Biological Conservation 178 (2014) 88–96

Contents lists available at ScienceDirect

Biological Conservation

journal homepage: www.elsevier.com/locate/biocon

Scuba diving damage and intensity of tourist activities increases coral disease prevalence

Joleah B. Lamb [a,b,c,*], James D. True [d], Srisakul Piromvaragorn [c,d], Bette L. Willis [a,b,c]

[a] School of Marine and Tropical Biology, James Cook University, Townsville, Queensland 4811, Australia
[b] Australian Institute of Marine Science & James Cook University (AIMS@JCU), Townsville, Queensland 4811, Australia
[c] Australian Research Council (ARC) Centre of Excellence for Coral Reef Studies, Townsville, Queensland 4811, Australia
[d] Center for Biodiversity in Peninsular Thailand, Faculty of Science, Prince of Songkla University, Hat Yai, Songkla 90112, Thailand

A R T I C L E I N F O

Article history:
Received 25 March 2014
Received in revised form 27 June 2014
Accepted 29 June 2014

Keywords:
Ciliates
Coral disease
Damage
Diving
Koh Tao
Scuba
Skeletal eroding band
Sedimentation
Snorkelling
Thailand
Tourism
White syndrome
Wounds

A B S T R A C T

Recreational diving and snorkeling on coral reefs is one of the fastest growing tourism sectors globally. Damage associated with intensive recreational tourist use has been documented extensively on coral reefs, however other impacts on coral health are unknown. Here, we compare the prevalence of 4 coral diseases and 8 other indicators of compromised coral health at high and low use dive sites around the island of Koh Tao, Thailand. Surveys of 10,499 corals reveal that the mean prevalence of healthy corals at low use sites (79%) was twice that at high use sites (45%). We also found a 3-fold increase in coral disease prevalence at high use sites, as well as significant increases in sponge overgrowth, physical injury, tissue necrosis from sediment, and non-normally pigmented coral tissues. Injured corals were more susceptible to skeletal eroding band disease only at high use sites, suggesting that additional stressors associated with use intensity facilitate disease development. Sediment necrosis of coral tissues was strongly associated with the prevalence of white syndromes, a devastating group of diseases, across all sites. We did not find significant differences in mean levels of coral growth anomalies or black band disease between high and low use sites. Our results suggest that several indicators of coral health increase understanding of impacts associated with rapid tourism development. Identifying practical management strategies, such as spatial management of multiple reef-based activities, is necessary to balance growth of tourism and maintenance of coral reefs.

1. Introduction

Global decline in coral reef health is a critical conservation concern, especially for the estimated 275 million people that live within 30 km of coral reefs and draw extensively on them for livelihood and food security (Bellwood et al., 2004; Burke et al., 2011). There is pressing demand to find income-generating alternatives to destructive and extractive uses of marine resources (Birkeland, 1997). Tourism is generally considered a favorable alternative, typically providing an incentive to preserve natural areas, thereby contributing to environmental protection, sustainable use practices, and the restoration of biological diversity (Buckley, 2012). Coral reef-based tourism is one of the fastest growing tourism sectors worldwide (Ong and Musa, 2011). However, because the majority of coral reefs are located in developing and often undermanaged island and coastal regions (Donner and Portere, 2007), the unrestricted growth and rapid development of reef-based tourism often undermines the conservation priorities necessary to sustain it.

Coral disease outbreaks are now recognized as a significant factor in the accelerating degradation of coral reefs, and it is commonly assumed that a variety of human-related activities have altered environmental conditions, potentially impairing coral resistance to microbial infections or increasing pathogen virulence (Altizer et al., 2013). Anthropogenic activities implicated in disease outbreaks and rising prevalence levels (i.e., the number of cases of a disease in a given population at a specific time) include proximity to human population centers (Aeby et al., 2011a), coastal land alteration and dredging (Guilherme Becker et al., 2013; Pollock et al., 2014), terrestrial runoff of sediment or agricultural herbicides (Owen et al., 2002; Haapkylä et al., 2011), sewage outfalls containing human enteric microorganisms (Patterson et al., 2002), increases in nutrient concentrations (Bruno et al., 2003), aquaculture and fish farms (Harvell et al., 1999; Garren et al., 2009), a reduction in the diversity of reef fish assemblages (Raymundo et al., 2009), and sunscreens (Danovaro et al., 2008).

* Corresponding author. Present Address: Cornell University, Department of Ecology and Evolutionary Biology, Ithaca, NY 14850, USA. Tel.: +1 607 216 5021.
E-mail address: joleah.lamb@jcu.edu.au (J.B. Lamb).

http://dx.doi.org/10.1016/j.biocon.2014.06.027
0006-3207/

Until recently, recreational reef-based activities, such as diving and snorkeling, were thought to have little direct impact on coral assemblages. However, over the past two decades, numerous studies have been conducted on the physical impacts and management of diving on coral reefs worldwide. Most concluded that diving could adversely affect coral assemblages through physical injury (e.g., Hawkins and Roberts, 1992, 1993; Davis and Tisdell, 1995; Hawkins et al., 1999, 2005) or sediment deposition (Zakai and Chadwick-Furman, 2002). In a few studies, coral disease has been associated with the presence of concentrated tourist activities (Hawkins et al., 1999; Winkler et al., 2004; Lamb and Willis, 2011), however no studies have attempted to directly link coral susceptibility or disease prevalence with measures of dive site use intensity, such as levels of physical injury or sediment deposition. Minor damage and resuspension of sediment by most divers may seem trivial, but by compounding other reef stresses associated with tourism, they could undermine the resilience of local reef ecosystems (Nyström et al., 2000) and reduce recovery rates following natural disturbances (Connell, 1997). In addition, a variety of other factors could increase coral disease prevalence and reduce health at intensively dived tourist sites in rapidly developing regions, including possible increases in nutrients from vessel sewage and wastewater and elevated levels of resuspended sediment associated with shoreline erosion from boat wakes and crowding.

The island of Koh Tao, located in the western Gulf of Thailand, has rapidly grown as a tourist and recreational destination, leading to the replacement of small-scale hook-and-line or traditional hand net fisheries by reef-related tourist activities (Yeemin et al., 2006). From 1992 to 2003, the number of tourists increased by 375% and now considered the hub of scuba diving certification in Southeast Asia, estimated to generate US$62 million per year to the local economy (Larpnun et al., 2011). At present, the island has approximately 50 dive operators that accommodate greater than 300,000 visitors per year to a total reef area of 2 km^2 (Weterings, 2011; Larpnun et al., 2011), reaching intensities of use beyond levels seen even in regions heavily impacted by damage, such as the Red Sea (<250,000 divers/year to 4 km^2 of reef area: Zakai and Chadwick-Furman, 2002).

Here, we use the prevalence of four coral diseases and eight additional indicators of compromised coral health to assess the effects of recreational diving intensity on coral reefs surrounding Koh Tao. To date, the concurrent use of multiple field-based signs of disease and other indicators of compromised health to classify stress associated with human activities on reef corals has not been undertaken. Using a multitude of indicators to assess coral health may, for the first time, improve our capacity to identify more specific sources of impacts from tourism on reef corals. In light of predicted increases in tourism and recreational activities globally, the results of this study will aid in the development of practical management strategies to mitigate the impacts of frequent visitation that increase the likelihood of coral disease outbreaks and ensure long-term persistence of corals reefs and livelihoods in developing coastal regions.

2. Methods

2.1. Data collection

We conducted surveys around the island of Koh Tao in September 2011, approximately 1 year following a bleaching event and subsequent wet season in the Gulf of Thailand (Fig. 1 and Supplementary Material). We selected a total of 10, 90 m^2 sites distributed around the island and located approximately 100 m from shore. Based on questionnaires from 23 of the largest dive operators on the island, Weterings (2011) found that most of the dive sites around Koh Tao were unevenly visited and a select number were often frequented by up to 10 dive operators in a single day. Due to ease of access, dive sites with the highest levels of use are often located nearest to the large number of operators located in the west and southwest regions of the island (Fig. 1). We surveyed the top 5 dive sites that are heavily and constantly used by visitors throughout the year (i.e., more than 5 boat operators with a minimum of 50 in-water visitors/site/day) (high use sites), and 5 sites that had similar coral assemblages but had few to no in-water visitors each year (low use sites).

At each site, three 15 m $\times$ 2 m belt transects were laid randomly along reef contours at 3–6 m in depth and approximately 5 m apart, consistent with standardized protocols developed by the Global Environment Facility (GEF) and World Bank Coral Disease Working Group (Beeden et al., 2008), which allow the data from this study to be directly compared to other coral disease datasets collected globally. Specifically, within each 30 m^2 belt transect (90 m^2 per dive site), every scleractinian coral over 5 cm in diameter was identified to genus and further classified as either diseased (i.e., affected by one or more of the following disease classes recorded in the Indo-Pacific region (see Fig. 1): white syndromes, skeletal eroding band, black band disease, brown band disease, and/or growth anomalies); showing other signs of compromised health (i.e., affected by one or more of the following: tissue necrosis due to sediment, bleaching, non-normal pigmentation of tissue, overgrowth by sponges, red or green algae, and cuts and scars from predation by crown-of-thorns starfish and corallivorous marine snails); physically damaged (recently exposed skeleton from breakage or severe abrasions); or healthy (i.e., no visible signs of disease lesions, other compromised health indicators or physical damage) (Willis et al., 2004; Lamb and Willis, 2011). Standard line-intercept surveys were used to determine coral cover and community composition by estimating the linear extent of each coral to the nearest centimeter along the central line of each 15 m transect.

2.2. Data analyses

The prevalence of coral disease and other signs of compromised health was calculated within each 30 m^2 belt transect by dividing the number of colonies with one of the four diseases or eight other compromised health categories recorded in this study by the total number of colonies present, i.e., 15 prevalence values per disease or category, both for the group of high use and low use sites.

Differences in overall disease assemblages were investigated using multivariate community analyses. A nested permutational multivariate analysis of variance (Anderson et al., 2008) was used to test for differences between high and low use levels, with site (random factor) nested within use-level (fixed factor). The analysis was based on a zero-adjusted Bray–Curtis similarity matrix (Clarke and Gorley, 2006), type III partial sums of squares, and 999 random permutations of the residuals under the reduced model. To identify indicators of disease and other signs of compromised coral health between the two use-levels (those contributing most to the patterns in multivariate space), we used a principal coordinates analysis (PCO) performed on a Bray–Curtis similarity matrix using square root transformed data due to strong linear pairs of variables (Clarke and Gorley, 2006; Anderson et al., 2008). We calculated Pearson correlations of the ordination axes with the original disease and other compromised health data, where indicators with strong correlations (defined in this study as $\geqslant 0.6$) were then overlaid as vectors on a bi-plot.

Similarities between coral communities at the family-level were illustrated using a non-metric multidimensional scaling plot (nMDS), with hierarchical clusters overlaid from dendrograms based on a Bray–Curtis similarity matrix from square-root transformed data at the transect level (Clarke and Gorley, 2006). We used a nested analysis of similarity (ANOSIM) to test differences in coral assemblages between use-levels, where we nested

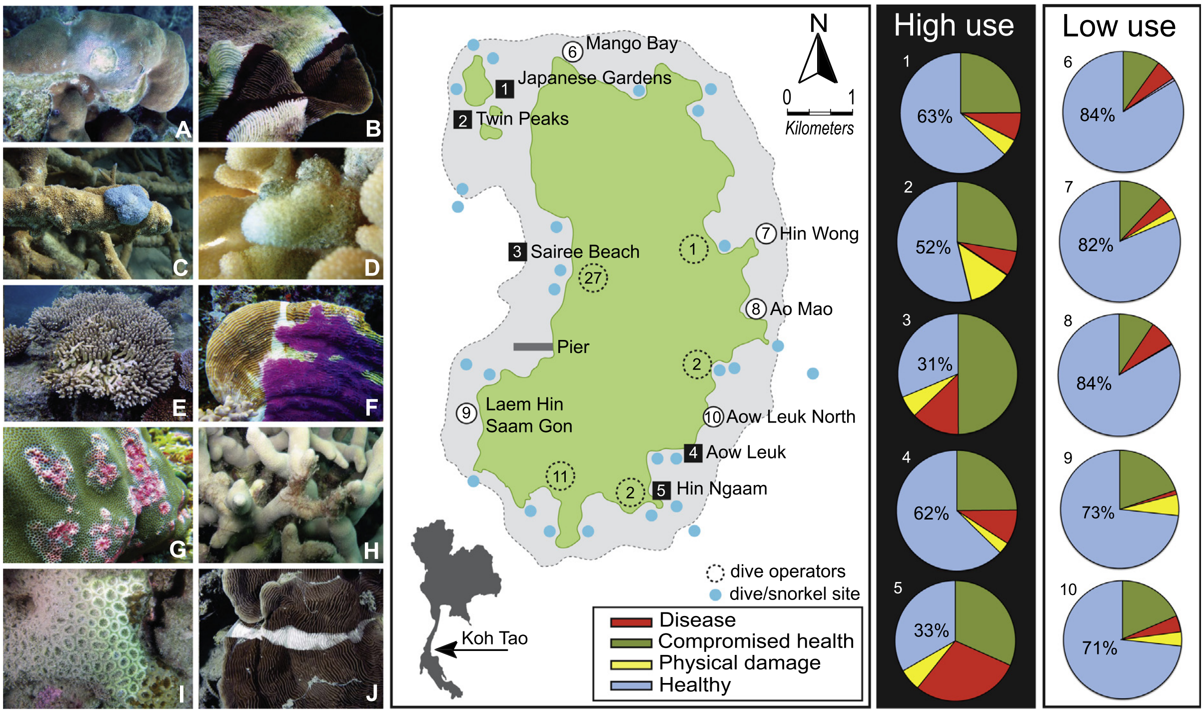

Fig. 1. Locations of survey sites with high (solid squares, numbered 1–5) and low visitor use (open circles, numbered 6–10) around the island of Koh Tao, Thailand. Individual pie charts represent the mean proportion of coral colonies at each site classified within 4 health status categories[a]: disease (including (A) white syndromes, (B) black band disease, (C) growth anomalies, and (D) skeletal eroding band disease); (E) physical damage (recently exposed skeleton); other compromised health indicators (including (F) sponge overgrowth, (G) non-normally pigmented tissue responses, (H) algae overgrowth, (I) sediment damage, and (J) bleaching; or healthy. Category means were calculated from 3 transects per site. Percentages indicated within each pie graph represent healthy colonies. [a]Standardised signs of disease and compromised coral health as per Beeden et al. (2008), an output of the Global Environment Facility and World Bank Coral Disease Working Group.

site (random factor) into use-level (fixed factor). Multivariate analyses were performed in PRIMER v6 and PERMANOVA+ add-in (PRIMER-E Ltd., Plymouth, UK).

To analyze patterns of coral disease among broad taxonomic groups (e.g., Veron, 2000), coral families were assigned to 1 of 3 disease susceptibility categories on the basis of previous studies of coral disease prevalence in the Indo-Pacific region (Willis et al., 2004; Kaczmarsky, 2006; Aeby et al., 2011a,b; Lamb and Willis, 2011; Ruiz-Moreno et al., 2012): the highly disease susceptible and abundant family, Acroporidae; the disease susceptible families Pocilloporidae and Poritidae; and the disease resistant families Agariciidae, Faviidae, Fungiidae, Merulinidae, and Mussidae. Differences in mean prevalence of disease, other signs of compromised health and physical damage among high and low use sites were compared using a 2-way nested analysis of variance (ANOVA), where site (random factor) was nested within use-level (fixed factor). We tested associations between continuous variables with Pearson product moment correlations (PPMC) with the confidence interval set at 0.95. Coral disease susceptibility as a result of visually assessed compromised health was evaluated using a Pearson's chi-square test. Prior to all univariate analyses, assumptions of normality (Shapiro–Wilks) and homogeneity of variance (Levene's test of homogeneity) were tested and data were transformed to meet assumptions of normality where necessary. Univariate statistics were performed in R v3.0.2 (R Core Team, 2013).

3. Results

3.1. Effects of intensive use on coral health and disease

Assemblages of disease and other compromised health signs differed significantly between use-level (*pseudo*-$F_{1,8} = 3.63$, $P = 0.008$), with the prevalence of visually healthy corals contributing most strongly to driving this separation (39.5% of variation on the PCO1 axis: Fig. 2 and Table S1). The mean prevalence of healthy corals recorded at high use sites was 45.2% ± 6.2 SE (range = 31–63%, $n = 5983$ corals surveyed), approximately half the mean percentage of healthy corals recorded at low use sites (78.8% ± 2.5 SE, range = 71–84%, $n = 4516$ corals surveyed).

3.1.1. Disease prevalence

Mean overall coral disease prevalence was approximately 3-fold greater at sites with high visitation (mean ± SE = 14.5% ± 4.0; 727 cases of disease) compared to low use sites (5.2% ± 1.3; 197 cases of disease; Table 1). At low use sites, disease prevalence ranged between 1.2% and 8.5% (median = 5.6%), whereas it ranged between 6.9% and 29.9% (median = 11.4%) at high use sites. Both the maximum prevalence and maximum number of cases of each of the four diseases were recorded at high use sites (Table 1). No cases of brown band disease, a common ciliate disease in the Indo-Pacific (Willis et al., 2004), were recorded during these surveys. The two most prevalent diseases, skeletal eroding band (SEB) and white syndromes (WS), were 2-fold and 4-fold greater, respectively, at high use sites than at low use sites (Table 1 and Fig. 3a). Mean black band disease (BBD) prevalence was low at all sites, however it was 9-times greater at high use sites than low use sites, although it did not differ significantly between use levels (Table 1 and Fig. 3a). There was no difference in the mean prevalence of growth anomalies between the two use levels (Table 1 and Fig. 3a).

3.1.2. Prevalence of other signs of compromised health and physical damage

When combined, overall mean prevalence of the 8 other compromised health categories was approximately 2 times greater at high use sites (mean ± SE = 32.3% ± 9.4; 1897 corals with other

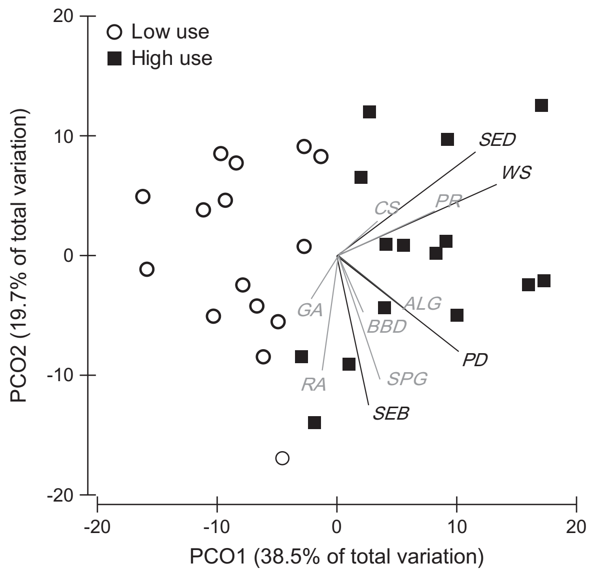

Fig. 2. Principal coordinates analysis (PCO) of coral health and disease variables. Spatial variation in 4 coral disease and 8 other compromised coral health indicators at the transect level, for high use (solid squares) and low use sites (open circles) for the first two principal components. Analysis performed on a Bray–Curtis similarity matrix using square root-transformed data, with vectors depicting original variables and Pearson correlation values (gray vectors ⩾ 0.2, black vectors ⩾ 0.6) representing relative contributions of disease or other compromised coral health signs on the observed variation in use-level. Coral diseases: SEB = skeletal eroding band, WS = white syndromes, BBD = black band disease, GA = growth anomalies; other compromised coral health indicators: PD = physical damage, SED = sediment necrosis, SPG = sponge overgrowth, ALG = algal overgrowth, PR = pigmentation response, RA = red algal overgrowth, CS = cuts and scars from predation, and BL = bleaching.

signs of compromised health) compared to low use sites (15.0% ± 4.1; 752 cases; Table 1). Four of these compromised health categories were significantly more prevalent at high use sites (Table 1 and Fig. 3b). Specifically, there was a 12-fold increase in corals with tissue necrosis from sediment and a 9-fold increase in corals with exposed skeleton (physical damage) at sites with high use (Table 1). In addition, approximately 3 times as many corals at high use sites had non-normally pigmented tissue (pigmentation responses) or were actively overgrown by sponges (Table 1 and Fig. 3b). There was no significant difference in the prevalence of bleaching, algal overgrowth or cuts and scars associated with predation between the two use levels (Table 1 and Fig. 3b).

Table 1
Number of cases of coral disease and other signs of compromised health at sites with low levels of recreational use ($n = 15$ transects, 4516 colonies surveyed) and high levels of recreational use ($n = 15$ transects, 5983 colonies surveyed), and results of the main effects nested analysis of variance of mean prevalence (%) between use-level groups. Mean prevalence for each variable (±SE) shown in Fig. 3. Analyses performed on data transformed to the square root.

Variable	Number of cases		Main effect	
	Low use	High use	*F*	*P*
Total disease	197	727	23.4	<0.001*
Skeletal eroding band	153	464	10.9	<0.004*
White syndromes	32	185	9.0	<0.007*
Black band	4	51	0.8	<0.37
Growth anomalies	8	27	<0.2	<0.73
Total other compromised health	752	1927	35.3	<0.001*
Algal overgrowth	324	416	1.3	<0.27
Pigmentation response	107	337	5.5	<0.03*
Physical damage	31	339	22.3	<0.001*
Sediment necrosis	13	192	16.6	<0.001*
Red algal overgrowth	133	265	0.7	<0.41
Sponge overgrowth	46	216	19.9	<0.001*
Predation scars	84	136	3.6	<0.09
Bleaching	14	26	<0.1	<0.84

* Indicate significant differences set at $\alpha = 0.05$.

3.1.3. Patterns and susceptibility of diseases and other signs of compromised coral health

Sites with a high prevalence of corals showing other signs of compromised health also had high levels of disease ($r_{28} = 0.54$; $P < 0.005$; Fig. 4a). Patterns in the assemblages of diseases and other compromised health indicators differed among sites within use-level (*pseudo*-$F_{8,20} = 4.073$, $P < 0.01$), although differences (19.7% of variation on the PCO2 axis: Fig. 2 and Table S1) were largely driven by the prevalence of skeletal eroding band and physical damage, which were strongly correlated across all sites ($r_{28} = 0.78$, $P < 0.001$; Fig. 4b), and the prevalence of white syndrome and sediment necrosis, which were also strongly correlated across all sites ($r_{28} = 0.67$, $P < 0.001$; Fig. 4c). Coral colonies with recently exposed skeleton were more likely to also have skeletal eroding band disease (22%) than colonies without recent physical damage (6%) at high use sites ($\chi^2_1 = 136.1$, $P < 0.001$), but at low use sites, recent physical damage did not affect the susceptibility of corals to SEB (7% compared to 5%: $\chi^2_1 = 0.45$, $P = 0.51$). Colonies with tissue necrosis associated with sediment were also more likely to have white syndrome lesions than colonies without sediment damage at both high (26% compared to 3%: $\chi^2_1 = 256.0$, $P < 0.001$) and low use sites (31% compared to 4%: $\chi^2_1 = 28.2$, $P < 0.001$).

3.2. Host density, cover and composition as potential drivers of disease

Mean coral density (number of colonies per m^2) did not vary significantly between low (mean ± SE = $10.0/m^2 \pm 1.1$) and high use sites ($13.3/m^2 \pm 1.9$; $F_{1,8} = 0.8$, $P = 0.40$). Moreover, the number of disease cases (Table 1) was not associated with coral density at sites with low ($r_{13} = 0.30$, $P = 0.28$) or high recreational use ($r_{13} = 0.13$, $P = 0.63$).

The composition of coral assemblages was at least 60% similar among all transects surveyed in this study (Bray–Curtis similarity), and did not differ significantly between high and low use sites (*Global R* = 0.11, $P = 0.18$; Fig. 5a). On average, corals in the disease resistant families (Agariciidae, Faviidae, Fungiidae, Merulinidae, and Mussidae) accounted for the largest percentage of coral cover at both low and high use sites ($F_{1,8} = 0.5$, $P = 0.51$; Fig. 5b), and benthic cover of disease resistant coral families was not associated with coral disease prevalence at low ($r_{13} = 0.32$, $P = 0.24$) or high use sites ($r_{13} = 0.04$, $P = 0.89$; Fig. 5f). Percent cover of Acroporidae and the disease susceptible families, Pocilloporidae and Poritidae, was marginally greater at high use sites ($F_{1,8} = 0.7$, $P = 0.41$ and $F_{1,8} = 1.7$, $P = 0.22$, respectively; Fig. 5b), but disease prevalence was not correlated with cover of these groups (low: $r_{13} = 0.12$, $P = 0.68$ and $r_{13} = 0.11$, $P = 0.69$; high: $r_{13} = 0.09$, $P = 0.76$ and $r_{13} = 0.14$, $P = 0.62$; Fig. 5d and e). Total percent cover of all coral families combined did not influence disease prevalence at high use sites ($r_{13} = 0.45$, $P = 0.10$), however there was a significant positive correlation between total disease prevalence and total coral cover at low use sites ($r_{13} = 0.54$, $P = 0.04$; Fig. 5c).

4. Discussion

This study reveals that intensive site use associated with reef-based tourist activities significantly reduces the overall health of corals, undermining the value of the resource necessary for sustaining the growing nature-based tourism industry. Consistency in the pattern of substantially elevated levels of disease at high use sites highlights the urgent need to identify and mitigate potential

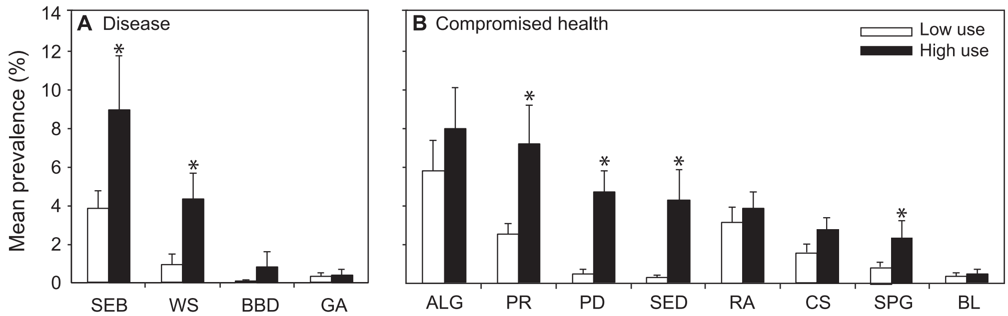

Fig. 3. Effect of use-level on coral disease and other compromised health indicators. Prevalence (mean ± SE) of (A) coral disease (SEB = skeletal eroding band, WS = white syndromes, BBD = black band disease, GA = growth anomalies) and (B) other compromised coral health signs (ALG = algal overgrowth, PR = pigmentation response, PD = physical damage, SED = sediment necrosis, RA = red algal overgrowth, CS = cuts and scars from predation, SPG = sponge overgrowth, and BL = bleaching) at low use sites (open bars, 4516 colonies surveyed) and high use visitor sites (solid bars; 5983 colonies surveyed). Analyses performed on data transformed to the square root and asterisks indicate significant differences set at $\alpha = 0.05$ for each individual indicator.

causes of increased disease prevalence at these sites, particularly as additional impacts are anticipated with accelerated development of infrastructure along coastal regions to support tourism growth.

Differences in coral cover, density or family composition are unlikely to have caused the striking differences in disease prevalence among sites, given that percent cover of all corals and of disease-susceptible families did not differ among high and low use sites. Similarities in coral cover, density and composition among sites that clearly differed in a range of coral health indicators contribute to the emerging consensus that percent cover of live coral is of limited value as an indicator of ecosystem health, as it typically failed to separate areas affected by human activities from those less affected (Muthiga and McClanahan, 1997; Hawkins et al., 1999; Dinsdale and Harriott, 2004). We conclude that percent cover is not appropriate as the sole indicator of impacts when assessing reef-based activities, but is useful when used in conjunction with other indicators. We note, however, that in the group of low use sites, disease prevalence was positively correlated with total cover, potentially reflecting transmission of pathogens via direct colony-to-colony contact (Riegl, 2002). At high use sites, it is more likely that increased susceptibility to infection associated with localized environmental stressors led to higher prevalence of coral disease.

4.1. New approaches to identifying and managing stressors affecting coral health

Linking indicators of stress with potential causes, so that action can be initiated before irreversible declines in health occur, has been challenging for corals. Bleaching is one of the few readily identifiable signs of coral stress, but bleaching has been associated with a wide range of stressors, like changes in water temperature and light (Brown, 1997), ocean acidification (Hoegh-Guldberg et al., 2007), bacterial infections (Kushmaro et al., 1997), herbicides (Jones et al., 2003), and sunscreen (Danovaro et al., 2008). Our study of coral health impacts associated with the intensity of diving-related activities provides valuable insights for linking a range of compromised health indicators with potential stressors, and highlights the need for multiple metrics of coral health and disease to deduce sources of stress on coral reefs and aid in developing practical management strategies for mitigating them. Moreover, while multiple metrics of coral health increase capacity to differentiate between human impacts and other drivers of disease in this study, monitoring the prevalence of healthy corals can be readily implemented into existing coral survey programs with little to no additional training, thereby providing more comprehensive and meaningful reef health assessments. Inclusion of this basic metric in monitoring programs would also contribute to much-needed baseline data (e.g., Willis et al., 2004) to enable future detection of changes in the health of reef corals and the success of management intervention.

4.1.1. Sediment stress

The 12-fold greater prevalence of sediment-associated tissue necrosis at high use sites represented one of the greatest differences in coral health indicators between sites exposed to high versus low intensity recreational diving. Recreational divers significantly increased turbidity and resuspended sediment at popular dive sites in the Red Sea, each causing approximately nine sediment clouds to settle back onto corals per dive (Zakai and Chadwick-Furman, 2002). In addition, wakes generated by boat traffic can redistribute and increase turbidity from sediment resuspension and shoreline erosion, with turbidity taking between 4 and 24 h to return to background levels following disturbance (Yousef et al., 1980; Jones, 2011). Although corals possess mechanisms to actively remove sediment particles, such mechanisms are energetically costly (Hubbard and Pocock, 1972; Rogers, 1990; Philipp and Fabricius, 2003), thus corals at intensively used sites suffer depleted energy budgets from even low levels of chronic sediment deposition (Rogers, 1990; Philipp and Fabricius, 2003), leading to localized bleaching and tissue necrosis.

The high correlation found between the prevalence of sediment-associated tissue necrosis and the prevalence of white syndromes, regardless of site use intensity, signifies that localized direct contact with sediment may be a primary factor contributing to this disease. Sediment could act as both a disease reservoir and potentially a vector when resuspended as a result of tourist-related activities, and could also increase the likelihood of infection by stressing coral hosts (Lafferty and Holt, 2003; Pollock et al., 2014). On hurricane-damaged reefs, Brandt et al. (2013) reported that another tissue loss disease, white plague, occurred primarily on fragments in direct contact with sediment, and hypothesized a link with bacterial overgrowth. Evidence that sediment damage to corals is reduced following treatment with antibiotics (Hodgson, 1990), and that growth rates of coral-associated microbes increased 10-fold and led to rapid tissue loss following exposure to elevated levels of carbon (Kline et al., 2006), further support this link. Whether sediment accumulation causes coral disease by introducing pathogens or is a general sign of coral stress to other environmental stressors warrants further study. Practical and readily-introduced solutions for reducing sedimentation include limiting boat traffic and site crowding, and the establishment of no-wake zones and speed limits when traveling within close proximity to reefs.

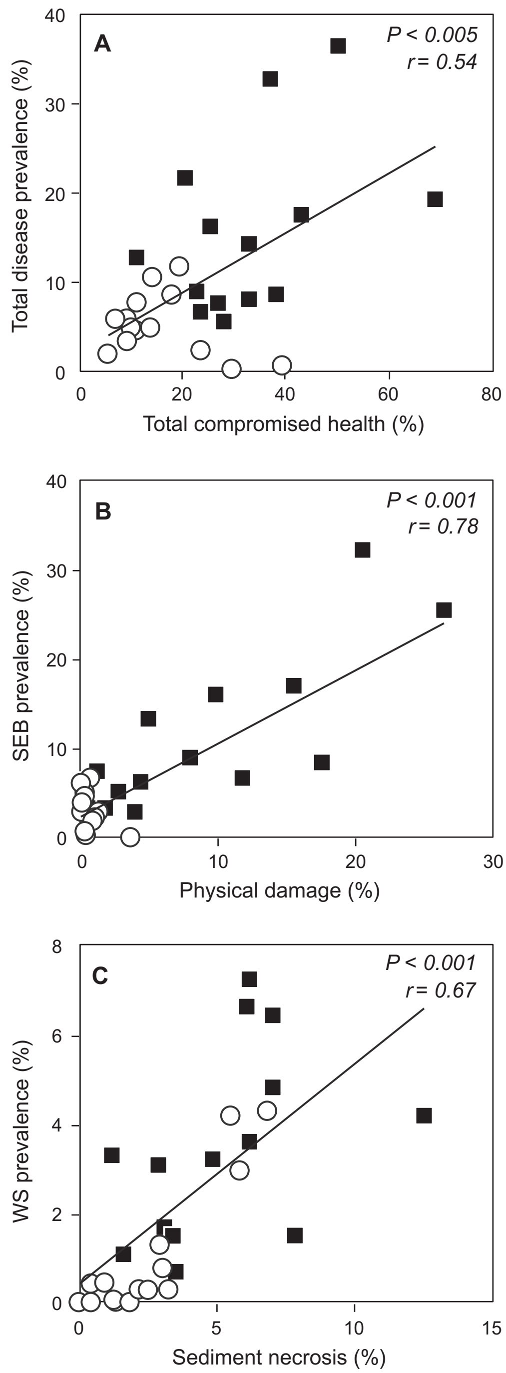

Fig. 4. Associations between the prevalence of (A) total coral disease and other signs of compromised coral health, (B) recent physical damage and skeletal eroding band (SEB) disease, and (C) tissue necrosis due to sediment and white syndromes (WS). Open circles indicate low use sites and black squares indicate high use sites in each panel. Pearson product–moment correlations conducted on transects pooled from low and high use sites.

Although overall levels of black band disease (BBD) were low and not significantly different between high and low use sites, the 9-fold increase of BBD at high use sites further corroborative evidence that sediment accumulation plays a key role in diving-related disturbances. The biogeochemical microenvironment beneath BBD microbial mats, which represent complex and diverse polymicrobial consortia (Sutherland et al., 2004; Kaczmarsky, 2006; Sato et al., 2009), is characterized by anoxia, high sulfide concentrations and low pH, conditions that are lethal to underlying coral tissues (Glas et al., 2012). These toxic conditions are most pronounced under low light conditions (Glas et al., 2012), therefore sediment accumulation on coral surfaces could provide an anaerobic microenvironment conducive to microbial mat formation, while increased turbidity (and associated decreased light levels) could facilitate the rapid establishment of conditions characteristic of the disease.

4.1.2. Nutrient enrichment

The marked increase in sponge overgrowth at high use sites further suggests that nutrient enrichment is a significant issue associated with intensive tourist use. Increased primary production associated with nutrient enrichment and sediment favors benthic filter-feeding organisms, particularly sponges, which then typically outcompete corals (Pastorok and Bilyard, 1985). On Grand Cayman, a 5-fold increase in the biomass of *Cliona delitrix*, *a sponge* overgrowing the coral *Montastrea cavernosa*, and a 6-fold increase in bacterial biomass was recorded on fringing reefs exposed to discharges of untreated fecal sewage compared to a control site 1 km away (Rose and Risk, 1985). In other studies, widespread overgrowth of corals by the cyanobacteriosponge *Terpios hoshinota* on Japanese reefs was particularly noteworthy in pollution-stressed zones (Rützler and Muzik, 1993), and bacteria similar to those detected in black band disease were detected on sponge-covered but not on sponge-free corals (Tang et al., 2011), suggesting that *T. hoshinota* might benefit from the presence of bacteria associated with unhealthy corals.

Inputs of nutrients, pathogens, and other wastewater-derived pollution have also been linked to several other coral diseases (Bruno et al., 2003). For example, sewage outfalls containing the human gut microbe *Serratia marscens* have been associated with a type of white syndrome infecting and decimating acroporid corals off the coast of Florida (Patterson et al., 2002). Nutrient enrichment from sewage and wastewater pollution is one of the few stressors that, with proper research, policy, and management, can be effectively mitigated. Fecal indicator bacteria, such as *Enterococcus*, *can be monitored* (Gronewold et al., 2008) or alternatively, stable isotope analysis can detect the presence of sewage-derived nitrogen within an ecosystem. In Mexico, δ^{15}N values of the common sea fan were more variable near a developed tourist site than at an undeveloped site, with 84% of the observed variation explained by tourist visitations in the preceding year (Baker et al., 2013). Due to the unregulated and rapid expansion of dive tourism in many developing countries, most tourist vessels are not equipped with proper storage systems for wastewater and sewage. Tertiary treatment systems on fitted to vessels can remove up to 90% of nutrients (Judd, 2010). Because pollutants cannot be isolated in open marine systems and may have implications beyond local coral assemblages (McCallum et al., 2004), the possibility of disease dispersing from sites with higher levels of environmental stress is concerning. It is also possible that land-based pollutants are elevated on the western side of the island near terrestrial tourism infrastructure and the main shipping pier, however our paired high and low use site in this location further indicates that reef-based tourism intensity can still cause significant disparities in disease levels adjacent to developed coastal areas.

4.1.3. A general indicator of stress

Non-normal pigmentation of coral tissue, or pigmentation response, has been characterized as a general immune response to a physical or pathogenic challenge (Willis et al., 2004; Bongiorni and Rinkevich, 2005; Palmer et al., 2008). Pigmented tissues possess high levels of melanin, an important component of invertebrate innate immunity that can act as a defensive barrier against foreign bodies (Palmer et al., 2008), therefore the elevated prevalence of pigmented tissue recorded at high use sites may

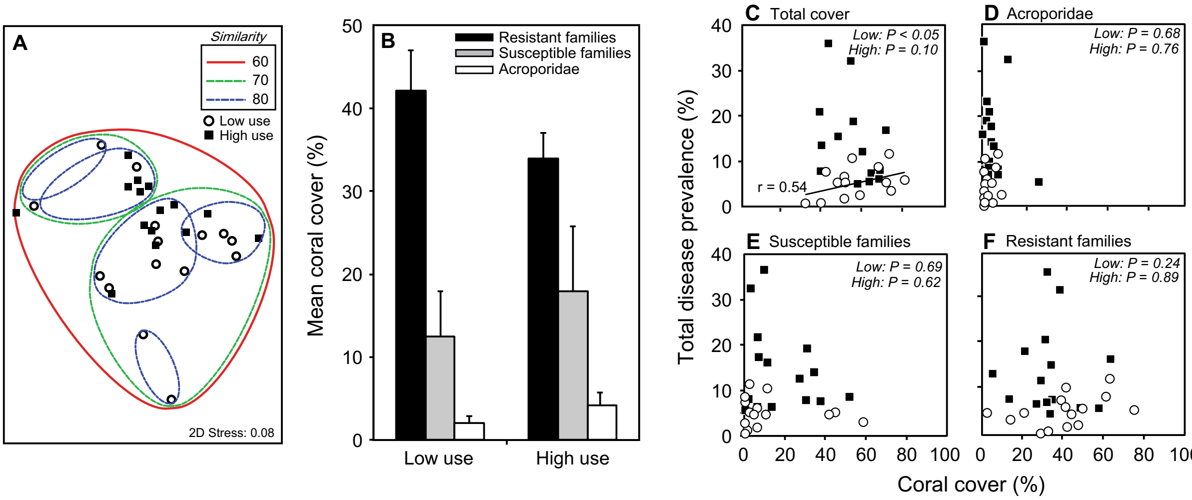

Fig. 5. (A) Spatial variation in the taxonomic composition of percent coral cover by family at the transect level assessed using a non-metric multidimensional scaling (nMDS) plot and hierarchical clusters overlaid from dendrograms based on a Bray–Curtis similarity matrix on square root-transformed data. Distances between transects signifies similarity of coral community composition and the similarity scale on clusters indicates the percentage of similarity between transects (range = 0–100). Taxonomic patterns of (B) mean coral cover (±SE) between sites with low visitor use and associations between prevalence of overall coral disease and percent (C) total coral cover, (D) Acroporidae, (E) susceptible coral families, and (F) resistant coral families at sites. Low use = open circles, High use = black squares. Disease resistant families: Agariciidae, Faviidae, Fungiidae, Merulinidae, and Mussidae; disease susceptible families: Pocilloporidae and Poritidae; and the highly disease susceptible family Acroporidae.

represent signs of a general immune response to a multitude of factors, including invading foreign pathogens, physical injury or sediment accumulation.

4.2. Coral physical injury increases disease susceptibility

The 9-fold increase in the prevalence of recent coral damage at high use sites suggests that physical injury and lacerations from direct diver contact play an important role in increased disease prevalence at these sites. Moreover, corals with physical injury were four times more susceptible to skeletal eroding band disease compared to colonies without injury at high use sites. Ongoing chronic injuries could reduce immune function associated with the regeneration of coral tissue, resulting in increased susceptibility to disease (Mydlarz et al., 2006). In experimental studies, artificially-inflicted wounds enhanced the ability of ciliates associated with skeletal eroding band disease to form dense band-like aggregations that caused tissue loss of up to 0.3 cm day^{-1} on the Great Barrier Reef (Page and Willis, 2008). Increased presence of this ciliate disease has been documented near other tourist locations (Winkler et al., 2004; Lamb and Willis, 2011), however our study is the first to demonstrate a strong link between the prevalence of physical injury and the presence of skeletal eroding band disease. Repair of broken tips takes up to 2 months (Kobayashi, 1984), therefore physical injury may provide a primary site for the invasion of pathogens and ciliates or reduce immune system function, extending the impact timeframe well beyond the immediate time of injury.

Additional microbial or environmental factors at high use sites may be necessary for the development of the band-like ciliate aggregations that cause tissue loss characteristic of skeletal eroding band disease. In contrast to high use sites, injury did not appear to affect the likelihood of skeletal eroding band infections at low use sites. While mean levels of damage found in this study were two times higher than on frequently dived reefs of Saba and Bonaire in the Caribbean (Hawkins et al., 1999, 2005), they were markedly lower than on the more heavily dived reefs of Egypt and Israel, where approximately 10% of colonies were broken (Riegl and Velimirov, 1991; Hawkins and Roberts, 1992). Significant increases in loose fragments of coral at heavily dived sites (Hawkins and Roberts, 1993) raises the possibility of colony-to-colony pathogen transmission if fragments are already infected (Brandt et al., 2013). While marine-based tourist activities do not represent disease agents themselves, they nevertheless appear to cause lesions that compromise the health of corals.

4.3. Spatial and remedial management strategies to manage coral reef health

Results from this study suggest that spatial management strategies to reduce or restrict activities that impact coral health will benefit reef corals, such as in developing coastal regions of Thailand, where fisheries and tourism are valuable for both nutritional and employment purposes (Tapsuwan and Asafu-Adjaye, 2008). Like Koh Tao, many coral reefs are located in poor, developing countries (Donner and Portere, 2007), where use restrictions can undermine local livelihoods and are difficult to justify and enforce (McClanahan et al., 2005). Total prohibition on use, while perhaps ideal from an ecosystem management perspective, may pose an unrealistically difficult burden on local communities and consequently result in little support or compliance (Cinner et al., 2009). Users are generally more likely to support restrictions on specific types of use rather than outright closures (McClanahan et al., 2005). In addition to suggestions discussed above, rotational dive site use or mooring exclusivity to a single operator may be preferred to complete site restrictions. On the Great Barrier Reef, mean disease prevalence was generally less than 1% at several popular dive sites that were visited irregularly or frequently rotated by permitted operators (Lamb and Willis, 2011). Since divers visiting Koh Tao are often diving for the first time, it may be possible to construct alternative training sites by installing appropriate structures, or artificial reefs, in ecologically unobjectionable locations.

Using multiple metrics of coral health may be a more suitable indicator for selecting appropriate management strategies and assessing their success and failure on reefs facing increasing levels of human and disturbance. When site access is unrestricted, individual users have little or no incentive to conserve it, therefore alternative and practical management options that have greater potential for compliance in developing tropical countries are urgently required. The economic value of coral reef tourism for developing coastal communities highlights the importance of improved management practices for conserving the coral reef resource underpinning the industry. Educating and involving local communities in sustainable practices that provide long-term revenues can decrease over-exploitation for short-term profits.

Acknowledgements

Funding for this study was provided by an Australian Institute of Marine Science and James Cook University (AIMS@JCU) International Research Award to JBL, an Australian Research Council Discovery Grant to BLW, and Prince of Songkla University Research Grant No. SCI530221S to JDT and SP. We thank Chad Scott and Devrim Zahir of the New Heaven Dive School in Koh Tao and Aorn Sillapasathitwong and Theeranai Phetsom from the Prince of Songkla University for logistical and field support.

Appendix A. Supplementary material

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.biocon.2014.06.027.

References

Aeby, G.S., Williams, G.J., Franklin, E.C., Haapkyla, J., Harvell, C.D., et al., 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 (2), e16887.
Aeby, G.S., Williams, G.J., Franklin, E.C., Kenyon, J., Cox, E.F., et al., 2011b. Patterns of coral disease across the Hawaiian archipelago: relating disease to environment. PLoS ONE 6 (5), e20370.
Altizer, S., Ostfeld, R.S., Johnson, P.T., Kutz, S., Harvell, C.D., 2013. Climate change and infectious diseases: from evidence to a predictive framework. Science 341 (6145), 514–519.
Anderson, M.J., Gorley, R.N., Clarke, K.R., 2008. PERMANOVA+ for PRIMER: Guide to Software and Statistical Methods. PRIMER-E, Plymouth, UK.
Baker, D.M., Rodríguez-Martínez, R.E., Fogel, M.L., 2013. Tourism's nitrogen footprint on a Mesoamerican coral reef. Coral Reefs, 1–9.
Beeden, R., Willis, B., Raymundo, L., Page, C., Weil, E., 2008. Underwater Cards for Assessing Coral Health on Indo-Pacific Reefs. Coral Reef Targeted Research and Capacity Building for Management Program. Currie Communications, Melbourne, <www.gefcoral.org>.
Bellwood, D.R., Hughes, T.P., Folke, C., Nystrom, M., 2004. Confronting the coral reef crisis. Nature 429, 827–833.
Birkeland, C., 1997. Life and Death of Coral Reefs. Chapman and Hall, New York.
Bongiorni, L., Rinkevich, B., 2005. The pink-blue spot syndrome in *Acropora eurystoma* (Eilat, Red Sea): a possible marker of stress? Zoology 108, 247–256.
Brandt, M.E., Smith, T.B., Correa, A.M.S., Vega-Thurber, R., 2013. Disturbance driven colony fragmentation as a driver of a coral disease outbreak. PLoS ONE 8, e57164.
Brown, B.E., 1997. Coral bleaching: causes and consequences. Coral Reefs 16, 129–138.
Bruno, J.F., Petes, L.E., Harvell, C.D., Hettinger, A., 2003. Nutrient enrichment can increase the severity of coral diseases. Ecol. Lett. 6, 1056–1061.
Buckley, R., 2012. Sustainable tourism: research and reality. Ann. Tourism Res. 39 (2), 528–546.
Burke, L.M., Reytar, K., Spalding, M., Perry, A., 2011. Reefs at Risk Revisited. World Resources Institute, Washington, DC.
Cinner, J.E., McClanahan, T.R., Graham, N.A.J., Pratchett, M.S., Wilson, S.K., Raina, J.-B., 2009. Gear-based fisheries management as a potential adaptive response to climate change and coral mortality. J. Appl. Ecol. 46, 724–732.
Clarke, K.R., Gorley, R.N., 2006. PRIMER v6: User Manual/Tutorial. PRIMER-E Ltd., Plymouth, UK.
Connell, J.H., 1997. Disturbance and recovery of coral assemblages. Coral Reefs S16, 101–113.
Danovaro, R., Bongiorni, L., Corinaldesi, C., Giovannelli, D., Damiani, E., Astolfi, P., Greci, L., Pusceddu, A., 2008. Sunscreens cause coral bleaching by promoting viral infections. Environ. Health Perspect. 116, 441–447.
Davis, D., Tisdell, C., 1995. Recreational scuba-diving and carrying capacity in marine protected areas. Ocean Coast. Manage. 26 (1), 19–40.
Dinsdale, E.A., Harriott, V.J., 2004. Assessing anchor damage on coral reefs: a case study in selection of environmental indicators. Environ. Manage. 33 (1), 126–139.
Donner, S.D., Portere, D., 2007. The inequity of the global threat to coral reefs. Bioscience 57, 157–162.
Garren, M., Raymundo, L., Guest, J., Harvell, C.D., Azam, F., 2009. Resilience of coral-associated bacterial communities exposed to fish farm effluent. PLoS ONE 4 (10), e7319.
Glas, M.S., Sato, Y., Ulstrup, K.E., Bourne, D.G., 2012. Biogeochemical conditions determine virulence of black band disease in corals. ISME J. 6 (8), 1526–1534.
Gronewold, A.D., Borsuk, M.E., Wolpert, R.L., Reckhow, K.H., 2008. An assessment of fecal indicator bacteria-based water quality standards. Environ. Sci. Technol. 42 (13), 4676–4682.
Guilherme Becker, C., Dalziel, B.D., Kersch-Becker, M.F., Park, M.G., Mouchka, M., 2013. Indirect effects of human development along the coast on coral health. Biotropica 45 (3), 401–407.
Haapkylä, J., Unsworth, R.K.F., Flavell, M., Bourne, D.G., Schaffelke, B., Willis, B.L., 2011. Seasonal rainfall and runoff promote coral disease on an inshore reef. PLoS ONE 6, e16893.
Harvell, C.D., Kim, K., Burkholder, J.M., Colwell, R.R., Epstein, P.R., Grimes, E.E., Hoffmann, E., Lipp, K., Osterhaus, A.D.M.E., Overstreet, R.M., Porter, J.W., Smith, G.W., Vasta, G.R., 1999. Emerging marine diseases–climate links and anthropogenic factors. Science 285, 1505–1510.
Hawkins, J.P., Roberts, C.M., 1992. Effects of recreational SCUBA diving on fore-reef slope communities of coral reefs. Biol. Conserv. 62, 171–178.
Hawkins, J.P., Roberts, C.M., 1993. Effects of recreational scuba diving on coral reefs: trampling on reef-flat communities. J. Appl. Ecol., 25–30.
Hawkins, J.P., Roberts, C.M., Van'T Hof, T., De Meyer, K., Tratalos, J., Aldam, C., 1999. Effects of recreational scuba diving on Caribbean coral and fish communities. Conserv. Biol. 13 (4), 888–897.
Hawkins, J.P., Roberts, C.M., Kooistra, D., Buchan, K., White, S., 2005. Sustainability of scuba diving tourism on coral reefs of Saba. Coast. Manage. 33 (4), 373–387.
Hodgson, G., 1990. Tetracycline reduces sedimentation damage to corals. Mar. Biol. 104, 493–496.
Hoegh-Guldberg, O., Mumby, P.J., Hooten, A.J., Steneck, R.S., Greenfield, P., Gomez, E., Harvell, C.D., Sale, P.F., Edwards, A.J., Caldeira, K., Knowlton, N., Eakin, C.M., Iglesias-Prieto, R., Muthiga, N., Bradbury, R.H., Dubi, A., Hatziolos, M.E., 2007. Coral reefs under rapid climate change and ocean acidification. Science 318 (5857), 1737–1742.
Hubbard, J.A.E.B., Pocock, Y.P., 1972. Sediment rejection by recent scleractinian corals: a key to paleo-environmental reconstruction. Geol. Rundsch. 61, 598–626.
Jones, R.J., 2011. Environmental effects of the cruise tourism boom: sediment resuspension from cruise ships and the possible effects of increased turbidity and sediment deposition on corals (Bermuda). Bull. Mar. Sci. 87, 659–679.
Jones, R.J., Muller, J., Haynes, D., Schreiber, U., 2003. Effects of herbicides diuron and atrazine on corals of the Great Barrier Reef, Australia. Mar. Ecol. Prog. Ser. 251, 153–167.
Judd, S., 2010. The MBR Book: Principles and Applications of Membrane Bioreactors for Water and Wastewater Treatment. Elsevier.
Kaczmarsky, L., 2006. Coral disease dynamics in the central Philippines. Dis. Aquat. Organ. 69, 9–21.
Kline, D.I., Kuntz, N.M., Breitbart, M., Knowlton, N., Rohwer, F., 2006. Role of elevated organic carbon levels and microbial activity in coral mortality. Mar. Ecol. Prog. Ser. 314, 119–125.
Kobayashi, A., 1984. Regeneration and regrowth of fragmented colonies of the hermatypic corals *Acropora formosa* and *Acropora nasuta*. Galaxea 3, 13–23.
Kushmaro, A., Rosenberg, E., Fine, M., Loya, Y., 1997. Bleaching of the coral *Oculina patagonica* by Vibrio AK-1. Mar. Ecol. Prog. Ser. 147 (1), 159–165.
Lafferty, K.D., Holt, R.D., 2003. How should environmental stress affect the population dynamics of disease? Ecol. Lett. 6 (7), 654–664.
Lamb, J.B., Willis, B.L., 2011. Using coral disease prevalence to assess the effect of concentrating tourism activities on offshore reefs in a tropical marine park. Conserv. Biol. 25 (5), 1044–1052.
Larpnun, R., Scott, C., Surasawadi, P., 2011. Practical coral reef management on small island: controlling sediment on Koh Tao, Thailand. In: Wilkinson, C., Brodie, J. (Eds.), Catchment Management and Coral Reef Conservation: A Practical Guide for Coastal Resource Managers to reduce Damage from Catchment Areas based on Best Practice Case Studies. Global Coral Reef Monitoring Network and Reef and Rainforest Research Centre, Townsville, Australia, p. 120 pp.
McCallum, H.I., Kuris, A., Harvell, C.D., Lafferty, K.D., Smith, G.W., Porter, J., 2004. Does terrestrial epidemiology apply to marine systems? Trends Ecol. Evol. 19 (11), 585–591.
McClanahan, T.R., Maina, J., Davies, J., 2005. Perceptions of resource users and managers towards fisheries management options in Kenyan coral reefs. Fish. Manage. Ecol. 12 (2), 105–112.
Muthiga, N.A., McClanahan, T.R., 1997. The effect of visitor use on the hard coral communities of the Kisite Marine Park, Kenya. In: Proceedings of the 8th international coral reef symposium, vol. 2, pp. 1879–1882.
Mydlarz, L.D., Jones, L.E., Harvell, C.D., 2006. Innate immunity, environmental drivers and disease ecology of marine and freshwater invertebrates. Evol. Syst. 37, 251–288.
Nyström, M., Folke, C., Moberg, F., 2000. Coral reef disturbance and resilience in a human dominated environment. Trends Ecol. Evol. 15 (10), 413–417.
Ong, T.F., Musa, G., 2011. An examination of recreational divers' underwater behaviour by attitude–behaviour theories. Curr. Issues Tourism 14 (8), 779–795.
Owen, R., Knap, A.H., Toaspern, M., Carbery, K., 2002. Inhibition of coral photosynthesis by the antifouling herbicide Irgarol 1051®. Mar. Pollut. Bull. 44, 623–632.
Page, C.A., Willis, B.L., 2008. Epidemiology of skeletal eroding band on the Great Barrier Reef and the role of injury in the initiation of this widespread coral disease. Coral Reefs 27, 257–272.
Palmer, C.V., Mydlarz, L.D., Willis, B.L., 2008. Evidence of an inflammatory-like response in non-normally pigmented tissues of two scleractinian corals. Proc. Roy. Soc. B: Biol. Sci. 275, 2687–2693.

Pastorok, R.A., Bilyard, G.R., 1985. Effects of sewage pollution on coral-reef communities. Mar. Ecol. Prog. Ser. 21, 175–189.
Patterson, K.L., Porter, J.W., Ritchie, K.B., Polson, S.W., Mueller, E., Peters, E.C., Santavy, D.L., Smith, G.W., 2002. The etiology of white pox, a lethal disease of the Caribbean Elkhorn coral, *Acropora palmata*. Proc. Natl. Acad. Sci. 99, 8725–8730.
Philipp, E., Fabricius, K., 2003. Photophysiological stress in scleractinian corals in response to short-term sedimentation. J. Exp. Mar. Biol. Ecol. 287, 57–78.
Pollock, F.J., Lamb, J.B., Field, S.N., Heron, S.F., Schaffelke, B., Shedrawi, G., Bourne, D.G., Willis, B.L., 2014. Sediment and turbidity associated with offshore dredging increase coral disease prevalence on nearby reefs. PLoS ONE 9 (7), e102498.
R Core Team, 2013. R: A Language and Environment for Statistical Computing. R Foundation for Statistical Computing, Vienna, Austria. <www.R-project.org>.
Raymundo, L.J., Halford, A.R., Maypa, A.P., Kerr, A.M., 2009. Functionally diverse reef-fish communities ameliorate coral disease. Proc. Natl. Acad. Sci. 106 (40), 17067–17070.
Riegl, B., 2002. Effects of the 1996 and 1998 positive sea-surface temperature anomalies on corals, coral diseases and fish in the Arabian Gulf (Dubai, UAE). Mar. Biol. 140, 29–40.
Riegl, B., Velimirov, B., 1991. How many damaged corals in Red Sea reef systems? A quantitative survey. In: Coelenterate Biology: Recent Research on Cnidaria and Ctenophora. Springer, Netherlands, pp. 249–256.
Rogers, C., 1990. Responses of coral reefs and reef organisms to sedimentation. Mar. Ecol. Prog. Ser. 62, 185–202.
Rose, C.S., Risk, M.J., 1985. Increase in *Cliona delitrix* infestation of *Montastrea cavernosa* heads on an organically polluted portion of the Grand Cayman fringing reef. Mar. Ecol. 6, 345–363.
Ruiz-Moreno, D., Willis, B.L., Page, A.C., Weil, E., Cróquer, A., Vargas-Angel, B., Jordan-Garza, A.G., Jordán-Dahlgren, E., Raymundo, L., Harvell, C.D., 2012. Global coral disease prevalence associated with sea temperature anomalies and local factors. Dis. Aquat. Organ. 100, 249–261.
Rützler, K., Muzik, K., 1993. *Terpios hoshinota*, a new cyanobacteriosponge threatening Pacific reefs. Sci. Mar. 57, 395–403.
Sato, Y., Willis, B.L., Bourne, D.G., 2009. Successional changes in bacterial communities during the development of black band disease on the reef coral, *Montipora hispida*. ISME J. 4 (2), 203–214.
Sutherland, K.P., Porter, J.W., Torres, C., 2004. Disease and immunity in Caribbean and Indo-Pacific zooxanthellate corals. Mar. Ecol. Prog. Ser. 266, 273–302.
Tang, S.-L., Hong, M.-J., Liao, M.-H., Jane, W.-N., Chiang, P.-W., Chen, C.-B., Chen, C.A., 2011. Bacteria associated with an encrusting sponge (*Terpios hoshinota*) and the corals partially covered by the sponge. Environ. Microbiol. 13, 1179–1191.
Tapsuwan, S., Asafu-Adjaye, J., 2008. Estimating the economic benefit of scuba diving in the Similan Islands, Thailand. Coast. Manage. 36, 431–442.
Veron, J.E.N., 2000. Corals of the World, vols. 1–3. Australian Institute of Marine Science.
Weterings, R., 2011. A GIS-based assessment of threats to the natural environment on Koh Tao, Thailand. Kasetsart J. (Nat. Sci.) 755, 743–755.
Willis, B.L., Page, C.A., Dinsdale, E.A., 2004. Coral disease on the Great Barrier Reef. In: Coral Health and Disease. Springer-Verlag, Berlin.
Winkler, R., Antonius, A., Abigail Renegar, D., 2004. The skeleton eroding band disease on coral reefs of Aqaba, Red Sea. PSZNI Mar. Ecol. 25, 129–144.
Yeemin, T., Sutthacheep, M., Pettongma, R., 2006. Coral reef restoration projects in Thailand. Ocean Coast. Manage. 49, 562–575.
Yousef, Y.A., McLellon, W.M., Zebuth, H.H., 1980. Changes in phosphorus concentrations due to mixing by motorboats in shallow lakes. Water Res. 14 (7), 841–852.
Zakai, D., Chadwick-Furman, N.E., 2002. Impacts of intensive recreational diving on reef corals at Eilat, northern Red Sea. Biol. Conserv. 105 (2), 179–187.