 2010, January–February 2011, and July–August 2011 to assess seasonal fluctuations in coral health. Shallow and deep sites were surveyed between January and March 2011 to determine the spatial variation in prevalence of each condition. Due to the genus-specific disease patterns and to enable comparison with previous studies in Hawai'i (Williams et al. 2010a; Aeby et al. 2011a), prevalence of each disease was calculated for the primary reef-building genera. For example, *Porites* growth anomalies (PorGA) prevalence = (number of *Porites* colonies with GAs/total number of *Porites* colonies surveyed) × 100. The prevalence of colonies with ALOG, GastPRD, DC, BLE, and COTS were calculated for all colonies. The frequency of occurrence (percentage of transects surveyed with at least one lesion) was calculated to estimate the commonality of each condition. HOBO Water Temperature Pro version 2 data loggers (HoboTemp; Onset Co., Pocasett, MA) were launched at 1-h intervals and deployed at all shallow sites to monitor temperature continuously between July 2010 and August 2011.

Coral cover assessments

To broaden our interpretation of the health of WHI's coral communities beyond the scope of the 2-yr coral health assessments and target sites for future reef management, we also assessed trends in coral cover between 2003 and 2011. These surveys were conducted at the deep West Hawai'i Aquarium Project sites (Fig. 1) with photoquadrats using the following digital cameras: Olympus 5060 (Fall 2003), Olympus 7070 (Spring 2007), and an Olympus E-PL1 camera (Spring 2011). Images were taken at a fixed height (0.75 m) above the benthos with manual white balance. Images were taken at 1 m intervals along each of the four 10-m transects, producing 11 images per transect and analyzed using the Coral Point Count with Excel extensions software program (Kohler and Gill 2006). Data were pooled by transect using 30 randomly generated points per frame. A total of 30 points were deemed appropriate following a power analysis on a subset of sites. The proportion of each benthic category was determined for each image, and percentage of cover was calculated for each transect, total percentage of cover was obtained by calculating the mean percentage of cover of the four transects.

Statistical analyses

Several types of multivariate analyses were used to assess temporal and spatial variation in coral health (types of lesions recorded in surveys) assemblages using the 'vegan' package in R version 3.0.2. Due to logistical constraints preventing the comparison of depth across multiple seasons, separate spatial and temporal permutational multivariate analyses of variance (PERMANOVA) were conducted (Anderson 2001) using the 'adonis' function with 9,999 random permutations. The data were square-root transformed to reduce the influence of highly prevalent conditions, and analyses were conducted using a Bray–Curtis similarity matrix to remove the effect of joint absences (Anderson et al. 2011). PavDK was only found in 10 of surveys; hence these conditions were excluded from multivariate analyses. In the temporal PERMANOVA, site (shallow only) was treated as a random effect and survey month as a fixed effect. In the spatial PERMANOVA, a two-way design was used to test the effects of site and depth. A canonical analysis of principal coordinates (CAP) was used to visualize differences in coral health assemblages and determine which condition(s) most contributed to patterns in multivariate space across survey months, sites, and depth. This analysis is particularly useful for testing a priori hypotheses (e.g., effects of survey month or site) over multivariate space using dissimilarity measures such as Bray–Curtis (Anderson and Willis 2003). Coral health assemblages were also ordinated with unconstrained non-metric multidimensional scaling to confirm patterns. Similarities in the patterns between these methods gave weight to the reliability of these ordinations; hence only CAP bi-plots are presented. A similarity of percentages (SIMPER) analysis was run on both matrices and used to confirm multivariate results. SIMPER decomposes Bray–Curtis dissimilarities between all pairs of survey months or sites to identify those conditions that contributed most to variation in coral health.

To determine how prevalence of individual conditions varied temporally (survey month) or spatially (site × depth), univariate permutational ANOVAs ('vegan' package, 'adonis' function) and nonparametric one-way ANOVAs were used in R version 3.0.2. The presence of zeros necessitated using Euclidean distance as the dissimilarity measure for permutational ANOVAs. Nonparametric approaches were used for prevalence on conditions that were right skewed. Univariate analyses were only

conducted for conditions identified as indicator conditions in CAP analysis. To determine whether seasonal variation in particular coral health conditions were attributed to thermal fluctuation, prevalence was regressed against average bimonthly mean and maximum daily temperature using linear regressions.

To determine whether percentage of coral cover changed between 2003 and 2011 at West Hawai'i Aquarium Project sites, mean percentage of coral cover was analyzed for the entire coast and separately by site using linear mixed-effect models. For each site, transect nested within site was treated as a random effect and year as a fixed effect. Markov chain Monte Carlo simulations were used to detect differences between years (see Baayen et al. 2008). This method generates random samples from a posterior distribution of parameter values for fixed effects (Bolker et al. 2009). A total of 50,000 iterations were used to estimate the highest posterior density interval for each parameter, using 95 % of the probability distribution (credible intervals). Highest posterior density intervals not overlapping zero indicated conservative significant effects $\alpha = 0.05$ (Baayen et al. 2008).

Results

WHI corals were affected by a total of 12 diseases and biological interactions/compromised health states that varied in their prevalence and frequency of occurrence (Table 1; ESM Fig. S1). PorGA, PorTRM, ALOG, GastPRD, and DC were the most widespread lesions found at 100, 98, and 98, 95 and 81 % of all transects, respectively (Table 1). The most prevalent conditions were PorGA (13.7 ± 0.82 %), PorTRM (9.5 ± 0.90 %), ALOG (9.9 ± 0.54 %), and DC (5.6 ± 0.33 %) (Table 1). While subacute tissue loss diseases were recorded on *Porites* spp. and *Pocillopora* spp., these diseases occurred at low prevalence (<0.5 %). Alternatively, the major contributors to coral tissue mortality were ALOG and GastPRD. ALOG was primarily associated with red filamentous turf algae (ESM Fig. S1c), and GastPRD was attributed to *Drupella cornus* (ESM Fig. S1d; Turner 1994) and *Coralliophila violacea* (Oren et al. 1998).

Seasonal patterns in coral health and temperature

PERMANOVA results indicted that overall coral health differed significantly between survey months when nested within site (Table 2). However, multivariate ordination showed weak clustering of coral health conditions by survey month (Fig. 2a). SIMPER analysis confirmed the lack of strong temporal or seasonal patterns with no greater than 15 % of coral health conditions contributing to similarities between survey months. When analyzed individually, PorTRM, ALOG, and BLE varied significantly, albeit minimally, over time (Table 2; ESM Table S1). The only condition that showed mild and significant seasonality was ALOG, which was elevated during summer months (July and August) (Fig. 2b; ESM Table S1).

Table 1 Mean % prevalence (±SE) and frequency of occurrence of coral health conditions found in shallow and deep zones in January–February 2011, as well as all surveys between January 2010 and August 2011 along WHI

	Prevalence			Frequency of occurrence
	Shallow	Deep	All surveys	All surveys
PorGA	18.9 ± 2.34	13.3 ± 1.41	13.7 ± 0.82	100.0
PorTRM	12.4 ± 2.08	6.0 ± 0.1	9.5 ± 0.90	98.4
ALOG	10.6 ± 0.76	8.37 ± 1.15	9.9 ± 0.54	98.4
GastPRD	2.09 ± 0.31	2.75 ± 0.53	2.4 ± 0.23	95.3
DC	5.75 ± 0.83	2.44 ± 0.53	5.6 ± 0.33	90.6
BLE	0.59 ± 0.15	0.50 ± 0.09	1.5 ± 0.24	59.4
PorTLS	0.47 ± 0.13	0.79 ± 0.15	0.65 ± 0.07	56.3
PorMFTL	0.52 ± 0.18	0.42 ± 0.15	0.22 ± 0.04	34.4
PocTLS	0.56 ± 0.24	1.8 ± 0.94	2.2 ± 0.44	17.2
COTS	0.10 ± 0.08	0.16 ± 0.05	0.17 ± 0.04	15.6
PavDK	4.97 ± 3.25	0	2.90 ± 0.96	10.9
MonGA	0.11 ± 0.01	0.002 ± 0.002	0.14 ± 0.14	4.7

Ke'ei deep was not surveyed in January–February 2011, hence Ke'ei shallow was removed from shallow/deep comparison but included in mean prevalence across all surveys

PorGA: *Porites* growth anomaly; PorTRM: *Porites* trematodiasis; ALOG: algal overgrowth; GastPRD: gastropod predation; DC: discoloration; BLE: bleaching; PorTLS: *Porites* tissue loss syndrome; PorMFTL: *Porites* multifocal tissue loss syndrome; PocTLS: *Pocillopora* tissue loss; COTS: crown-of-thorns starfish predation; PavDK: *Pavona varians* dark spot; MonGA: *Montipora* growth anomaly

Table 2 Summary of PERMANOVA results testing effects of survey month (site as random effect) and univariate tests for coral health conditions identified as indicator conditions in CAP

Source of variation	*df*	MS	Pseudo-*F*	*P* (from permutations)
PERMANOVA				
Survey month	3	0.45	6.33	0.001
Residual	137	0.07		
Univariate permutational ANOVAs				
PorGA	3	3.16	0.98	0.448
PorTRM	3	9.85	2.98	0.032
ALOG	3	3.81	8.46	<0.0001
			χ^2	*P*
Univariate Kruskal–Wallis tests				
BLE	3		10.26	0.016

Analyses only include shallow sites

Overall, there was minimal seasonal fluctuation in average mean daily (24.59–26.40 °C) and maximum daily (25.00–26.76 °C) temperature (Fig. 2b). Temperature did not significantly predict ALOG prevalence (Linear regression: mean daily temperature $R^2 = 0.1731$, $P = 0.068$; maximum daily temperature $R^2 = -0.083$, $P = 0.499$).

Spatial patterns in coral health

We observed considerable spatial variation in the prevalence of the most common conditions. Coral health assemblages differed significantly between sites, depth, and the interaction of site and depth (Table 3). Multivariate ordination indicated that assemblages at the site level were predominately driven by PorGA, PorTRM, GastPRD, and DC (Fig. 3). These conditions were most characteristic of the following sites, PorGA: Mauna Lani, Ka'ūpūlehu, Kailua, and Hōnaunau; PorTRM: Mauna Lani, Kaloko-Honōkohau, and Ka'ūpūlehu; GastPRD: Kailua, Keauhou, and Honaunau; and DC: Mauna Lani, Kaloko-Honōkohau, and Ka'ūpūlehu (Fig. 3; ESM Table S2). At the depth level, PorGA, PorTRM, and DC prevalence were 1.42, 2.06, and 1.71 times higher in shallow zones, respectively, and GastPRD did not vary significantly with depth (Tables 1, 3). However, these depth effects, especially for PorGA, were strongly influenced by high prevalence at several sites and not consistent across all sites as evidenced by the significant site × depth interactions (Fig. 3; ESM Table S2). While ALOG prevalence weakly explained patterns in multivariate space, univariate analyses demonstrated that it varied significantly as function of site, depth, but not the interaction (Table 3), and was especially prevalent at Hōnaunau (19.02 ± 2.53 %) and Puakō (15.2 ± 0.72 %) (ESM Table S2). SIMPER analysis confirmed these spatial patterns and indicated that PorTRM, PorGA, ALOG, and DC alone contributed to up to 33, 24, 24, and 17 % of observed between site differences, respectively.

Long-term trends in coral cover

Overall, percentage of coral cover along deep WHI sites did not change significantly between 2003 and 2011 (Table 4, Highest Posterior Density interval: −1.30, 3.42). However, six of the eight deep sites declined by 2.19–12.01 %, with significant coral decline at Mauna Lani and Ka'ūpūlehu between 2003 and 2011 (Table 4). Two sites increased in % coral cover between these years, from 4.06 to 4.92 % with significant increases at Ho'okena (Table 4, HPD interval: −6.79, −0.28).

Discussion

Comparison of WHI to MHI patterns in disease prevalence

During this study, we conducted 164 surveys along the leeward coast of the Island of Hawai'i, which has one of the largest expanses of intact reefs in the Main Hawaiian Islands (MHI) and whose coral health has been largely overlooked. Similar to other regions such as Guam (Myers and Raymundo 2009) and the Philippines (Raymundo et al. 2005; Kaczmarsky 2006), WHI corals are affected by chronic and subacute diseases that primarily impact the dominant reef-building genus *Porites*. Unlike many regions in the Indo-Pacific (e.g., Willis et al. 2004; Sato et al. 2009), our WHI sites and others (Walsh et al. 2013) are currently unaffected by acute tissue loss diseases (e.g., black band disease and white syndrome). Our results also indicate that WHI coral health was highly influenced by conditions such as ALOG, GastPRD, and DC (Table 1).

One of our major findings is that PorGA and PorTRM prevalence, the most widespread diseases in the MHI (Aeby et al. 2011a), were 21 and 8 times greater, respectively, along WHI (PorGA 13.7 ± 0.82 %, PorTRM: 9.5 ± 0.90 %) than average prevalence previously reported by Aeby et al. (2011a) for the MHI (PorGA 0.64 ± 0.15 %; PorTRM: 1.1 ± 0.3 %). These pronounced regional differences are also consistent with other MHI assessments (Vargas-Ángel and Wheeler 2009). One potential explanation for WHI's anomalously high disease prevalence is the strong influence of host abundance. Percentage of coral cover has been positively correlated with disease prevalence in Hawai'i (Williams et al. 2010a; Aeby et al. 2011a; Couch 2014) and other Indo-Pacific reefs (e.g., Bruno et al. 2007; Myers and Raymundo 2009; Aeby et al. 2011b), and was on average 2.98–140.33 times higher in WHI than percentage of *Porites* cover across the MHI, including Hawai'i Island (Aeby et al. 2011a).

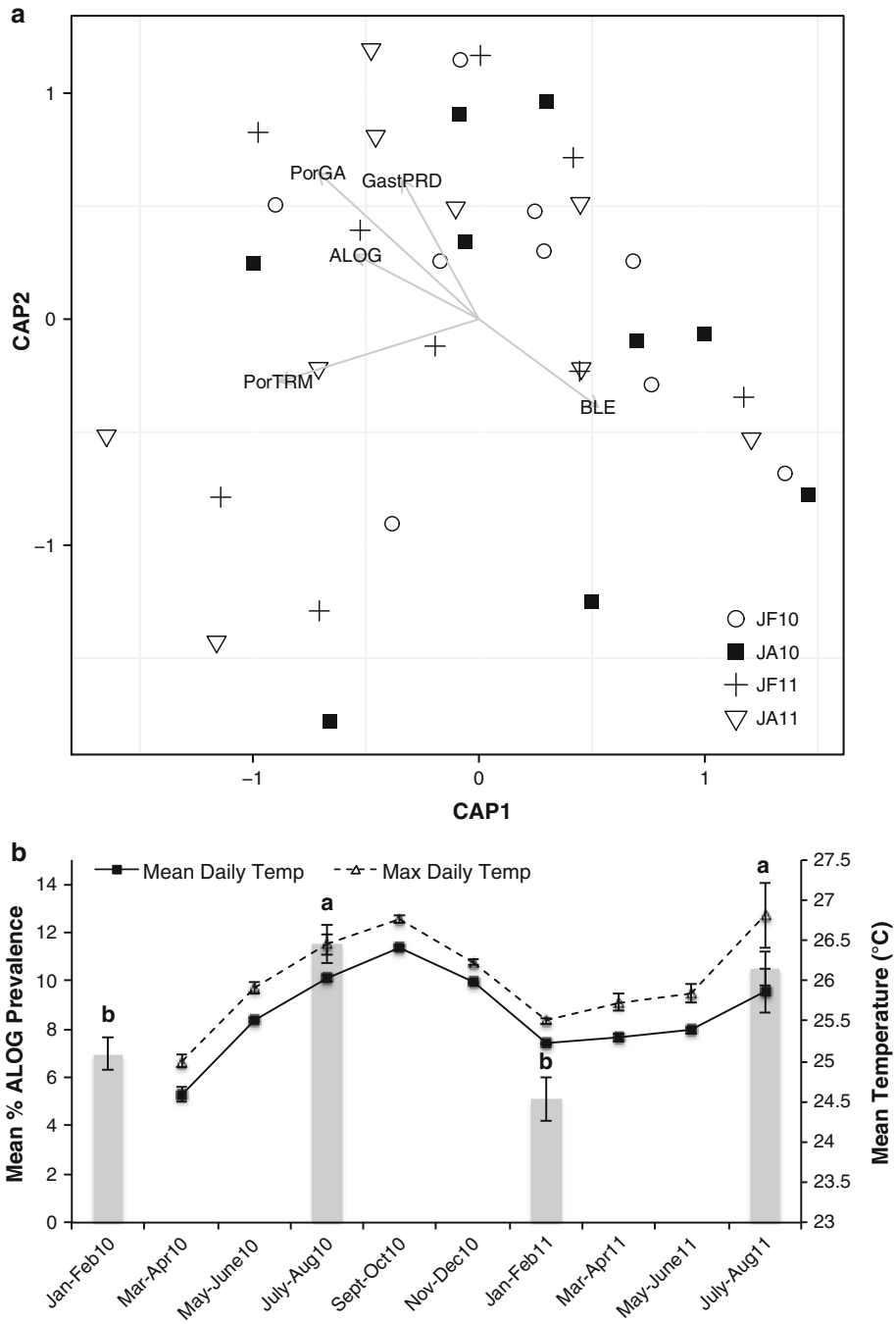

Fig. 2 **a** Canonical analysis of principal coordinates (CAP) for average prevalence of coral conditions at shallow sites in January–February 2010 (JF10), July–August 2010 (JA10), January–February 2011 (JF11), and July–August 2011 (JA11). Ordination is based on the Bray–Curtis coefficient of square-root transformed prevalence data. Bi-plot indicates the coral health conditions (vectors) exerting the strongest influence on the patterns in multivariate space (in *gray*). The length and direction of each vector indicates the strength and sign, respectively, of the relationship between that coral health condition and the CAP axes (only Pearson's correlations > 0.5 are shown). PorGA: *Porites* growth anomaly; PorTRM: *Porites* trematodiasis; ALOG: algal overgrowth; GastPRD: gastropod predation; BLE: bleaching. **b** Algal overgrowth (ALOG) (*gray bars*), bi-monthly mean ± SE daily temperature (*solid line*), and mean ± SE maximum daily temperature (*dashed line*) across all shallow sites. *Letters* indicate significant differences between survey months for each condition (post hoc HSD tests $P < 0.5$)

Seasonal patterns in coral health and temperature

Along WHI, we recorded significant but minimal temporal fluctuation in coral health assemblages. We detected evidence of seasonality in ALOG, which was 1.7–2 times higher in summer compared to winter months and consistent with enhanced macroalgal growth during summer months on other MHI reefs (Stimson et al. 1996). While ALOG along WHI was not correlated with seasonal changes in temperature, algal growth, and coral–algal interactions may also be influenced by fluctuation in other environmental conditions such as nutrients and UV irradiation (Stimson et al. 1996; Smith et al. 2005). The lack of seasonality in disease is particularly interesting as some Indo-Pacific diseases are often promoted during seasonal increases in temperature and UV irradiation (e.g., Boyett et al. 2007; Bruno et al. 2007; Sato et al. 2009). Along WHI, PorTLS prevalence increased marginally but not significantly in summer months, which is consistent with patterns across the Hawaiian Archipelago (Aeby et al. 2011a). One explanation for the lack of

Table 3 Summary of PERMANOVA results testing effects of site × depth and univariate tests for coral health conditions identified as indicator conditions in CAP

Source of variation	*df*	MS	Pseudo-*F*	*P* (from permutations)
PERMANOVA				
Site	7	0.25	11.05	<0.001
Depth	1	0.28	12.61	<0.001
Site × depth	7	0.13	5.93	<0.001
Residual	48	0.02		
Univariate permutational ANOVAs				
PorGA				
Site	7	8.23	17.8	<0.001
Depth	1	3.94	8.53	0.0053
Site × depth	7	8.05	17.41	<0.001
PorTRM				
Site	7	13.13	35.08	<0.001
Depth	1	10.94	29.21	<0.001
Site × depth	7	5.06	13.52	<0.001
DC				
Site	7	3.52	8.27	<0.001
Depth	1	13.27	31.16	<0.001
Site × depth	7	1.81	4.24	0.0011
GastPRD				
Site	7	2.5	12.97	<0.001
Depth	1	0.37	1.92	0.1758
Site × depth	7	4.47	3.32	0.0051
ALOG				
Site	7	4.96	14.12	<0.001
Depth	1	4.47	12.74	<0.001
Site × depth	7	0.89	2.54	0.025

Analyses only include January–February 2011 data

seasonality is that Hawai'i's higher latitude, exposure to deep oceanic waters and regular flushing from high wave energy, reduces the threat of severe thermal stress (daily max = 27 °C, this study) observed in regions such as the Great Barrier Reef (daily max = 32 °C, Sato et al. 2009). This pattern could also reflect the lack of temperature sensitivity of etiological agents associated with tissue loss diseases in Hawai'i, which was recently demonstrated in *Montipora* white syndrome in Kāne'ohe Bay (Ushijima et al. 2014). While Hawai'i has been somewhat protected from extreme thermal stress associated with climate change, Hawaiian corals have a lower temperature threshold than corals at lower latitudes (Jokiel and Coles 1990). With global temperatures predicted to exceed the bleaching threshold of 1 °C summer maxima by 2100 (Hoegh-Guldberg 1999; IPCC 2007), it is increasingly important to monitor coral disease on subtropical reefs.

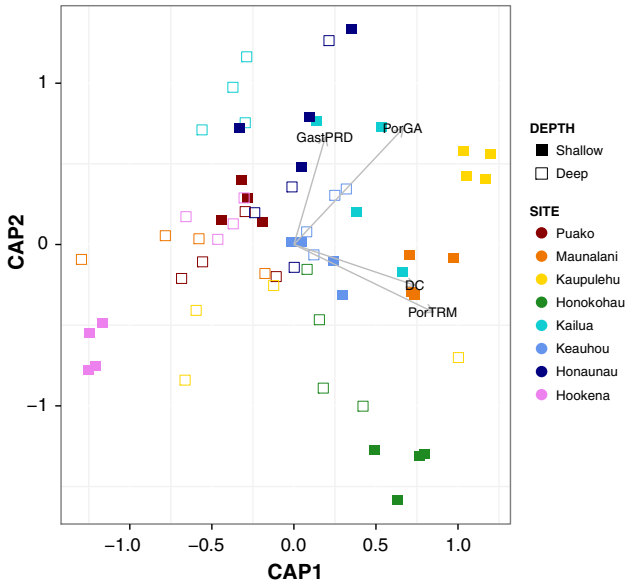

Fig. 3 Canonical analysis of principal coordinates (CAP) for prevalence of coral health conditions at eight sites (*different colors*) in shallow (*open squares*) and deep zones (*closed squares*) (3–4 transects/depth/site). Ordination is based on the Bray–Curtis coefficient of square-root transformed prevalence data. Bi-plot indicates the coral health conditions (vectors) exerting the strongest influence on the patterns in multivariate space (in *gray*). The length and direction of each vector indicates the strength and sign, respectively, of the relationship between that coral health condition and the CAP axes (only Pearson's correlations > 0.5 are shown). PorGA: *Porites* growth anomaly; PorTRM: *Porites* trematodiasis; PorTLS: *Porites* tissue loss syndrome; PorMFTL: *Porites* multifocal tissue loss syndrome; PocTLS: *Pocillopora* tissue loss; ALOG: algal overgrowth; GastPRD: gastropod predation; DC: discoloration; BLE: bleaching

Table 4 Long-term percentage of coral cover and change in coral cover between 2003 and 2011 in deep sites (WHAP sites) by site and overall

Site	2003	2007	2011	2011–2003	
				% cover	HPD intervals
Puakō	46.22	47.83	34.21	−12.01	−0.899, 7.940
Mauna Lani	38.12	31.21	28.43	**−9.69***	**2.728, 8.296**
Ka'ūpūlehu	38.29	31.15	27.05	**−11.24***	**0.138, 11.749**
Honōkohau	44.26	48.55	48.32	4.06	−7.974, 2.444
Kailua	52.28	61.86	46.55	−5.73	−5.546, 3.037
Keauhou	30.19	31.1	28	−2.19	−6.608, 7.670
Ke'ei	28.94	28.67	26.7	−2.24	−5.687, 7.539
Ho'okena	34.02	39.62	38.94	**4.92***	**−6.791, −0.276**
Overall	39.04	40.00	34.78	−4.26	−1.30, 3.42

Highest posterior density (HPD) intervals that do not overlap 0

* and bold values indicate significant changes in coral cover

Hōnaunau is not included due to insufficient within site replication

Spatial patterns in disease and biological interactions

We observed considerable spatial heterogeneity in coral health assemblages along WHI with coral health primarily driven by PorGA, PorTRM, DC, ALOG, and GastPRD. While the goal of this study was to describe the patterns rather than the processes affecting coral health, these patterns are likely governed by intrinsic (e.g., host) and extrinsic (e.g., environmental) factors specific to each condition. Consistent with previous MHI studies, PorGA prevalence was significantly higher in shallow zones (Domart-Coulon et al. 2006; Stimson 2010; Williams et al. 2010a; Aeby et al. 2011a). In concurrent WHI research, colony size was the strongest demographic predictor of PorGA prevalence with 40 % of the spatial variation explained by depth × size interactions (Couch 2014). This suggests that elevated PorGA prevalence in shallow zones may be partially due to larger average colony size on shallow reefs. While GA etiology is still unclear, PorGA prevalence has been positively correlated with UV radiation anomalies (Aeby et al. 2011a) and may be the result of elevated damage to UV-absorbing compounds in shallow zones (Coles and Seapy 1998). However, this hypothesis has not been supported experimentally (Stimson 2010) and does not explain the lack of PorGA seasonality along WHI. PorGA prevalence has also been linked with local stressors and was positively correlated with human population density (Aeby et al. 2011b), nutrient concentration (e.g., Kaczmarsky and Richardson 2010), and proximity to submarine groundwater (Walsh et al. 2013). Elevated PorGA prevalence in shallow zones was also primarily driven by anomalously high prevalence in three of the eight sites, suggesting that specific site-level factors are driving prevalence.

PorTRM was significantly elevated in shallow zones but also demonstrated unique site × depth interactions. Caused by the digenetic trematode, *Podocotyloides stenometra* (Aeby 1998), PorTRM dynamics are influenced by the distribution of the parasite and its *Porites*, molluscan, and butterflyfish hosts (Aeby 1998, 2007). Across WHI and Kāne'ohe Bay reefs, PorTRM prevalence is highest at intermediate coral abundance (Aeby 2007; Williams et al. 2010a; Couch 2014) and positively related to butterflyfish density (Williams et al. 2010a), suggesting that density-dependent transmission of the trematode *P. stenometra* is necessary for persistence of this disease.

As the primary contributors to coral mortality, ALOG was significantly higher in shallow zones. ALOG was primarily the result of red turf algae, morphologically similar to *Corallophila huysmansii,* and *Anotrichium tenue* (ESM Fig. 1c) and commonly associated with tissue mortality across the Pacific (Jompa and McCook 2003; Willis et al. 2004; Myers and Raymundo 2009). While these algae are hypothesized to excrete allelotoxic compounds used to overgrow coral tissue (Jompa and McCook 2003) and may be respond to increased UV irradiation in shallow zones, the processes governing their growth and distribution are unknown. Our results highlight that while invasive macroalgal blooms have garnered the most attention in Hawai'i due the speed and scale at which they alter communities, the role of native algal species in colony-level health should not be underestimated.

Alternatively, GastPRD did not vary significantly between depths. Along WHI, *D. cornus* consumed *Porites, Pocillopora*, and *Montipora*, with *C. violacea* only found on *Porites.* While we did not measure gastropod density during this study, their distribution was likely determined by coral community structure. For example, drupellids typically prefer highly complex habitats found in branching corals, which offer more shelter from predation and waves (Schoepf et al. 2010; Al-Horani et al. 2011). To our knowledge, this is the first study to highlight spatial variation of GastPRD in Hawai'i. There are no reported *Drupella* outbreaks in Hawai'i, but this corallivore is found throughout the Hawaiian Archipelago (G. Aeby and B. Vargas-Ángel pers. comm.).

DC was also significantly elevated in shallow zones and varied significantly as a function of site and the interaction. While we observed a variety of discolored lesions, 75 % of these lesions were associated *Porites* pigmentation response (data not shown), which is characterized by pink, often swollen tissue (Willis et al. 2004). Tissue DC is one of the common signs of compromised health in scleractinians and is often attributed to a variety of localized stressors (Aeby 1998; Willis et al. 2004; Raymundo et al. 2005; Ravindran and Raghukumer 2006). Because DC has also been linked to the production of physiochemical barriers, it has been attributed to a generalized defense response (Palmer et al. 2008). Elevated DC prevalence in shallow zones, as well as site-level variation may be explained by a combination of *Porites* abundance and localized disturbances (e.g., high wave action, abrasion, and algal overgrowth), which are typically elevated in shallow zones.

Sites of concern for coral reef management

Consistent with coral community dynamics across the state (K. Rodgers pers. comm), mean percentage of coral cover along WHI did not change significantly between 2003 and 2011. However, three WHI sites (Puakō, Mauna Lani, and Ka'ūpūlehu) experienced coral cover loss close to or above the proposed 10 % threshold that can be attributed to natural cycles (Jokiel et al. 2004). While significant coral cover decline has occurred in the Kohala District as a result of sedimentation (Walsh et al. 2013), coral cover loss may be linked other local stressors. Despite WHI's reputation as one of the most intact sections of reef in the Hawaiian

Archipelago, our study highlights common patterns of coral cover decline and impaired coral health at several regions.

Puakō and Mauna Lani, connected by the same reef system in South Kohala, have experienced 12.01 and 9.69 % coral cover loss, respectively, (Table 4). These results are consistent with a reported 48 % decline in Puakō's coral cover during the last 40 yrs, as well as substantial declines in fish biomass, abundance, and diversity (Minton et al. 2012). During the last 60 yrs, this region has experienced extensive coastal development, as well as improved access for tourists and fishing activities (Minton et al. 2012). At the colony level, high DC prevalence also suggests elevated localized disturbance. Declining reef health may also be linked to and/or exacerbated by the high ALOG (second and third highest along the coast) as well as chronic diseases (e.g., PorGA).

Another location of interest is Ka'ūpūlehu. Kahuwai Bay (the shallow zone within Ka'ūpūlehu) in particular has the highest PorGA prevalence (41 %), moderate levels of DC (5 %), and 12 % of all colonies partially overgrown by algae. This reef is situated within an embayment whose watershed has undergone extensive resort development during the last 25 yrs, yet it is still unclear whether these activities have affected reef condition. Ka'ūpūlehu's deep zone also experienced an 11.24 % loss in coral cover following strong winter storms in 2004 and negligible recovery (Walsh et al. 2013). Reef recovery may also be further hampered by one of the few reported COTS outbreaks (59 individuals 200 m^{-2}) along WHI (Walsh et al. 2013).

Reef condition is also of concern in Hōnaunau Bay. Although coral cover appeared unchanged between 2008 (42.96 %) and 2011 (42.26 %; W. Walsh pers. comm.), 22 % of all *Porites* spp. had GAs, and perhaps more concerning was that this region had the highest level of ALOG (17.07 %) and GastPRD, namely drupellid and corallivores (6.52 %). Blooms of *Leptolyngbya crosbyana* have also caused recent widespread mortality of *Porites compressa* in Hōnaunau (Smith et al. 2008) and continue to affect these reefs. Small-scale agriculture, poor sewage treatment, and an especially high snorkeling, recreational diving, and boating primarily affect the Hōnaunau watershed and adjacent coastal waters.

One of the common characteristics of all four regions is evidence of coastal eutrophication with summer phytoplankton blooms (authors, pers. obs.) and/or unusually high $\delta^{15}N$ values (Dailer et al. 2011). Given the strong negative correlation between GAs and water motion (Burns et al. 2011; Couch 2014), local stress may be exacerbated in embayments such as Ka'ūpūlehu and Hōnaunau, which have restricted water flow compared to exposed regions. While impaired coral health in these regions is concerning, the synergistic effects of reduced top-down and unregulated bottom-up stressors, restricted water flow, and elevated terrestrial input render all of WHI particularly vulnerable to reef decline.

In conclusion, Pacific coral disease has become a major threat to reefs but is not the only ecological process affecting coral health. One of the challenges facing coral reef managers is identifying reefs at risk and minimizing local stressors. Our study highlights that ongoing coral cover loss and impaired coral health at the colony level may affect long-term WHI reef condition. Chronic diseases such as PorGA and PorTRM generally do not result in rapid colony mortality but can impair coral growth (Aeby 1991; Stimson 2010) and reproduction (Domart-Coulon et al. 2006; Work et al. 2008a), potentially affecting long-term coral population demographics. The high prevalence and considerable spatial variation in coral health not only allowed us to target sites for future reef management (Puakō, Mauna Lani, Ka'ūpūlehu and Hōnaunau) but also highlights the importance of considering other biological interactions/compromised health states when investigating processes influencing colony-level health. This study advances our understanding of coral disease ecology and supports reef resilience planning by identifying vulnerable areas that would benefit most from targeted conservation and management efforts.

Acknowledgments Special thanks to K. Gaab, N. Rozet, L. Kramer, G. Key, R. Martin, B. Carmen, K. Osada-D'Avella, and Kona Diving Company for field support. We also thank C.D. Harvell, G. Aeby, E. Weil, L. Raymundo, and M. Takabayashi for helpful feedback on project development. We are grateful to D. Ruiz Moreno and M. Donahue for assistance with statistical analyses. This study was generously supported by The Kohala Center, The NOAA Coral Reef Conservation Grant Program (Grant # NA09NMF4630121 to C.D. Harvell & NA09NOS4260100 to Hawai'i DAR), NSF Graduate Research Fellowship awarded to C. Couch and Cornell University. Thanks to C.D. Harvell, E. Peters, M. Mouchka, A. Tracy, C. Burge, I. Hewson, and J. Thaler for manuscript review.

References

Aeby G (1991) Behavioral and ecological relationships of a parasite and its hosts within a coral reef system. Pac Sci 45:263–269

Aeby GS (1998) A digenean metacercaria from the reef coral, *Porites compressa*, experimentally identified as *Podocotyloides stenometra*. J Parasitol 84:1259–1261

Aeby GS (2007) Spatial and temporal patterns of infection of *Porites* trematodiasis on the reefs of Kaneohe Bay, Oahu, Hawaii. Bull Mar Sci 80:209–218

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

Aeby GS, Williams GJ, Franklin EC, Kenyon J, Cox EF, Coles S, Work TM (2011a) Patterns of coral disease across the Hawaiian Archipelago: relating disease to environment. PLoS ONE 6:e20370

Aeby G, Williams GJ, Franklin EC, Haapkyla J, Harvell CD, Neale S, Page CA, Raymundo LJ, Vargas-Angel B, Willis BL, Work TM, Davy SK (2011b) Growth anomalies on the coral genera *Acropora* and *Porites* are strongly associated with host density and human population size across the Indo-Pacific. PLoS ONE 6:e16887

Al-Horani FA, Hamdi M, Al Rouson SA (2011) Prey selection and feeding rates of *Drupella cornus* (Gastropoda: Muricidae) on corals from the Jordanian Coast of the Gulf of Aqaba, Red Sea. Jordan Journal of Biological Sciences 4:191–198

Anderson MJ (2001) A new method for nonparametric multivariate analysis of variance. Austral Ecol 26:32–46

Anderson MJ, Willis TJ (2003) Canonical analysis of principal coordinates: a useful method of constrained ordination for ecology. Ecology 84:511–525

Anderson MJ, Crist TO, Chase JM, Vellend M, Inouye BD, Freestone AL, Sanders NJ, Cornell HV, Comita LS, Davies KF (2011) Navigating the multiple meanings of β diversity: a roadmap for the practicing ecologist. Ecol Lett 14:19–28

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

Baayen RH, Davidson DJ, Bates DM (2008) Mixed-effects modeling with crossed random effects for subjects and items. J Mem Lan 59:390–412

Battista TA, Costa BM, Anderson SM (2007) Shallow-water benthic habitats of the main eight Hawaiian Islands (DVD). In: NOAA Technical Memorandum NOS NCCOS 61 BB (ed), Silver Spring, MD

Beeden R, Willis BL, Raymundo L, Page CA, Weil E (2008) Underwater cards for assessing coral health on Indo-Pacific reefs. Global Environmental Facility Coral Reef Targeted Research Program, St. Lucia

Birkeland C (1997) Life and death of coral reefs. Springer

Bolker BM, Brooks ME, Clark CJ, Geange SW, Poulsen JR, Stevens MHH, White JSS (2009) Generalized linear mixed models: a practical guide for ecology and evolution. Trends Ecol Evol 24:127–135

Boyett HV, Bourne DG, Willis BL (2007) Elevated temperature and light enhance progression and spread of black band disease on staghorn corals of the Great Barrier Reef. Mar Biol 151:1711–1720

Bruno JF, Selig ER, Casey KS, Page CA, Willis BL, Harvell CD, Sweatman H, Melendy AM (2007) Thermal stress and coral cover as drivers of coral disease outbreaks. PLoS Biol 5:1220–1227

Burns JHR, Rozet NK, Takabayashi M (2011) Morphology, severity, and distribution of growth anomalies in the coral, *Montipora capitata*, at Wai'Opae, Hawai'i. Coral Reefs 30:819–826

Coles SL, Seapy DG (1998) Ultra-violet absorbing compounds and tumorous growths on acroporid corals from Bandar Khayran, Gulf of Oman, Indian Ocean. Coral Reefs 17:195–198

Couch CS (2014) Intrinsic host and extrinsic environmental factors of coral health and disease. Ph.D. thesis, Cornell University, p 224

Dailer M, Smith C, Glenn C (2011) Preventing the introduction and spread of nutrient driven invasive algal blooms and coral reef degradation in West Hawai'i. Final Report to the Hawai'i Coral Reef Initiative (9/30/2011). Grant Number NA09NOS4260242

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

Domart-Coulon IJ, Traylor-Knowles N, Peters E, Elbert D, Downs CA, Price K, Stubbs J, McLaughlin S, Cox E, Aeby G, Brown PR, Ostrander GK (2006) Comprehensive characterization of skeletal tissue growth anomalies of the finger coral *Porites compressa*. Coral Reefs 25:531–543

Done TJ (1992) Phase shifts in coral reef communities and their ecological significance. In: Jaccarini V, Martens E (eds) The ecology of mangrove and related ecosystems. Springer, pp 121–132

Franklin EC, Jokiel PL, Donahue MJ (2013) Predictive modeling of coral distribution and abundance in the Hawaiian Islands. Mar Ecol Prog Ser 481:121–132

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

Harvell CD, Jordán-Dahlgren E, Merkel S, Rosenberg E, Raymundo L, Smith GW, Weil E, Willis BL (2007) Coral disease, environmental drivers and the balance between coral and microbial associates. Oceanography 20:58–81

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

Hoegh-Guldberg O (1999) Climate change, coral bleaching and the future of the world's coral reefs. Mar Freshw Res 50:839–866

Hughes TP (1994) Catastrophes, phase shifts, and large scale degradation of a Caribbean coral reef. Science 265:1547–1551

Hunter CL, Evans CW (1995) Coral reefs in Kaneohe Bay, Hawaii: two centuries of western influence and two decades of data. Bull Mar Sci 57:501–515

IPCC (2007) Climate Change 2007: The Physical Basis. Contribution of Working Group I to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change, IPCC Geneva

Jokiel P, Coles S (1990) Response of Hawaiian and other Indo-Pacific reef corals to elevated temperature. Coral Reefs 8:155–162

Jokiel PL, Brown EK, Friedlander A, Rodgers K, Smith WR (2004) Hawai'i coral reef assessment and monitoring program: spatial patterns and temporal dynamics in reef coral communities. Pac Sci 58:159–174

Jompa J, McCook LJ (2003) Coral-algal competition: macroalgae with different properties have different effects on corals. Mar Ecol Prog Ser 258:87–95

Kaczmarsky LT (2006) Coral disease dynamics in the central Philippines. Dis Aquat Org 69:9–21

Kaczmarsky L, Richardson LL (2010) Do elevated nutrients and organic carbon on Philippine reefs increase the prevalence of coral disease? Coral Reefs 30:253–257

Kim K, Harvell CD (2004) The rise and fall of a six-year coral-fungal epizootic. Am Nat 164:S52–S63

Kittinger JN, Pandolfi JM, Blodgett JH, Hunt TL, Jiang H, Maly K, McClenachan LE, Schultz JK, Wilcox BA (2011) Historical reconstruction reveals recovery in Hawaiian coral reefs. PLoS ONE 6:e25460

Kohler KE, Gill SM (2006) Coral Point Count with Excel extensions (CPCe): A Visual Basic program for the determination of coral and substrate coverage using random point count methodology. Comput Geosci 32:1259–1269

Minton D, Conklin E, Weiant P, Wiggins C (2012) 40 years of decline on Puako's coral reefs: A review of historical and current data (1970–2010). Honolulu, The Nature Conservancy, p 140

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

Nicolet K, Hoogenboom M, Gardiner N, Pratchett M, Willis B (2013) The corallivorous invertebrate *Drupella* aids in transmission of brown band disease on the Great Barrier Reef. Coral Reefs 32:585–595

Nugues M, Bak R (2009) Brown-band syndrome on feeding scars of the crown-of-thorn starfish *Acanthaster planci*. Coral Reefs 28:507–510

Oren U, Brickner I, Loya Y (1998) Prudent sessile feeding by the corallivore snail, *Coralliophila violacea* on coral energy sinks. Proc R Soc B-Biol Sci 265:2043–2050

Palmer CV, Mydlarz LD, Willis BL (2008) Evidence of an inflammatory-like response in non-normally pigmented tissues of two scleractinian corals. Proc R Soc B-Biol Sci 275: 2687–2693

Ravindran J, Raghukumer C (2006) Pink-line syndrome, a physiological crisis in the scleractinian coral *Porites lutea*. Mar Biol 149:347–356

Raymundo LJ, Rosell KB, Reboton C, Kaczmarsky L (2005) Coral diseases on Philippine reefs: genus *Porites* is a dominant host. Dis Aquat Org 64:181–191

Ruiz-Moreno D, Willis BL, Page AC, Weil E, Croquer A, Vargas-Angel B, Jordan-Garza AG, Jordan-Dahlgren E, Raymundo L, Harvell CD (2012) Global coral disease prevalence associated with sea temperature anomalies and local factors. Dis Aquat Org 100:249–261

Sato Y, Bourne DG, Willis BL (2009) Dynamics of seasonal outbreaks of black band disease in an assemblage of *Montipora* species at Pelorus Island (Great Barrier Reef, Australia). Proc R Soc B-Biol Sci 276:2795–2803

Schoepf V, Herler J, Zuschin M (2010) Microhabitat use and prey selection of the coral-feeding snail *Drupella cornus* in the northern Red Sea. Hydrobiologia 641:45–57

Smith JE, Runcie JW, Smith CM (2005) Characterization of a large-scale ephemeral bloom of the green alga *Cladophora sericea* on the coral reefs of West Maui, Hawai'i. Mar Ecol Prog Ser 302:77–91

Smith JE, Shaw M, Edwards RA, Obura D, Pantos O, Sala E, Sandrin SA, Smirga SS, Hatay M, Rohwer FL (2006) Indirect effects of algae on coral: algae-mediated, microbe-induced coral mortality. Ecol Lett 9:835–845

Smith JE, Kuwabara J, Coney J, Flanagan K, Beets J, Brown D, Stanton F, Takabayashi M, Duplessis S, Griesemer B (2008) An unusual cyanobacterial bloom in Hawaii. Coral Reefs 27:851

Stimson J, Larned S, McDermid K (1996) Seasonal growth of the coral reef macroalga *Dictyosphaeria cavernosa* (Forskål) Børgesen and the effects of nutrient availability, temperature and herbivory on growth rate. J Exp Mar Biol Ecol 196:53–77

Stimson J (2010) Ecological characterization of coral growth anomalies on *Porites compressa* in Hawai'i. Coral Reefs 30:133–142

Street JH, Knee KL, Grossman EE, Paytan A (2008) Submarine groundwater discharge and nutrient addition to the coastal zone and coral reefs of leeward Hawai'i. Mar Chem 109:355–376

Takabayashi M, Gregg TM, Farah E, Burns J, Teves K, Cody NH (2008) The prevalence of skeletal growth anomaly and other afflictions in scleractinian corals in Waiopae, Hawaii. Proc 11th Int Coral Reef Symp:820–824

Tissot BN, Walsh WJ, Hallacher LE (2004) Evaluating effectiveness of a marine protected area network in West Hawai'i to increase productivity of an aquarium fishery. Pac Sci 58:175–188

Turner S (1994) Spatial variability in the abundance of the corallivorous gastropod *Drupella cornus*. Coral Reefs 13:41–48

US Census Bureau (2007) County Population Facts Hawaii Population Estimate: 2007

Ushijima B, Videau P, Burger A, Shore-Maggio A, Runyon CM, Sudek M, Aeby GS, Callahan SM (2014) *Vibrio coralliilyticus* strain OCN008 is an etiological agent of acute *Montipora* white syndrome. Appl Environ Microbiol:AEM. 03463–03413

Vargas-Angel B (2009) Coral health and disease assessment in the US Pacific Remote Island Areas. Bull Mar Sci 84:211–227

Vargas-Ángel B, Wheeler B (2009) Coral health and disease assessment in the US Pacific Territories and Affiliated States. Proc 11th Int Coral Reef Symp:175–179

Walsh WJ, Cotton S, Barnett C, Couch CS, Preskitt L, Tissot B, Osada-D'Avella K (2013) Long-term monitoring of coral reefs of the Main Hawaiian Islands. Final report 2009 NOAA Coral Reef Conservation Report; NA06NOS4260113 97

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

Williams GJ, Work TM, Aeby GS, Knapp IS, Davy SK (2010b) Gross and microscopic morphology of lesions in Cnidaria from Palmyra Atoll, Central Pacific. J Invertebr Pathol 106:165–173

Willis BL, Page CA, Dinsdale EA (2004) Coral disease on the Great Barrier Reef. In: Rosenberg E, Loya Y (eds) Coral health and disease. Springer-Verlag, Berlin, pp 69–104

Work TM, Aeby GS (2006) Systematically describing gross lesions in corals. Dis Aquat Org 70:155–160

Work TM, Aeby GS, Coles SL (2008a) Distribution and morphology of growth anomalies in *Acropora* from the Indo-Pacific. Dis Aquat Org 78:255–264

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