BIOTROPICA 45(3): 401–407 2013 10.1111/btp.12019

Indirect Effects of Human Development Along the Coast on Coral Health

C. Guilherme Becker[1,3], **Benjamin D. Dalziel**[1], **Mônica F. Kersch-Becker**[1], **Mia G. Park**[2], and **Morgan Mouchka**[1]

[1] Department of Ecology and Evolutionary Biology, Cornell University, Ithaca, NY 14853, U.S.A.

[2] Department of Entomology, Cornell University, Ithaca, NY 14853, U.S.A.

ABSTRACT

Accelerated human development in coastal areas is causing declines in coral reefs worldwide, but the mechanisms by which development leads to reef degradation are often difficult to identify. Here, we use Akaike information criterion model averaging in combination with path analysis to test for the direct and indirect effects of potential environmental stressors on two coral diseases on the west coast of Hawai'i. We quantified human-altered land cover and human population density at two spatial scales: inland area of 1-km radius and watershed. We then tested for the effects of these land cover variables, as well as seawater chlorophyll α concentrations, depth, host coral cover, and proximity to surface water discharge on the density of growth anomalies (GA) and prevalence of trematodiasis (TRE) affecting *Porites lobata,* the locally dominant reef-building coral species. Our analyses showed that human-altered land cover measured at 1-km scale was a strong indirect predictor of GA. Specifically, we found that human interference adjacent to the coast predicted higher chlorophyll α concentrations, which in turn predicted higher GA density. We also found that chlorophyll α and depth were strong negative predictors of TRE, and host coral cover a positive predictor. Our results indicated that GA are likely regulated by indirect land-based anthropogenic impacts, whereas TRE is mostly affected by host density-dependent forces. Path analysis can serve as a useful tool to rapidly identify the scale and indirect effects of anthropogenic stressors related to coral diseases, allowing for accurate conservation planning in the face of limited resources for tropical conservation.

Key words: growth anomalies; landscape; pollution; *Porites*; reef health; trematodiasis.

Coral reef ecosystems are declining worldwide, exhibiting severe decreases in abundance and diversity (Jackson 2001, Harvell *et al.* 2002, Hoegh-Guldberg *et al.* 2007, Carpenter *et al.* 2008). Declines have been attributed to human exploitation, land-based pollution, climate change, and disease outbreaks (Rogers 1990, Hughes 1994, Jackson 1997, Jackson *et al.* 2001, Harvell *et al.* 2002, Hughes *et al.* 2003, Rodgers & Cox 2003, Wolanski *et al.* 2003, Jokiel *et al.* 2004, Orr *et al.* 2005, Hoegh-Guldberg *et al.* 2007, Carpenter *et al.* 2008); all of which may act synergistically to exacerbate coral reef degradation. Disease outbreaks, for instance, can be triggered by land-based pollution (Haapkyla *et al.* 2011) and increased sea-surface temperatures (Kim & Harvell 2002, Bruno *et al.* 2007, Williams *et al.* 2010), yet there is still a need to link patterns and processes to better understand the mechanisms driving coral reef declines.

A recent study found a positive association between human population density and growth anomalies (hereafter GA) in corals of the locally abundant genus *Porites* across the Indo-pacific (Aeby *et al.* 2011a). GA are tumor-like abnormalities that result in reduced coral growth (Cheney 1975), partial colony mortality, and decreased host fitness due to reductions in fecundity, feeding potential, and defense mechanisms (Work *et al.* 2008, McClanahan *et al.* 2009). These anomalies have been widely documented throughout the Hawaiian archipelago and are most prevalent in *Porites* (Domart-Coulon *et al.* 2006, Friedlander *et al.* 2008, Williams *et al.* 2010, Stimson 2011). The genus *Porites* is also susceptible to infections by the trematode *Podocotyloides stenometra* Pritchard (Cheng & Wong 1974, Aeby 1998), which is known to cause *Porites* trematodiasis (hereafter TRE). TRE has also been documented throughout the Hawaiian Islands (Aeby *et al.* 2011b) and is characterized by pink swollen nodules that can cause reductions in colony growth of up to 50 percent, (Aeby 1991) leading to decreases in host fitness. Contrary to the pattern reported for GA, TRE is expected to be more prevalent in more pristine reefs where biodiversity is higher (Aeby *et al.* 2011b), as the pathogen has a complex life cycle and requires stable populations of three obligate hosts: a molluscan, *Porites*, and coral-feeding fish (Aeby 2007).

The Big Island of Hawai'i is currently experiencing rapid rates of human population and economic growth, with lava fields converted to resorts, residential areas, and golf courses (U.S. Census Bureau 2010). As human development is relatively new, pockets of natural coastline still remain, creating a mosaic landscape with a wide range of levels of human development. Recent work suggests that coral populations on the island are being impacted by anthropogenic development (Jokiel *et al.* 2004), which has increased land-based pollution (Hamnett *et al.* 2006). The combination of relatively low coral species diversity and varying levels of human interference on the landscape makes Hawai'i a useful system to examine the links between human development and coral disease.

Rapid changes in coral health often necessitate the brisk design and implementation of research programs aimed at improving

Received 27 April 2012; revision accepted 2 October 2012.

[3]Corresponding author; e-mail: cgb58@cornell.edu

conservation strategies. However, rapidly collected data afford less statistical power to detect the effect of the multiple interacting factors on coral health. Traditional analyses based on pairwise correlations between distinct response and predictor variables, such as general linear models (GLMs), may thus not positively identify important causal relationships in data collected during a rapid response to an ecological emergency. Path analysis differs from GLMs in that it allows us to account for indirect causal associations among explanatory variables (Shipley 2002), assessing the relative strength of each effect explaining disease risk in our focal coral species. Here, we modeled both GA density and TRE prevalence on *Porites lobata* in relation to several reef- and land-based explanatory variables at two spatial scales (*i.e.*, *P. lobata* cover, water chlorophyll α concentrations [a proxy for anthropogenic nutrient input], depth, distance to nearest surface water discharge, human population density, and human-altered land cover). As GA and TRE demonstrate very different disease dynamics, with GA prevalence being more prone to be driven by anthropogenic stress than TRE (Aeby 2007, Aeby *et al.* 2011a,b), we were interested in taking a comparative approach to determine how our analyses would predict the direct and indirect factors that influence disease. Considering both direct and indirect associations between variables can enhance our ability to examine disease dynamics of reefs exposed to anthropogenic stress.

METHODS

STUDY SYSTEM.—We surveyed eight reefs spanning 107 km of the west coast of the Big Island of Hawai'i HI, USA, representing varying levels of human development (Fig. S1). Sites were accessible by land and shallow enough to be surveyed by snorkeling. We focused on the massive coral *Porites lobata* Dana (1846), the dominant reef-building species in shallow reefs of West Hawai'i (Gosliner *et al.* 1996). *Porites lobata* grows to form large lobes, and the colony itself can cover several meters.

SAMPLING DESIGN.—At each site, we randomly assigned three 10-m transects (maximum distance among transects = 200 m) to areas with the greatest proportion of continuous reef at snorkeling depth ($\leq$ 3 m). For each transect, we placed four 50 $\times$ 50 cm quadrats on each side of the transect line for a total of 8 quadrats per transect (Coyer & Witman 1990). Within each quadrat, the area of *P. lobata* cover, the number of GAs, and the presence/absence of TRE lesions were counted. We then calculated both GA density as the number of tumors per square meter of *P. lobata*, and TRE prevalence as the proportion of quadrats where *P. lobata* was infected by *Podocotyloides stenometra,* characterized by pink nodules on coral heads (Aeby 1998). We averaged transect data for GA density and TRE prevalence within each sampling location (N = 8) for all analyses. We conducted all surveys in January 2009 to avoid seasonal effects in our disease dataset.

LAND-BASED METRICS.—Using freely available spatial information, we assessed several land-base metrics at both inland area of 1-km radius around sampling locations and at the watershed level (*i.e.*, area delimited by ridgelines that includes rivers and major streams flowing in the same direction [GDSI – Geographic Decision System International 1995]; average watershed area = 549.01 $\pm$ 555.21 km^2 SD). The local scale data (1-km radius) can be informative if land-based stressors occur mainly close to the shoreline. Watershed scale data can be more useful to detect potential effect of land-based pollution on corals, as the drainage basin is the studied unit. Thus, extracting spatial information at these two scales can provide us with a better picture of the mechanisms and extent of potential anthropogenic influence on coral health.

We used land cover data (NOAA – National Oceanic & Atmospheric Administration 2011) produced using 2.4 m Quickbird® satellite imagery from circa 2005 to extract data on human-altered land cover using Arc View 9.3 (ESRI 2009). This metric was calculated as the percentage of both urban and agricultural lands (*e.g.*, cattle fields, crops) at both watershed level and inland area of 1-km radius from each sampling location (Fig. S1). We calculated human population density (individuals per square km) using a 1-km resolution raster from Global-Urban Mapping Project v.1.0 (CIESIN – Center for International Earth Science Information Network, Columbia University, IFPRI – International Food Policy Research Institute, the World Bank, & CIAT – Centro Internacional de Agricultura Tropical 2011). Although the resolution of this variable can be considered coarse when measured at 1-km radius, these are the best available data for our analyses. We also used GIS information on surface flowing freshwater (GDSI – Geographic Decision System International 1995) to calculate the distance between each sampling site and the nearest surface water discharge.

REEF-BASED VARIABLES.—As a proxy for anthropogenic nutrient input, we measured average chlorophyll α concentrations (μg/mL) from the water within 15 cm of the reef at each sampling site. Three 1-L water samples (one per transect) were stored on ice and in the dark until processing (which was performed within 5 h of collection). We filtered 500 mL of sampled water through 0.45 μm Whatman GF/F glass fiber filters and then stored these filters in 20 mL of 90 percent ethanol in a refrigerator for 24 h. We used a fluorometer (Aquafluor™, Turner Designs, San Jose, CA, USA) to measure chlorophyll α concentration from the ethanol solutions (Maxwell & Johnson 2000). To avoid temporal effects on chlorophyll α assessment, all water samples were processed within 4 d. We also recorded the average depth (m) at all sampling sites because it may influence the interaction between land-base pollution and primary productivity, as both algal blooms and pollutants are more likely to affect shallow reefs than deep reefs (Lapointe *et al.* 2005).

STATISTICAL ANALYSES.—We used model averaging based on Akaike information criterion (AIC), including all land- and reef-based factors as explanatory variables, and coral disease (GA density or TRE prevalence) as response variables. Model averaging allows us to make inferences based on a set of candidate models, not just the best-fit model. This General Linear Model

(GLM) screening approach ranks parameter estimates from each possible model using AIC weights, and thus allows us to detect variables that will most likely influence each disease (JMP v. 10.0, SAS Institute, 2012; Cary, NC, USA). After this initial screening, we used the four variables with the highest estimate ranks associated to GA and TRE in path analyses (Shipley 2002). We started with the full biological relevant model, including causal associations among the top four variables selected in our initial model averaging screening, and then removed variables in turn until we reached the model with the maximum likelihood according to the Expected Cross-Validation Index (ECVI), an AIC-based index. We ran AIC model averaging using JMP v.10 (SAS 2012) and path analyses using Systat v.10.2 (Systat 2002).

RESULTS

Porites lobata cover ranged from 11 to 60 percent within the quadrats at each site. Density of growth anomalies (GA) ranged from 0.03 to 0.41 tumors/m^2 and trematodiasis (TRE) appeared in sampling quadrats with a frequency of zero to 63.64 percent. Our model averaging screening indicated that both GA and TRE were better predicted by reef-based than land-based variables (Table 1). We found that the top four most likely variables explaining GA were chlorophyll α concentrations, depth, *P. lobata* cover, and human-altered land cover at 1-km radius, whereas the best predictors of TRE were chlorophyll α concentrations, depth, *P. lobata* cover, and human population density at 1-km radius (Tables 1 and 2).

When analyzing the power of these explanatory variables using path analysis, we found a strong positive effect of chlorophyll α concentration on GA density in *P. lobata* (standardized path coefficient = 0.769 ± 0.251 SE, $t = 3.07$, $P < 0.05$; Fig. 1A). This positive association was mediated by an indirect effect of human-altered land cover associated with an increase in chlorophyll α concentration, a proxy for anthropogenic nutrient input (standardized path coefficient = 0.693 ± 0.196 SE, $t = 3.53$, $P < 0.01$; Fig. 1A). Therefore, the association between human-altered land cover and GA density only became evident after considering causal associations with chlorophyll α concentration, as the direct path linking human-altered land cover and GA is not significant in the path model or on a single linear regression ($F_{[1,6]} = 3.426$, $R^2 = 0.363$, $P = 0.114$). Depth had a non-significant influence on GA density when considered in the full path model and caused an increase in model likelihood when dropped.

The path model with the highest likelihood explaining TRE included chlorophyll α concentration and depth as negative predictors, and *P. lobata* cover as a positive predictor. In this case, indirect effects did not play a significant role explaining disease (Fig. 1B). Given the biological relevance of the association between human-altered land cover and chlorophyll α to explain GA (Fig. 1A), we also included this link in the path model to explain TRE. This additional path, however, only caused a decrease in model likelihood (path model including [ECVI = 3.778], and excluding [ECVI = 2.577] the link between human-altered land cover and chlorophyll α).

TABLE 1. *Model averaging results ranking the most important variables explaining growth anomalies (GA – tumors/m^2 of* Porites lobata*) and trematodiasis (TRE – prevalence of lesions per quadrats with* P. lobata*) according to AIC weights.*

Parameter Ranking	Estimate	Std Error
Growth Anomalies		
Intercept	12.176	–
Chlorophyll α (μg/mL)	1.282	0.397
Depth (m)	−0.493	0.913
P. lobata cover/m^2	0.122	2.911
Human-altered land cover [1000 m radius (%)]	0.039	0.074
Population density (Watershed [inhabitants/km^2])	0.006	0.038
Human-altered land cover (Watershed [%])	0.003	0.019
Population density (1000 m radius [inhabitants/km^2])	0.001	0.005
Distance to surface water discharge (m)	0.001	0.116
Trematodiasis		
Intercept	25.661	–
Depth (m)	−1.759	2.099
P. lobata cover/m^2	1.159	8.248
Chlorophyll α (μg/mL)	−0.275	0.365
Population density (1000-m radius [inhabitants/km^2])	0.067	0.032
Population density (Watershed [inhabitants/km^2])	−0.020	0.110
Human-altered land cover [Watershed (%)]	−0.016	0.052
Human-altered land cover (1000-m radius [%])	−0.016	0.114
Distance to surface water discharge (m)	0.010	0.307

DISCUSSION

Corals form the trophic and structural basis of reef ecosystems (Weis & Allemand 2009), making the entire reef community sensitive to coral disease dynamics (McClanahan *et al.* 2002, Hughes *et al.* 2003). At the same time, coral reefs provide ecological services essential to humans, safeguarding habitat for coastal fisheries, preventing coastal erosion, attracting tourism, and producing pharmacological compounds (Moberg & Folke 1999). As accelerated human development is concentrated in coastlines, it is important to rapidly determine the spatial scale and causative factors through which human development influences coral reefs. These factors often carry indirect effects, making it harder to disentangle the proximate causes of coral declines.

While we may be able to measure proximate relationships of reef deterioration, integrating ecological processes to predict human impacts over the reef community poses a greater challenge

TABLE 2. *Growth anomalies (GA – tumors/m^2 of* Porites lobata*), trematodiasis (TRE – prevalence of lesions per quadrats with* P. lobata*),* P. lobata *cover (*P. lobata *cover/m^2), chlorophyll α concentration (μg/mL), average reef depth (m), human-altered land cover (1000-m radius [%]), and population density (watershed scale [inhabitants/km^2]) across eight sites on the west coast of the big island of Hawai'i.*

Site	Lat.	Long.	GA	TRE	*P. lobata* cover	*Chlorophyll* α	*Depth*	*Human-altered LC*	*Pop. density*
Lapakahi	20.174	−155.900	0.12	25.00	0.11	0.05	2.00	2.86	0.18
Spencer	20.025	−155.823	0.41	26.09	0.47	14.18	1.00	23.07	8.12
Mau'u'mae	20.016	−155.823	0.20	0	0.36	9.22	3.00	16.69	6.56
Pauoa	19.952	−155.861	0.20	18.18	0.48	7.94	3.00	36.98	10.69
Anaeho'omalu	19.916	−155.887	0.27	6.67	0.24	14.80	1.50	23.46	19.54
Kahalu'u	19.580	−155.967	0.25	63.64	0.46	4.75	0.50	19.13	325.90
Manini'owali	19.471	−155.920	0.21	19.05	0.60	3.60	3.00	10.93	121.65
Ho'okena	19.379	−155.897	0.03	37.50	0.48	1.18	2.80	1.62	11.28

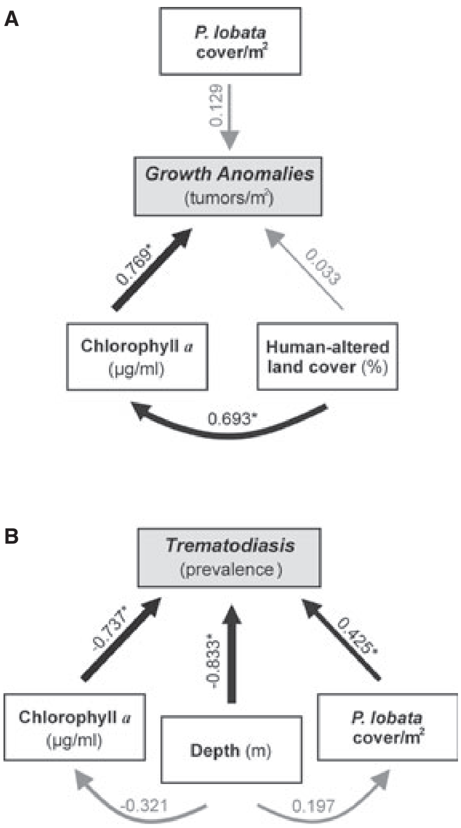

FIGURE 1. Path analysis models showing (A) the relative strengths of human-altered land cover, *Porites lobata* cover, and chlorophyll α on growth anomalies, and (B) the effects of chlorophyll α, *P. lobata* cover, and depth on trematodiasis in *P. lobata*. Numbers are standardized path coefficients (**P* < 0.05). The thickness of the arrows represents the relative strength of the relationship. Non-significant paths are shown in gray.

(Hughes *et al.* 2003). Here, we show that path analyses that include information about both reef-based and land-based factors can reveal links between human development and coral health through associations among interacting factors, identifying both the scale and direction of anthropogenic effects on coral disease dynamics. Our results indicate that human impacts concentrated along the coast are better predictors of coral health than human interference at watershed scale. In the face of the ordinary obstacles imposed by limited financial and logistical resources for tropical research, which results in small field datasets, we show that patterns and processes often become detectable only if considering causal relationships among multiple explanatory variables. Specifically, we found a positive association between human-altered land cover and GA density. This link, however, was only detected after jointly considering indirect associations between land-based disturbances and water chlorophyll α concentrations. We also found evidence linking TRE with *P. lobata* cover, implying that differences in disease dynamics among sites are partially explained by density-dependent factors driving infections (Aeby *et al.* 2011a,b).

The strongest path in our model explaining GA density showed an effect of human-altered land cover (measured at 1-km radius) via increasing chlorophyll α concentrations. Although spatial variation in water chlorophyll α can be induced by reef circulation (Pinazo *et al.* 2004) or uptake by benthic communities (Genin *et al.* 2009), high levels of chlorophyll α are often associated with anthropogenic nutrient runoffs into reef ecosystems. Increases in nutrient concentrations, in turn, have been linked to increased coral disease risk by depressing coral immune function or increasing pathogen virulence (Harvell *et al.* 1999, Haapkyla *et al.* 2011). For instance, increased nutrient inputs and reduced water quality have been associated with prevalence and severity of black band disease (Kuta & Richardson 2002, Bruno *et al.* 2003, Voss & Richardson 2006) and sea fan aspergillosis (Smith *et al.* 1996, Kim & Harvell 2002, Voss & Richardson 2006, Baker *et al.* 2007). Besides exacerbating coral disease, nutrient inputs can also lead to a number of factors that diminish reef health, including increased macroalgal blooms (Done & Potts 1992, Hughes 1994, Lapointe 1997, McCook *et al.* 2001) and decreased coral growth, reproduction, and photosynthesis (reviewed in Fabricius 2005). While the etiology of GA remains unknown, our results indicate that high chlorophyll α concentrations—a proxy for anthropogenic nutrient input—may lead to increased disease risk, thus providing a potential mechanism for the association between human development along the coast and GA density.

In contrast to our results for GA, we failed to detect a strong link between land- and reef-based variables explaining TRE. We also did not find significant causal associations among explanatory variables, thus the best path model explaining TRE had similar results than a traditional GLM. Our results point to an increased risk of TRE in shallow reefs with high host coral cover. As opposed to GA, we found evidence of decreased TRE prevalence in areas of higher chlorophyll α concentrations. As *P. stenometra* requires multiple hosts to complete its life cycle (Aeby 2007), prevalence of trematodiasis is expected to decrease in impacted reefs if obligatory host species, such as coral-feeding fish, are less abundant due to decreased reef health. A recent study indicated that abundance of Butterfly fish, a potential host, is positively correlated with TRE prevalence (Williams *et al.* 2010). Therefore, our results suggest that higher chlorophyll α concentrations may be indicative of poor reef health, leading to lower abundances of obligatory hosts responsible for higher TRE prevalence. Although human-altered land cover was a weak direct predictor of TRE, it explained GA through changes in chlorophyll α concentrations. Thus, one can argue that the same mechanism affecting GA could be explaining TRE. For this reason, we suggest caution in removing variables with weak direct explanatory power from the analysis, as they may still have potential indirect effects of high ecological relevance. Still, including the indirect effect of human-altered land cover explaining TRE through changes in chlorophyll α only caused decrease in model likelihood. Our findings indicate that TRE is more affected by host density-dependent forces, whereas GA is likely regulated by indirect land-based anthropogenic impacts.

Accelerated development in coastal areas coupled with limited resources for research in tropical regions is creating situations where coral conservation decisions must be based on limited data (Roberts *et al.* 2002). We therefore need data-efficient tools that integrate spatial ecology and epidemiology (Ostfeld *et al.* 2005). Such tools would allow us to better confront the ecological and anthropogenic variability inherent in reef health assessments. Here, we show that path analysis to detect spatial interactions among multiple factors can add significant leverage to disentangle the influence of direct and indirect stressors, increasing our potential to monitor and understand the effect of human development along the coast on coral reefs. Our approach represents a potentially useful tool to develop strategies that curb further degradation and develop prompt and effective mitigation and restoration programs. In terms of coral conservation in Hawai'i, our results indicate that any human activity affecting seawater primary productivity (*i.e.*, land-based pollution, nutrient runoffs) can affect host–pathogen interactions, thus causing detrimental cascading effects on the entire reef ecosystem. Mitigation efforts should focus on monitoring freshwater discharge near developing Hawaiian shorelines.

ACKNOWLEDGMENTS

We thank the Department of Ecology and Evolutionary Biology at Cornell University, Cornell Biogeochemistry and Biocomplexity Initiative, NSF IGERT (DGF0221658) and The Kohala Center, Waimea, Hawai'i, for funding and field support. Drew Harvell, Nelson Hairston, Jed Sparks, and Courtney Couch provided field guidance and reviewed previous versions of this manuscript.

SUPPORTING INFORMATION

Additional Supporting Information may be found in the online version of this article:

FIGURE S1. Spatial distribution of sampling sites throughout the west coast of Hawai'i.

LITERATURE CITED

Aeby, G. S., 1991. Behavioral and ecological relationships of a parasite and its host within a coral reef system. Pac. Sci. 45: 263–269.

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

Aeby, G. S., 2007. Spatial and temporal patterns of Porites trematodiasis on the reefs of Kaneohe Bay, Oahu. Hawaii. Bull. Mar. Sci. 80: 209–218.

Aeby, G. S., G. J. Williams, E. C. Franklin, J. Haapkyla, C. D. Harvell, S. Neale, C. A. Page, L. Raymundo, B. Vargas-Angel, B. L. Willis, T. M. Work and S. K. Davy, 2011a. Growth anomalies on the Coral Genera *Acropora* and *Porites* are strongly associated with host density and human population size across the Indo-Pacific. PLoS ONE 6: e16887.

Aeby, G. S., G. J. Williams, E. C. Franklin, J. Kenyon, E. F. Cox, S. Coles and T. M. Work, 2011b. Patterns of Coral Disease across the Hawaiian Archipelago: relating disease to environment. PLoS ONE 6: e20370.

Baker, D. M., S. E. MacAvoy and K. Kim, 2007. Relationship between water quality, delta N-15, and aspergillosis of Caribbean sea fan corals. Mar. Ecol. Prog. Ser. 343: 123–130.

Bruno, J. F., L. E. Petes, C. D. Harvell and A. Hettinger, 2003. Nutrient enrichment can increase the severity of coral diseases. Ecol. Lett. 6: 1056–1061.

Bruno, J. F., E. R. Selig, K. S. Casey, C. A. Page, B. L. Willis, C. D. Harvell, H. Sweatman and A. M. Melendy, 2007. Thermal stress and coral cover as drivers of coral disease outbreaks. PLoS Biol. 5: 1220–1227.

Carpenter, K. E., M. Abrar, G. Aeby, R. B. Aronson, S. Banks, A. Bruckner, A. Chiriboga, J. Cortes, J. C. Delbeek, L. DeVantier, G. J. Edgar, A. J. Edwards, D. Fenner, H. M. Guzman, B. W. Hoeksema, G. Hodgson, O. Johan, W. Y. Licuanan, S. R. Livingstone, E. R. Lovell, J. A. Moore, D. O. Obura, D. Ochavillo, B. A. Polidoro, W. F. Precht, M. C. Quibilan, C. Reboton, Z. T. Richards, A. D. Rogers, J. Sanciangco, A. Sheppard, C. Sheppard, J. Smith, S. Stuart, E. Turak, J. E. N. Veron, C. Wallace, E. Weil and E. Wood, 2008. One-third of reef-building corals face elevated extinction risk from climate change and local impacts. Science 321: 560–563.

Cheney, D. P., 1975. Hard tissue tumors of scleractinian corals. Adv. Exp. Med. Biol. 64: 77–87.

Cheng, T. C. and A. K. Wong, 1974. Chemical, histochemical, and histopathological studies on corals, *Porites* spp., parasitized by trematode metacercariae. J. Invert. Pathol. 23: 303–317.

CIESIN - Center for International Earth Science Information Network, Columbia University, IFPRI - International Food Policy Research Institute, the World Bank, and CIAT - Centro Internacional de Agricultura Tropical. 2011. Global Rural-Urban Mapping Project, Version 1 (GRUMPv1): population density grid. Socioeconomic Data and Applications Center (SEDAC), Columbia University. Palisades, NY.

Available at http://sedac.ciesin.columbia.edu/data/dataset/grump-v1-population-density (accessed January 2011).

Coyer, J. and J. Witman, 1990. The underwater catalog: a guide to methods in underwater research. Shoals Marine Laboratory, Cornell University press, New York.

Domart-Coulon, I. J., N. Traylor-Knowles, E. Peters, D. Elbert, C. A. Downs, K. Price, J. Stubbs, S. McLaughlin, E. Cox, G. Aeby, P. R. Brown and G. K. Ostrander, 2006. Compressive characterization of skeletal tissue growth anomalies of the finger coral *Porites compressa*. Coral Reefs 25: 531–543.

Done, T. J. and D. C. Potts, 1992. Influences of habitats and natural disturbances on contributions of massive *Porites* corals to reef communities. Mar. Biol. 114: 479–493.

ESRI. 2009. Arc view v.9.3, Richmond, California.

Fabricius, K. E., 2005. Effects of terrestrial runnof on the ecology of corals and coral reefs: review and synthesis. Mar. Poll. Bull. 50: 125–146.

Friedlander, A., G. Aeby, R. Brainard, E. Brown, K. Chaston, A. Clark, P. McGowan, T. Montgomery, W. Walsh, I. Williams and W. Wiltse, 2008. The state of coral reef ecosystems of the main Hawaiian Islands. NOAA Technical Memorandum NOS NCCOS 73: 219–264.

GDSI – Geographic Decision System International, 1995. Watershed unit boundaries for the 8 major Hawaiian islands, generated in Arc/Info and GRID using USGS DEM data. Hawaii Statewide GIS Program, Honolulu, Hawaii.

Genin, A., S. G. Monismith, M. A. Reidenbach, G. Yahel and J. R. Koseff, 2009. Intense benthic grazing of phytoplankton in a coral reef. Limnol. Oceanogr. 54: 938–951.

Gosliner, T., D. Behrens and G. Williams, 1996. Coral reef animals of the Indo-Pacific: animal life from Africa to Hawaii exclusive of the vertebrates. Sea Challengers, California.

Haapkyla, J., R. K. F. Unsworth, M. Flavell, D. G. Bourne, B. Schaffelke and B. L. Willis, 2011. Seasonal rainfall and runoff promote coral disease on an inshore reef. PLoS ONE 6: e16893.

Hamnett, M., M. Lui, and D. Johnson. 2006. Fishing, ocean recreation, and threats to Hawaii's coral reefs, University of Hawaii at Manoa, Hawai'i.

Harvell, C. D., K. Kim, J. M. Burkholder, R. R. Colwell, P. R. Epstein, D. J. Grimes, E. E. Hofmann, E. K. Lipp, A. D. M. E. Osterhaus, R. M. Overstreet, J. W. Porter, G. W. Smith and G. R. Vasta, 1999. Review: marine ecology - Emerging marine diseases - Climate links and anthropogenic factors. Science 285: 1505–1510.

Harvell, C. D., C. E. Mitchell, J. R. Ward, S. Altizer, A. P. Dobson, R. S. Ostfeld and M. D. Samuel, 2002. Ecology – Climate warming and disease risks for terrestrial and marine biota. Science 296: 2158–2162.

Hoegh-Guldberg, O., P. J. Mumby, A. J. Hooten, R. S. Steneck, P. Greenfield, E. Gomez, C. D. Harvell, P. F. Sale, A. J. Edwards, K. Caldeira, N. Knowlton, C. M. Eakin, R. Iglesias-Prieto, N. Muthiga, R. H. Bradbury, A. Dubi and M. E. Hatziolos, 2007. Coral reefs under rapid climate change and ocean acidification. Science 318: 1737–1742.

Hughes, T. P., 1994. Catastrophes, phase-shifts, and large-scale degradation of a Caribbean coral-reef. Science 265: 1547–1551.

Hughes, T. P., A. H. Baird, D. R. Bellwood, M. Card, S. R. Connolly, C. Folke, R. Grosberg, O. Hoegh-Guldberg, J. B. C. Jackson, J. Kleypas, J. M. Lough, P. Marshall, M. Nystrom, S. R. Palumbi, J. M. Pandolfi, B. Rosen and J. Roughgarden, 2003. Climate change, human impacts, and the resilience of coral reefs. Science 301: 929–933.

Jackson, J. B. C., 1997. Reefs since Columbus. Coral Reefs 16: S23–S32.

Jackson, J. B. C., 2001. What was natural in the coastal oceans? PNAS 98: 5411–5418.

Jackson, J. B. C., M. X. Kirby, W. H. Berger, K. A. Bjorndal, L. W. Botsford, B. J. Bourque, R. H. Bradbury, R. Cooke, J. Erlandson, J. A. Estes, T. P. Hughes, S. Kidwell, C. B. Lange, H. S. Lenihan, J. M. Pandolfi, C. H. Peterson, R. S. Steneck, M. J. Tegner and R. R. Warner, 2001. Historical overfishing and the recent collapse of coastal ecosystems. Science 293: 629–638.

Jokiel, P. L., E. K. Brown, A. Friedlander, S. K. Rodgers and W. R. Smith, 2004. Hawai'i coral reef assessment and monitoring program: spatial patterns and termporal dynamics in reef coral communities. Pac. Sci. 58: 159–174.

Kim, K. and C. D. Harvell, 2002. Aspergillosis of sea fan corals: dynamics in the Florida Keys. *In* J. W. Porter and K. G. Porter (Eds.). The Everglades, Florida Bay, and coral reefs of the Florida Keys: an ecosystem sourcebook, pp. 813–824. CRC Press, Florida.

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

Lapointe, B. E., 1997. Nutrient thresholds for bottom-up control of macroalgal blooms on coral reefs in Jamaica and southeast Florida. Limnol. Oceanogr. 42: 1119–1131.

Lapointe, B. E., P. J. Barile, M. M. Littler and D. S. Littler, 2005. Macroalgal blooms on southeast Florida coral reefs II. Cross-shelf discrimination of nitrogen sources indicates widespread assimilation of sewage nitrogen. Harmful Algae 4: 1106–1122.

Maxwell, K. and G. N. Johnson, 2000. Chlorophyll fluorescence-a practical guide. J. Exp. Bot. 51: 659–668.

McClanahan, T., N. Polunin and T. Done, 2002. Resilience in Coral Reefs. *In* L. H. Gunderson and L. Pritchard Jr. (Eds.). Resilience and behavior of large-scale systems, pp. 111–150. Island Press, Washington.

McClanahan, T. R., E. Weil and J. Maina, 2009. Strong relationship between coral bleaching and growth anomalies in massive *Porites*. Global Change Biol. 15: 1804–1816.

McCook, L. J., J. Jompa and G. Diaz-Pulido, 2001. Competition between corals and algae on coral reefs: a review of evidence and mechanisms. Coral Reefs 19: 400–417.

Moberg, F. and C. Folke, 1999. Ecological goods and services of coral reef ecosystems. Ecol. Econ. 29: 215–233.

NOAA – National Oceanic and Atmospheric Administration. 2011. C-CAP Coastal Change Analysis Program - Hawaii Land Cover Raster Layer at 2 m resolution.

Orr, J. C., V. J. Fabry, O. Aumont, L. Bopp, S. C. Doney, R. A. Feely, A. Gnanadesikan, N. Gruber, A. Ishida, F. Joos, R. M. Key, K. Lindsay, E. Maier-Reimer, R. Matear, P. Monfray, A. Mouchet, R. G. Najjar, G. K. Plattner, K. B. Rodgers, C. L. Sabine, J. L. Sarmiento, R. Schlitzer, R. D. Slater, I. J. Totterdell, M. F. Weirig, Y. Yamanaka and A. Yool, 2005. Anthropogenic ocean acidification over the twenty-first century and its impact on calcifying organisms. Nature 437: 681–686.

Ostfeld, R. S., G. E. Glass and F. Keesing, 2005. Spatial epidemiology: an emerging (or re-emerging) discipline. Trends Ecol. Evolut. 20: 328–36.

Pinazo, C., S. Bujan, P. Douillet, R. Fichez, C. Grenz and A. Maurin, 2004. Impact of wind and freshwater inputs on phytoplankton biomass in the coral reef lagoon of New Caledonia during the summer cyclonic period: a coupled three-dimensional biogeochemical modeling approach. Coral Reefs 23: 281–296.

Roberts, C. M., C. J. McClean, J. E. N. Veron, J. P. Hawkins, G. R. Allen, D. E. McAllister, C. G. Mittermeier, F. W. Schueler, M. Spalding, F. Wells, C. Vynne, and T. B. Werner. 2002. Marine biodiversity hotspots and conservation priorities for tropical reefs. Science 128: 0–1284.

Rodgers, K. S. and E. F. Cox, 2003. The effects of trampling on Hawaiian corals along a gradient of human use. Biol. Conserv. 112: 383–389.

Rogers, C. S., 1990. Responses of coral reefs and reef organisms to sedimentation. Mar. Ecol. Prog. Ser. 62: 185–202.

Shipley, B., 2002. A user's guide to path analysis, structural equations and causal inference. Cambridge University Press, Cambridge.

Smith, G. W., L. D. Ives, I. A. Nagelkerken and K. B. Ritchie, 1996. Caribbean sea-fan mortalities. Nature 383: 487–487.

Stimson, J., 2011. Ecological characterization of coral growth anomalies on *Porites compressa* in Hawai'i. Coral Reefs 30: 133–142.

Systat. 2002. Systat for windows v. 10.1, San Jose, California.

U.S. Census Bureau. 2010. Statewide population growth rates; Hawai`i State Department of Business, Economic Development & Tourism, Suitland, MD, USA.

Voss, J. D. and L. L. Richardson, 2006. Nutrient enrichment enhances black band disease progression in corals. Coral Reefs 25: 569–576.

Weis, V. M. and D. Allemand, 2009. What determines coral health? Science 324: 1153–1155.

Williams, G. J., G. S. Aeby, R. O. M. Cowie and S. K. Davy, 2010. Predictive modeling of coral disease distribution within a reef system. PLoS ONE 5: e9264.

Wolanski, E., R. Richmond, L. McCook and H. Sweetman, 2003. Mud, marine snow and coral reefs – The survival of coral reefs requires integrated watershed-based management activities and marine conservation. Amer. Sci. 91: 44–51.

Work, T. M., G. S. Aeby and S. L. Coles, 2008. Distribution and morphology of growth anomalies in *Acropora* from the Indo-Pacific. Dis. Aquat. Org. 78: 255–264.