Protected areas mitigate diseases of reef-building corals by reducing damage from fishing

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Abstract. Parks and protected areas have been instrumental in reducing anthropogenic sources of damage in terrestrial and aquatic environments. Pathogen invasion often succeeds physical wounding and injury, yet links between the reduction of damage and the moderation of disease have not been assessed. Here, we examine the utility of no-take marine reserves as tools for mitigating diseases that affect reef-building corals. We found that sites located within reserves had fourfold reductions in coral disease prevalence compared to non-reserve sites (80 466 corals surveyed). Of 31 explanatory variables assessed, coral damage and the abundance of derelict fishing line best explained differences in disease assemblages between reserves and non-reserves. Unexpectedly, we recorded significantly higher levels of disease, coral damage, and derelict fishing line in non-reserves with fishing gear restrictions than in those without gear restrictions. Fishers targeting stocks perceived to be less depleted, coupled with enhanced site access from immediately adjacent boat moorings, may explain these unexpected patterns. Significant correlations between the distance from mooring sites and prevalence values for a ciliate disease known to infest wounded tissue ($r = -0.65$), coral damage ($r = -0.64$), and the abundance of derelict fishing line ($r = -0.85$) corroborate this interpretation. This is the first study to link disease with recreational use intensity in a park, emphasizing the need to evaluate the placement of closures and their direct relationship to ecosystem health. Since corals are modular, ecological processes that govern reproductive and competitive fitness are frequently related to colony surface area therefore, even low levels of cumulative tissue loss from progressing diseases pose significant threats to reef coral persistence. Disease mitigation through reductions in physical injury in areas where human activities are concentrated is another mechanism by which protected areas may improve ecosystem resilience in a changing climate.

Key words: ciliates; coral reefs; disease; fishing line; Great Barrier Reef; no-take marine reserves; pathogens; physical injury; resilience; skeletal eroding band; white syndromes.

INTRODUCTION

Aquatic and terrestrial resources are under increasing pressure to provide food, employment, and recreation for millions of people, but there is widespread concern that increasing and often conflicting usage is leading to progressive degradation of ecosystem health (Tscharntke et al. 2005, Worm et al. 2006, Halpern et al. 2008). Diseases have emerged as a global threat to the conservation of many species (Altizer et al. 2013), at least partly because environmental conditions have been altered by human activities that compromise immune defenses or enhance the virulence of pathogens (Harvell et al. 2009). Although diseases may not immediately kill their hosts, they often reduce their fitness by deleteriously

affecting fecundity and growth, behavior, and resistance to other climate-driven impacts (Harvell et al. 2009, Wobeser 2013). The need to evaluate the veracity of management practices designed to protect ecosystem health is becoming increasingly urgent.

Tissue abrasions and injuries are known to facilitate disease development by providing a primary site for the invasion of pathogens or parasites in a wide variety of taxonomic groups, such as humans and other large-bodied mammals (Anderson and May 1991, Wobeser 2013), fishes (Austin and Austin 2007), trees and plants (Underwood 2012), insects (Ferrandon et al. 2007), and marine invertebrates like sponges and corals (Henry and Hart 2005, Mydlarz et al. 2006). Moreover, invertebrate immune responses are known to be depleted during regeneration of wounds, resulting in reduced capacity to develop an immune response following exposure to a foreign substance, further increasing the likelihood of disease development (Mydlarz et al. 2006). Protecting flora and fauna from physical disturbances associated

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with human use has prompted spatial management solutions, such as restricting site access or activities allowed within designated areas (De'ath et al. 2012, Newsome and Moore 2012). Many parks and protected areas have been instrumental in reducing damage in terrestrial and aquatic environments (Leung and Marion 2000, Dahlgren 2004), yet links between the reduction of damage and mitigation of disease have not been assessed, for either marine or terrestrial protected areas.

Outbreaks of diseases that affect reef-building corals have recently emerged as a significant driver of global coral reef degradation (Harvell et al. 2007). Coral reefs are predominantly managed through the creation of protected areas; thus reefs are an ideal model system to assess whether protected areas mitigate disease by reducing levels of human use. Several lines of reasoning suggest that protected areas are likely to influence levels of disease in coral populations, although influences could be either beneficial or detrimental to coral health. For example, areas that exclude activities that damage corals may reduce disease prevalence by limiting injuries that facilitate an entry point for coral pathogens (Page and Willis 2008, Nicolet et al. 2013, Katz et al. 2014, Lamb et al. 2014, Pollock et al. 2014). Higher levels of coral disease at sites associated with high-intensity tourism (Lamb and Willis 2011, Lamb et al. 2014) support this hypothesis. In addition, many of the fishing methods and gear types used to catch coral reef fishes cause direct physical damage to corals (Roberts 1995, Bavestrello et al. 1997, Schleyer and Tomalin 2000, Asoh et al. 2004, Yoshikawa and Asoh 2004, Mangi and Roberts 2006). On the other hand, protected areas might facilitate the spread of pathogens through host populations by increasing cover or density of susceptible individuals (Bruno et al. 2007). Further assessments of these opposing predictions are needed to evaluate the value of protected areas in disease mitigation.

Fishing also has the potential to influence coral disease through indirect shifts in reef fish community structure. Loss of functional diversity and feeding guilds in reef fish communities (Mouillot et al. 2014) could disrupt the balance between corals, competitors, and organisms that act as potential vectors or reservoirs of pathogens. For example, high densities of herbivorous fish within reserves could reduce negative algal–coral interactions (Jompa and McCook 2003) by limiting growth of algae (Bellwood et al. 2003), which have been reported to act as reservoirs of pathogens on both Caribbean and Indo-Pacific reefs (Nugues et al. 2004, Smith et al. 2006). In addition to reducing habitat complexity, line fishing predominantly targets piscivo-rous species that are important in structuring coral reef fish assemblages (Hixon and Webster 2002), and indirectly, benthic communities (Roberts 1995, Graham et al. 2003, Mumby et al. 2006). Direct targeting of herbivorous fish by fishing has been implicated in reduced grazing pressure and subsequent shifts from coral to algal dominance on coral reefs (Mumby et al.

2006). Moreover, reserves could increase disease prevalence if they increase densities of fishes that cause changes in coral-associated microbial communities towards more pathogenic taxa (Casey et al. 2014), or act as vectors for coral pathogens by injuring coral tissues during feeding (Aeby and Santavy 2006, Raymundo et al. 2009).

Marine reserves within the Great Barrier Reef Marine Park (GBRMP) represent a particularly relevant case study to test the utility of protected areas as a management tool for disease mitigation. Corals within the GBRMP have been classified as the world's least threatened (Burke et al. 2011), citing only minor impacts of local anthropogenic disturbances, such as anchor damage, pollution, vessel groundings, and oil spills to date (De'ath et al. 2012). Thus, no other factors confound examination of the potential roles that recreational fishing impacts and reef fish assemblage structure may have in influencing coral disease prevalence. Furthermore, many inshore fringing reefs of the GBRMP are exposed to high levels of recreational use, with highly concentrated fishing effort in non-reserve areas and relatively effective protection from fishing within the reserves (Higgs and McInnes 2001, Day 2008). Although rarely measured, compliance with spatial fishing restrictions on the GBRMP is often assumed to be reasonable, particularly on near-shore reefs where surveillance and enforcement activities are relatively effective (Day 2008). For all these reasons, the GBRMP facilitates robust comparisons of coral health between reefs within reserves and in non-reserve areas that are exposed to recreational fishing activities.

In this study, we examine the utility of reserves to mitigate diseases that affect reef corals in the most recreationally fished inshore region of the GBRMP. We tested multiple factors, including differences in protected area status, coral injury, structure of reef fish assemblages, fishing gear restrictions, and several habitat and environmental characteristics, to evaluate variation in the assemblages of coral diseases and in individual types of disease between reserve and non-reserve zones.

METHODS

Study location and protected areas management

We conducted this study on fringing inshore coral reefs in the Whitsunday Islands (20°08′ S, 148°56′ E), a group of 53 islands located between ~2 km and 30 km from the mainland (Fig. 1). The islands are destinations for approximately half of the 1.4 million tourists that visit the GBRMP each year (Harriott 2002); thus recreational hook and line fishing pressure on the narrow fringing reef communities is very high (Higgs and McInnes 2001). Reefs in three management zones were surveyed to assess the efficacy of reserves as tools for mitigating coral disease (Fig. 1). Marine National Parks (MNP) are no-take reserves (reserves) where extractive activities, including fishing and collecting, are prohibited. Habitat Protection (HP) zones are open to
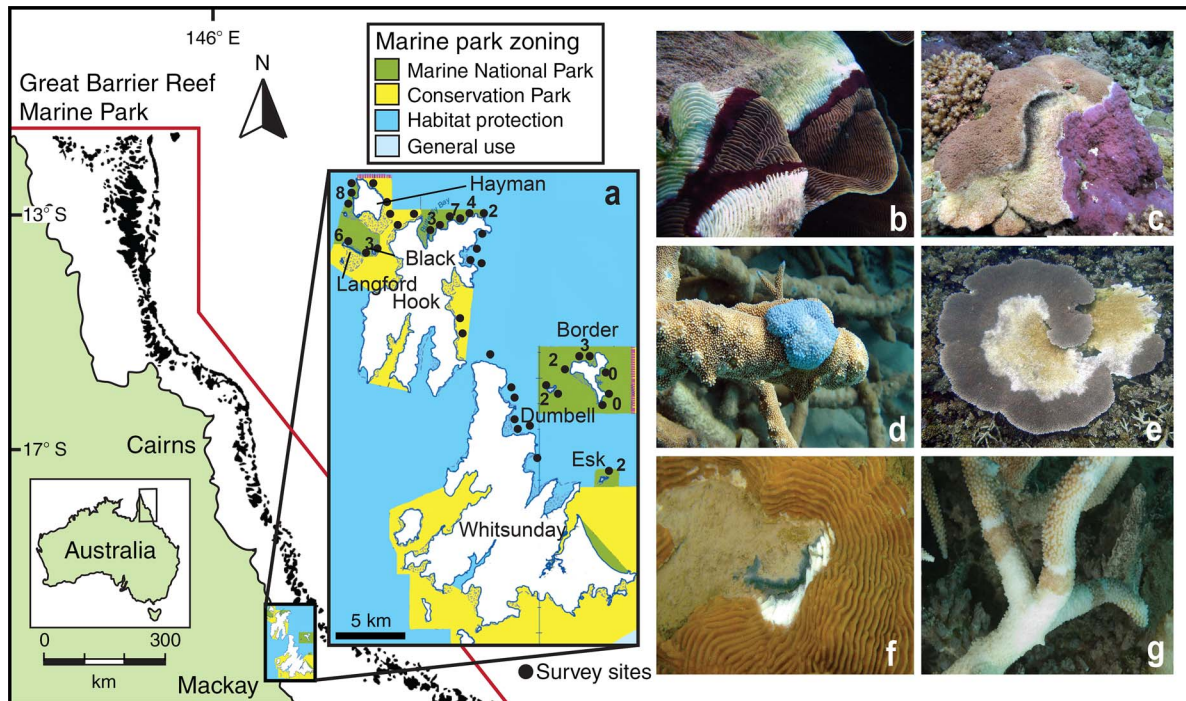
FIG. 1. Regional map of the Whitsunday Island group and sites surveyed within each of three management zones in the Great Barrier Reef Marine Park, Australia. (a) Areas shaded in green are no-take reserves and represent 33% of zones in the marine park; areas shaded in dark blue are open to fishing and comprise 28% of the marine park; areas shaded in yellow are open to fishing but with fishing gear limitations. Activities permitted in each zone are listed in the Appendix: Table A1 and at www.gbrmpa.gov.au. Black circles indicate survey sites and numbers within reserves indicate locations and numbers of permanent boat moorings. Photographs of six diseases commonly affecting reef corals in the Indo-Pacific (Beeden et al. 2008): (b) black band disease, (c) skeletal eroding band disease, (d) growth anomalies, (e) white syndromes, (f) atramentous necrosis, and (g) brown band disease.

hook and line fishing, spear fishing, and collecting. Conservation Park (CP) zones are also open to hook and line fishing, although limited to one line and hook per person; however, they are closed to spear fishing and collecting. The protected reefs around Border and Hook Island were zoned as Marine National Parks in 1987 (25 years of protection at the time of the present study). The reserves at Hayman, Langford, Black, Dumbell, and Esk Islands were established in 2004 (8 years of protection). All of the remaining study reefs have always been open to fishing; however the restricted fishing (Conservation Park) zones at Hook and Hayman Islands were also established in 2004.

At the end of the austral spring in late October and November 2012, we surveyed 21 long-term survey sites within MNP reserves ($n = 63$ transects) and 20 long-term sites open to fishing (non-reserves, $n = 60$ transects). These corresponded to sites of ongoing studies to assess the utility of reserves to maintain densities of coral trout (*Plectropomus* spp.), the primary target species of the recreational and commercial hook and line fishery in the Great Barrier Reef Marine Park (Mapstone et al. 2004). Densities of coral trout were shown to increase when protection status was changed from non-reserve to reserve (Mapstone et al. 2004, Russ et al. 2008). Of the sites open to fishing, 11 were within fished HP zones

($n = 33$ transects) and 9 sites ($n = 27$ transects) were within fishing-gear-restricted CP zones (Fig. 1). Additional comprehensive regulations and protection for each zone can be found in the Appendix; also *available online*.[5]

Coral health surveys

At each of the 41 sites, we surveyed coral health on three 15×2 m belt transects (see Plate 1). Transects corresponded to the first 15 m of concurrent transects for underwater visual census (UVC) of fish communities (see *Visual census of reef fishes and environmental data collection*). Within each 30-m^2 belt transect, we identified each coral colony >5 cm in diameter to genus and further classified each coral as either healthy (no disease observed) or affected by one or more of six common Indo-Pacific coral diseases: black band disease, skeletal eroding band, brown band disease, white syndromes, atramentous necrosis, or growth anomalies (Fig. 1 and Beeden et al. 2008). As an estimate of the intensity of site use, we recorded other external indicators of coral health, such as physical injury (recently exposed skeleton from breakage), the abundance and health status of corals entangled in derelict monofilament fishing line,

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[5] www.gbrmpa.gov.au.

PLATE 1. Recording visual signs of coral health and disease in the Great Barrier Reef Marine Park. Photo credit: John Rumney, Eye to Eye Marine Encounters.

apparent tissue death due to sediment accumulation, bleaching, non-normal pigmentation of tissue, and cuts and scars from predation by crown-of-thorns starfish and corallivorous marine snails (Willis et al. 2004, Lamb and Willis 2011, Lamb et al. 2014). We determined benthic coral and macroalgae cover using standard line-intercept surveys along each 15-m transect.

Visual census of reef fishes and environmental data collection

We used modified underwater visual census (UVC) technique to survey 238 species of diurnal, non-cryptic reef fish, from 17 families (see Williamson et al. 2014). Briefly, we deployed five replicate belt transects at each site on reef slopes, parallel to the reef crest and within a depth range of 4–12 m depending on the reef slope topography at each site. Transects were 50×6 m (300-m^2 survey area) for all species other than pomacentrids and small labrids, which we surveyed during return transect swims using a transect width of 2 m (100-m^2 survey area). We conducted fish community UVC surveys on SCUBA using two observers who swam in close proximity to each other. One observer surveyed the predatory species (predominantly Lethrinidae, Lutjanidae, Serranidae, Haemulidae, and larger species of Labridae), while the other surveyed the roving herbivores (predominantly Acanthuridae, Scaridae, and

Siganidae) and other non-fishery target groups (Chaetodontidae, Pomacanthidae, Pomacentridae, and small-bodied Labridae species). A third diver swam ~5 m behind the fish observers deploying the transect tapes. This synchronous transect deployment technique minimized diver avoidance or attraction behaviors of certain fishes and improved the accuracy of the UVC. Since reef topography and habitat complexity affect the abundance of reef fish (Beukers and Jones 1998), habitat structural complexity index (SCI) was calculated using visual estimates of rugosity and slope for each 10-m segment of each 50-m transect (Williamson et al. 2014). The first three transect tapes deployed at each site at the completion of the fish UVC were left in place for the coral community and disease surveys as described previously.

At each site, we sampled five replicate cores of the top 3 cm of bulk sediment along survey transects, with one core taken at intervals of ~10 m. Each sediment core was sampled by driving a 60-mL plastic syringe with the end removed perpendicularly into the sediment in order not to disturb the layers. Replicate cores for each site were placed in a sterile 50-mL polypropylene tube until examined for grain size classification. Replicate sediment cores were classified into an incremental categorical scale ranging between 1 (very coarse) and 10 (very fine) by taking the mode of five measurements for each

TABLE 1. Predictor variables, codes, and units included in the linear distance-based multiple regression model.

Variable	Code	Description and units	Minimum	Maximum
Protection status	Protection status	Categorical	Non-reserve or reserve areas	
Temperature	Temp	°C	25.9	30.4
Depth	Depth	m	4	12
Sediment grain size	GrainSize	$1 \leq \text{size} \leq 10$	3	10
Biological predictors				
Fish density	FishDens	no./200 m^2	45	749
Fish species diversity	FishDiv	H' index	1.2	3.4
Fish taxonomic diversity	FishTaxDiv	Δ index	66.7	98.1
Detritivores†	Dent	no./200 m^2	0	24
Algal croppers†	AlgCrop	no./200 m^2	0	112
Corallivores†	Corallivores	no./200 m^2	0	27
Benthic carnivores†	BenthCarn	no./200 m^2	1	44
Primary target predators†	PrimTarg	no./200 m^2	0	22
Secondary target predators†	SecTarg	no./200 m^2	0	176
Non-target predators†	NonTarg	no./200 m^2	1	32
Omnivorous pomacentrids†	OmPom	no./200 m^2	1	319
Planktivorous pomacentrids†	PlankPom	no./200 m^2	0	104
Territorial pomacentrids†	TerrPom	no./200 m^2	0	359
Excavating grazers†	ExGraz	no./200 m^2	0	23
Scraping grazers†	ScGraz	no./200 m^2	0	193
Benthic predictors				
Coral genera diversity	CoralDiv	H' index	0.03	3.3
Coral genera richness	CoralRich	d index	1.8	7.7
Coral cover	CoralCov	%	0	96.7
Coral density	CoralDens	no./30 m^2	111	2187
Acropora cover	AcroCov	%	0	31.3
Coral bleaching	Bleaching	%	0	16.3
Coral physical damage	Damage	%	0	10.3
Sediment tissue death	SedDeath	%	0	9.5
Coral *Drupella* scars	Pred	%	0	3.3
Fishing line	Fishing line	no./90 m^2	0	10
Macroalgae cover	MacroAlg	%	0	60

† See Appendix: Table A2 for reef fish species placed in each functional group.

sample (see Appendix). Working depth and water temperature at each site were calculated as the average of values recorded every five minutes using a Sensus Ultra temperature and depth recorder (ReefNet Incorporated, Mississauga, Ontario, Canada).

Modeling drivers of disease assemblages and statistical analyses

We calculated coral prevalence for each 30-m^2 belt transect by dividing the number of colonies with disease or other signs of compromised health by the total number of colonies present. A variety of measures of coral and fish community structure were modeled in combination with a number of environmental variables to evaluate their roles in ameliorating coral health (Table 1). Biodiversity indices were calculated according to the lowest taxonomic group using the total number of individuals surveyed per transect area (coral genera per 30 m^2 and fish species per 200 m^2). Prior to inclusion in the model, each fish species was grouped into one of 12 broad functional roles in coral reef habitats, and large- and intermediate-sized predators were grouped according to their fishery status in the GBRMP (Williamson et al. 2014; Appendix: Table A2). In addition, the taxonomic diversity of fish assemblages

(Δ) was calculated for each transect to assess the average relatedness or the degree to which species in a sample were related taxonomically, by measuring the average path length between every pair of species through a taxonomic tree. This measurement has been shown to be more sensitive to disturbance effects than traditional indices (Warwick and Clarke 1995).

Differences in pooled and individual disease types were tested using a univariate three-level nested analysis of variance, where wave exposure (sheltered vs. exposed) and protection status (reserves vs. non-reserves) were fixed factors, and site was nested within exposure and protection status as a random factor. Differences among management zones were examined using a univariate two-level nested analysis of variance, nesting site (random factor) into management zone (fixed factor). When comparisons were found to be significant ($P < 0.05$), we followed analyses with a posteriori Tukey's honestly significant difference (HSD) test. Associations between continuous variables were tested with Pearson product-moment correlations (PPMC), with the confidence interval set at 0.95. Prior to all univariate analyses, we tested assumptions of normality and homogeneity of variance. Data were transformed to meet assumptions of normality where necessary. Uni-
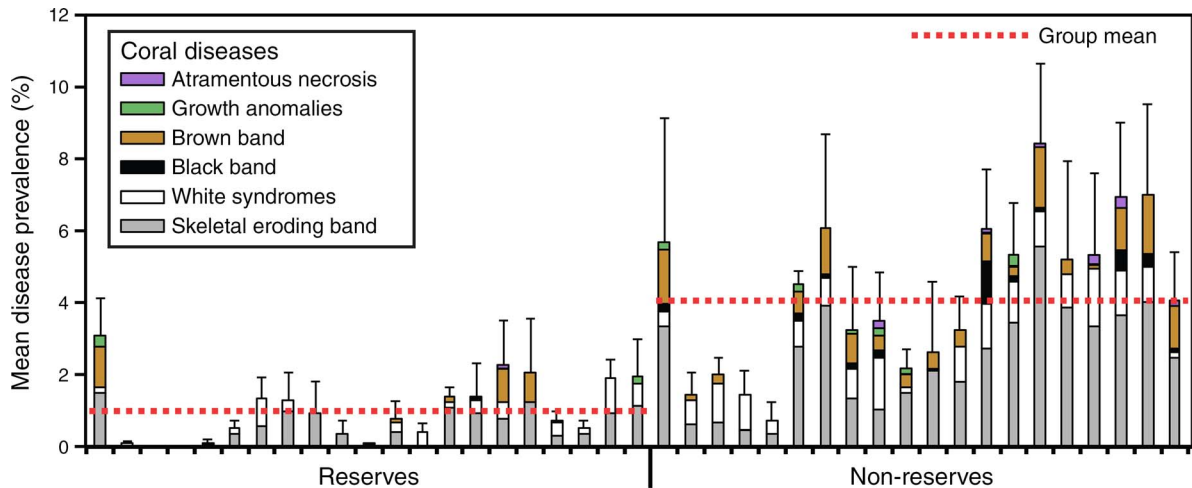
FIG. 2. Mean prevalence (+SE) of six coral diseases surveyed at each site ($n = 3$ replicate transects per site). The dashed line represents the group mean for sites protected from fishing (no-take reserves, $n = 21$ sites, 45 894 corals surveyed) or open to fishing ($n = 20$ sites, 34 972 corals surveyed; restricted and unrestricted fishing gear zones combined).

variate analyses were performed in R v3.0.2 (R Development Core Team 2012).

A multivariate distance-based linear regression model (McArdle and Anderson 2001) was used to measure the strength and significance of the relationships between coral disease assemblages and 31 predictor variables (Table 1). This model is robust to zero-inflated data sets and makes no assumptions about the distribution of the response variable. Regression-based models can be sensitive to variables that are correlated; therefore variables with correlations of >0.80 were identified using draftsman's plots and excluded from the final analysis (Anderson et al. 2008). Fish abundance values were down-weighted using a fourth-root transformation to account for clumped distributions of abundant schooling species (Anderson et al. 2008). Individual predictors were transformed on a case-by-case basis to meet assumptions of normality and then fitted conditionally in a stepwise manner using tests based on 9999 permutations of the residuals under the reduced model (McArdle and Anderson 2001, Anderson et al. 2008). Because of the large number of predictor variables, we based model selection (to obtain the best-fit model while maintaining model parsimony) on Bayesian Information Criterion (BIC [Schwarz 1978]).

To visualize each best-fit model, we used distance-based redundancy plots (dbRDA) (McArdle and Anderson 2001) based on the prevalence patterns between independent observations. The optimal predictor variable vector(s) (model base variables) was overlaid on a bi-plot (Anderson et al. 2008). In addition, variables that might be responsible for any differences detected in the dbRDA plots were investigated by calculating Pearson correlations with RDA axes. All modeling was based on zero-adjusted Bray-Curtis similarity matrices and analyses performed using

PRIMER v6 with PERMANOVA+ (Anderson et al. 2008).

RESULTS

Influence of marine protected areas on coral disease prevalence

Surveys of 80 866 scleractinian coral colonies at sites covering 3660 m^2 of fringing reef revealed that protection from fishing in no-take marine reserves had a significant impact on coral health (Figs. 1 and 2). Overall, pooled coral disease prevalence was ~4 times lower in reserves (1.0% ± 0.2% [mean ± SE], range = 0–2.9%, 272 colonies with disease) than at non-reserve sites (4.1% ± 0.4%; range = 0.7–8.1%, 848 colonies with disease; $F = 43.4$, $P < 0.001$; Fig. 2). Three diseases dominated disease assemblages at all sites, with skeletal eroding band accounting for ~60% of all disease cases, followed by white syndromes (16% of disease cases) and brown-band disease (15% of disease cases). For each of these diseases, prevalence was significantly decreased within reserves (Fig. 2 and Appendix: Table A3). In contrast, reserve protection did not significantly influence the mean prevalence of black-band disease, growth anomalies, or atramentous necrosis, which accounted for the remaining 9% of disease cases overall (Fig. 2). Disease prevalence did not differ significantly among sites differing in exposure to wave energy, either when all diseases were pooled or when they were considered individually (Appendix: Table A3).

Three indicators of disturbance (bleaching, coral damage, and the abundance of derelict fishing line) were significantly higher in non-reserves than in reserves (Appendix: Table A4). Taxonomic diversity of reef fish species (totals recorded across all sites: 238 species, 17 families, 12 functional groups), as well as the mean densities of coral trout (*Plectropomus* spp.) and plank-
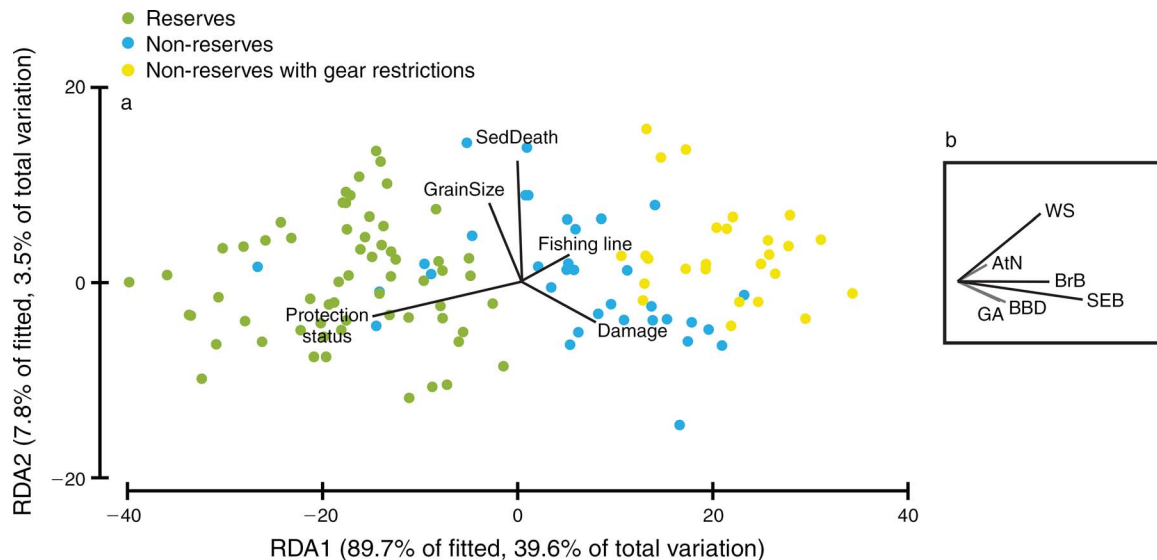
FIG. 3. Distance-based redundancy analysis (dbRDA) indicating the similarity in coral disease assemblages among transects in management zones. Green symbols denote no-take reserves ($n = 63$ transects); blue symbols denote non-reserves ($n = 33$ transects); and yellow symbols denote non-reserves with gear restrictions ($n = 27$ transects). Vectors in (a) depict significant zoning management, environmental, and biological variables (Table 1) forming the best-fit model identified using Bayesian Information Criterion. (b) Vectors represent coral diseases super-imposed on the ordination as vectors (raw Pearson correlations; vectors are offset to the right for ease of distinguishing them from vectors in panel a). The length and direction of the vectors represent the strength and direction of the relationship. The separation of the survey transects indicates a strong zoning protection status gradient increasing along RDA1. Model performed on a Bray-Curtis similarity matrix. SEB = skeletal eroding band, WS = white syndromes, BBD = black band disease, BrB = brown band disease, GA = growth anomalies, and AtN = atramentous necrosis.

tivorous pomacentrids, were all significantly higher within reserves than in non-reserves (Appendix: Table A4). All other benthic characteristics, additional indicators of coral health, density of fish functional groups, and structure of fish assemblages, did not differ significantly between reserves and non-reserves.

Modelled drivers of disease assemblages

Three measures of reserve status and two environmental variables were found to explain a high proportion of the variability in coral disease assemblages (44.2%; Appendix: Table A5). Protection from fishing explained the greatest percentage of the variance identified in the model, accounting for 39.6% of variability in the structure of coral disease assemblages among sites (BIC = 784.7, pseudo-F = 44.8, df = 120, P < 0.001). Sediment accumulation causing apparent coral tissue death was the most important benthic variable, explaining 3.5% of the variability in disease assemblages (BIC = 776.1, pseudo-F = 13.9, df = 119, P < 0.001). Although significant (P < 0.005), the remaining three variables combined (sediment grain size, coral physical damage, and the abundance of derelict fishing line) only represented a further 1.1% of variability in disease assemblages (BIC = 770.5, R^2 = 0.45; Appendix: Table A5).

The distance-based redundancy analysis (dbRDA) used to visualize results of the model revealed that disease assemblages recorded on the 122 transects clearly

separated along the RDA1 axis, which described protection from fishing (89.7% of fitted variation, Fig. 3 and Appendix: Table A5). Unexpectedly, assemblages of coral diseases at non-reserve sites with gear restrictions were more distinct from reserve assemblages along the RDA1 (redundancy plot) axis than assemblages at non-reserve sites without gear restrictions (Fig. 3). Although sediment accumulation causing apparent coral tissue death did not differ significantly between reserve and non-reserve zones (F = 2.4, P = 0.07; Appendix: Table A4), the model analysis revealed that variation in tissue loss from sediment influenced coral disease assemblages across all sites, regardless of protection status (RDA2, 7.8% of fitted variation; Fig. 3A and Appendix: Table A5). To determine which variables were best represented by protection from fishing and tissue death from sediment, raw Pearson correlations of each significant variable identified by the model were examined for correlations with RDA1 and RDA2, respectively. Coral damage (ρ = 0.527) was strongly associated with coral disease assemblages in fished zones, followed by the abundance of derelict fishing line (ρ = 0.346) (Fig. 3a and Appendix: Table A6). On the second axis, smaller sediment grain sizes (ρ = 0.487) were more representative of disease assemblages at sites with increased tissue loss from sediment (Fig. 3a and Appendix: Table A6).

To establish which individual diseases were driving changes in the overall assemblage structure of coral
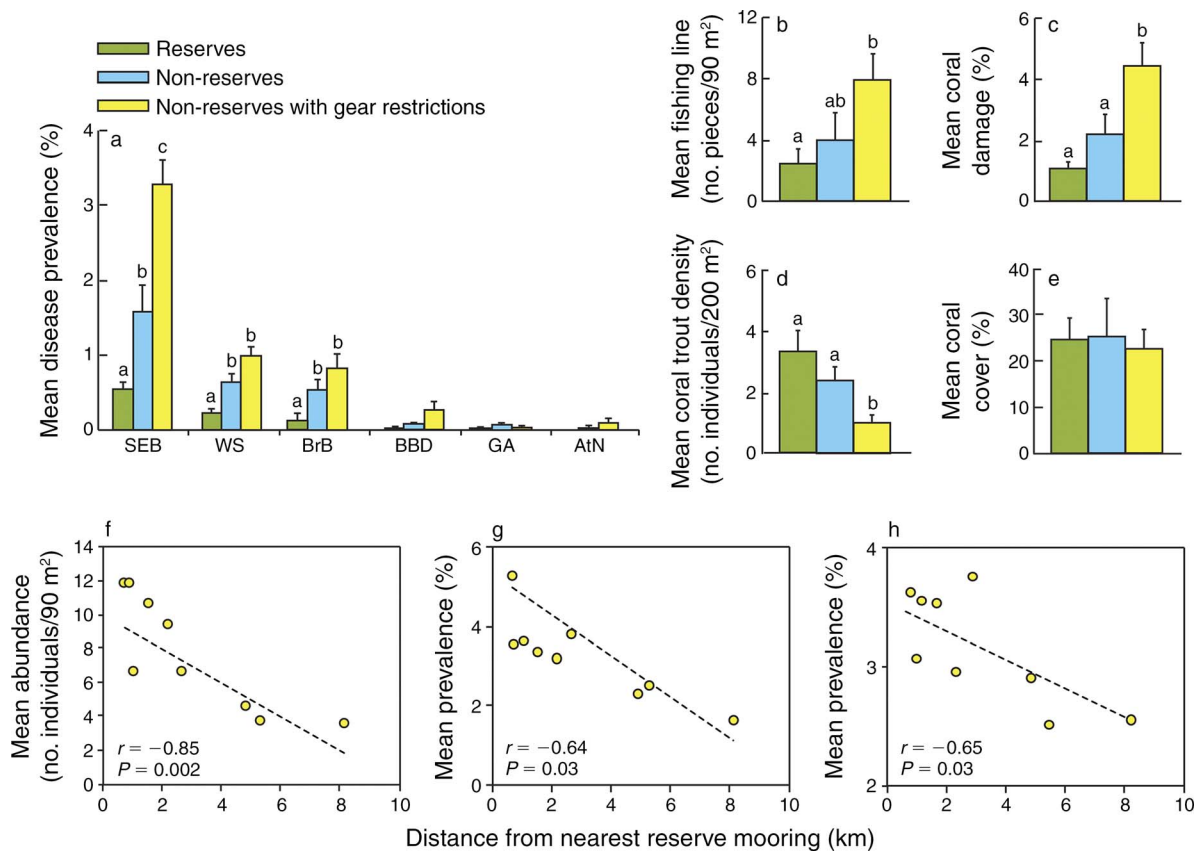
Fig. 4. Comparisons of coral disease and indicators of site use-intensity among three management zones (Top panel, a–e). Mean prevalence (+SE) of: (a) six coral diseases, (b) abundance of derelict fishing line, (c) prevalence of coral damage (recently exposed white skeleton), (d) density of coral trout (*Plectropomus* spp.), and (e) mean percentage of hard coral cover. No-take marine reserves, green bars, $n = 62$ transects; non-reserves: blue bars, $n = 33$ transects; and non-reserves with gear restrictions, yellow bars, $n = 27$ transects. Different letters indicate significant post hoc groups (Tukey HSD, $P < 0.05$). SEB = skeletal eroding band, WS = white syndromes, BBD = black-band disease, BrB = brown-band disease, GA = growth anomalies, and AtN = atramentous necrosis. (Bottom panel, f–h) Associations between distance (km) from the nearest location with permanent boat moorings within non-reserve sites with gear restrictions and the mean: (f) abundance of derelict fishing line, (g) prevalence of recent coral injury (recently exposed white skeleton), and (h) prevalence of skeletal eroding band disease. There were $n = 9$ sites with 3 replicate transects at each site; corals surveyed = 7602. Activities permitted in each zone are listed in the Appendix: Table A1 and www.gbrmpa.gov.au.

diseases, raw Pearson correlations between each disease and the original RDA axes were calculated and overlaid on the dbRDA (Fig. 3b and Appendix: Table A6). Because correlations for each disease were calculated without considering all other diseases contributing to the RDA axes, these results can only be used as a guide. All six coral diseases were more associated with non-reserve sites (positive correlations with RDA1), particularly skeletal eroding band disease ($\rho = 0.710$), brown band disease ($\rho = 0.553$), and white syndromes ($\rho = 0.451$). Black band disease, atramentous necrosis, and growth anomalies were less influenced by reserve protection (Fig. 3b). White syndromes had the strongest correlation with fished sites that also had increased levels of apparent tissue loss from sediment ($\rho = 0.202$) (Fig. 3b). In contrast, skeletal eroding band and brown band disease were associated with fished sites with lower levels

of tissue loss from sediment ($\rho = -0.166$ and $\rho = -0.128$, respectively) (Fig. 3b).

Influence of recreational site-use intensity on disease prevalence

Unexpectedly, coral disease prevalence in non-reserves with gear restrictions was ~2 times higher than in non-reserves without gear restrictions ($5.4\% \pm 0.5\%$; mean $\pm$ SE) vs. $2.9\% \pm 0.2\%$, respectively) and ~5 times higher than in reserves ($1.0\% \pm 0.2\%$; Appendix: Table A7). The prevalence of two diseases (skeletal eroding band and white syndromes) was significantly higher in all non-reserve sites (both gear restricted and unrestricted zones) compared to reserve sites (Fig. 4a). The prevalence of brown band disease did not differ between non-reserves with or without gear restrictions. Prevalence levels of black band disease, growth anomalies, and atramentous necrosis did not differ signifi-

cantly in any comparisons between any of the three management zones (Fig. 4a and Appendix: Table A7).

Similar to the unexpected patterns observed in the prevalence of coral diseases among management zones, the mean abundance of derelict fishing line and prevalence of coral damage (recently exposed white skeleton) reached the greatest levels at sites located in non-reserves with gear restrictions (Fig. 4b, c). In contrast, densities of coral trout were significantly lower in zones with fishing gear restrictions compared to reserves and non-reserves without gear restrictions (Fig. 4d). There was no difference in mean total coral cover among zones (Fig. 4e).

Within non-reserves with gear restrictions, there was an unexpected negative association between the distance (in kilometers) from the nearest area with permanent boat moorings and the mean abundance of derelict fishing line ($r = -0.85$, $P = 0.002$), percent coral damage ($r = -0.64$, $P = 0.03$), and prevalence of skeletal eroding band disease ($r = -0.65$, $P = 0.03$; Fig. 4f–h). Within non-reserves without gear restrictions, there were no significant associations between distance to permanent boat moorings and indicators of use level or of the prevalence of either individual or pooled coral diseases (all: $r < 0.45$, $P > 0.05$).

DISCUSSION

The fourfold lower levels of coral disease within no-take marine reserves compared with non-reserves provides clear evidence that protected areas are a promising approach for mitigating coral disease in locations where the intensity (concentration) of fishing effort is relatively high. Our analysis of 31 variables potentially influencing coral health reveals that protection from fishing is clearly the dominant factor predicting the structure of coral disease assemblages. Given that colony damage and the abundance of derelict fishing line were the major factors driving dissimilarities between reserves and non-reserves, we conclude that it is the activity of fishing itself, rather than changes in fish or other benthic communities caused by fishing, that accounts for the striking differences in disease levels between reserves and zones open to fishing.

Although levels of overall disease prevalence may be low when measured at one point in time, this vastly undervalues the longer-term ecological impacts of disease. Ongoing tissue loss caused by slowly progressing diseases could cause greater levels of coral mortality than immediate but short-term effects associated with tissue injury and structural damage associated with fishing activities. In the Caribbean, two dominant reef-building corals, *Acropora cervicornis* and *A. palmata*, have been nearly extirpated on some reefs by an outbreak of white band disease that caused tissue loss at rates of 0.5 cm/d (Patterson et al. 2002). Part of the reason for the likely undervaluation in our study is that many of these diseases have the potential to rapidly kill whole coral colonies, resulting in unobservable levels of disease-associated mortality during single snapshot surveys. For example, on the Great Barrier Reef, the reported average rate of tissue loss ranges from 1 cm/d for black band disease to 10 cm/d for brown band disease (Boyett 2006, Page and Willis 2008). Since corals are modular, ecological processes that govern reproductive and competitive fitness are commonly related to colony surface area (Zakai et al. 2000, McCook et al. 2001, Leuzinger et al. 2003). Therefore, even low levels of cumulative tissue loss from progressing diseases pose significant threats to reef coral persistence and resilience.

The lack of a significant relationship between coral disease and either the abundance or diversity of fish assemblages suggests that characteristics of reef fish assemblages do not exert a major influence on coral health in this study. Our results are contrary to the conclusion of a recent study on coral health within reserves in the Philippines, which suggested that taxonomic diversity of reef fish assemblages may be the principal driver of differences in the prevalence of coral diseases between reserves and non-reserves (Raymundo et al. 2009). The alternative conclusion reached in their study may reflect differences in the reef fish species targeted by fishers and their role in ecosystem functioning. For example, the removal of major groups of herbivores, such as those targeted in the Philippines (Abesamis et al. 2006), may increase the vulnerability of coral communities to phase shifts towards reefs dominated by algae (Bellwood et al. 2006), which are known to enhance dissolved organic carbon and stimulate microbial growth (Haas et al. 2011). However, such phase shifts have not been observed outside Philippine reserves (Stockwell et al. 2009). In the Great Barrier Reef Marine Park, herbivorous fishes are not targeted and only minimal numbers of large-bodied species are extracted by spear fishers (Mapstone et al. 2004). Evidence that coral trout and other highly targeted predatory fish species exert top-down control of fish assemblages on the Great Barrier Reef is weak at best (Williamson et al. 2014, Rizzari et al. 2015, but see Graham et al. 2003). Ecological similarity of taxa is often used as an indicator of reef degradation (Graham et al. 2006); however neither the dissimilarities in the structure of reef fish assemblages nor reduced densities of coral trout between reserves and non-reserves appear to be driving coral disease prevalence in the present study.

Mechanisms by which fishing activities influence disease

Our conclusion that it is the increased abundance of both derelict fishing line and injured corals in non-reserves compared to reserves that is the primary mechanism driving coral disease prevalence is supported by previous studies that have linked fishing with increased coral breakage (Roberts 1995, Bavestrello et al. 1997, Schleyer and Tomalin 2000, Asoh et al. 2004, Yoshikawa and Asoh 2004, Mangi and Roberts 2006). Injuries are generally assumed to enhance coral disease

transmission because they provide entry wounds for pathogens, disrupt the antibacterial mucus layer on the surface of corals (Ritchie 2006), and redirect energy towards healing processes (Mydlarz et al. 2006). For example, injury increased the susceptibility of corals to colonization by a ciliated protozoan, the pathogen that causes skeletal eroding band disease (Page and Willis 2008, Lamb et al. 2014). Similarly, ciliate infections causing brown band disease have been associated with coral tissue injury and predation by a coral-feeding gastropod (Nicolet et al. 2013) and the crown-of-thorns starfish (Nugues and Bak 2009, Katz et al. 2014). Wound repair in corals takes days to several weeks depending on the size of the injury (Work et al. 2012). Thus, wounds provide an extended period of time for the establishment of ciliate infections. In this study, the two most prevalent diseases, skeletal eroding band and brown band disease, are associated with ciliate-mediated tissue loss. The dominance of ciliate diseases in fished zones provides corroborative evidence for our conclusion that fishing activities causing wounding and breakage have a major impact on coral health.

This study highlights the extent of damage that unregulated fishing concentrated into small areas can have on coral assemblages and the subsequent risk of disease outbreaks if fishing activities are not effectively managed. For example, mean levels of damaged colonies in this study were markedly lower than levels reported on more heavily fished reefs in Hawaii (Asoh et al. 2004), South Africa (Schleyer and Tomalin 2000), and the Mediterranean reefs of northeastern Italy (Bavestrello et al. 1997). Since derelict fishing line can drift or become dislodged, management programs such as reef cleaning by divers has been suggested as a way of reducing impacts of fishing on coral health (Asoh et al. 2004). However, removal of fishing line directly from entangled corals could increase tissue damage, hindering recovery from injury or disease; thus caution is advised in considering such programs.

Apparent tissue loss from sediment sitting on the surface of coral tissues and sediment grain size were significant factors driving variation in disease assemblages, particularly white syndromes in non-reserve zones. Because the prevalence of tissue loss from sediment did not differ between reserves and non-reserves, it is likely that exposure to finer sediment grain sizes, coupled with mechanical damage as a result of fishing activities, caused the increased prevalence of white syndromes outside of reserves. Outbreaks of white diseases have been associated with mechanical sources of damage and stress in the past. On reefs surrounding the Caribbean island of Navassa, high levels of a white plague-like disease occurred one month after high damage associated with the passage of two hurricanes (Miller and Williams 2007), and within weeks of the passage of a hurricane in Puerto Rico (Bruckner and Bruckner 1997). More recently, a disease causing rapid tissue loss in multiple species was associated with colony fragmentation and

physical contact with sediment in the Virgin Islands (Brandt et al. 2013), implying a direct link with injury and sediment. Not only are fine sediment fractions the most difficult for corals to expel and remove (Weber et al. 2006), but fine sediments are also often positively correlated with total organic carbon content (De Falco et al. 2004). In experimental studies, elevated organic carbon contributed to disease development and mortality of corals, suggesting that coral pathogens are carbon-limited (Kline et al. 2006). Taken together, the multiple lines of evidence discussed above suggest that physical disruption of tissue as a result of fishing activities, in addition to reductions in energy resources because of the need to prioritize sediment removal and wound-healing processes, increases the probability of disease.

Levels of site-use influence coral disease

Gear restrictions, such as limitations to the number of fishing lines and hooks allowed, have been suggested as a practical and effective management strategy for reducing coral damage and entanglement associated with line fishing (Asoh et al. 2004). However, our study unexpectedly revealed that the prevalence of coral disease, coral damage, and derelict fishing line all increased significantly in areas open to fishing with gear restrictions compared to those without gear restrictions. It is plausible that fishers perceive stocks in zones without gear restrictions to be more depleted and therefore consciously avoid them. Ease of accessibility is another factor that can affect the amount of fishing pressure an area will experience (Wilcox and Pomeroy 2003). For instance, vessels generally disperse up to a median radius of 5 km from popular boat recreation sites, with a rapid decline in the number of vessels traveling >10 km (Smallwood et al. 2012). The presence of a high number of boat moorings within reserves that are immediately adjacent to zones with gear limitations may explain unexpected increases in coral disease within these latter zones compared to fished zones without gear limitations. This hypothesis is supported by the observed decrease in coral disease, damage, and derelict fishing line with distance from the nearest area with permanent boat moorings. Increases in damage or loss of coral habitat due to disease can lead to habitat fragmentation or patchiness, which has important implications for self-recruitment of many marine organisms (Pinsky et al. 2012) and movement patterns of reef fish (Chapman and Kramer 2000). This can affect the efficacy and management objectives of the marine reserve. These results further support the importance of recognizing the association between levels of human use and disease, particularly the need to evaluate the placement of protected areas and their direct relationship to the health of non-targeted species.

Limitations of protected areas to mitigate disease

The capacity of protected areas to moderate disease will depend upon the mechanism of disease pathogen-

esis. Climate warming is causing profound and often complex changes in the prevalence or severity of infectious diseases affecting plants and animals (Harvell et al. 2009, Altizer et al. 2013), which indicates that environmental factors may be of greater importance in governing disease prevalence than mechanical damage in some cases. Minor differences in the prevalence of coral growth anomalies, black band disease, and atramentous necrosis between reserves and non-reserves suggest that environmental factors enhancing pathogen virulence are more likely to govern the abundance of these diseases than factors associated with fishing activities, which function more by compromising host resistance. For example, the abundance of coral growth anomalies on reefs in Kenya were not influenced by reserve status; rather, anomalous warm water and environmental factors associated with bleaching were implicated (McClanahan et al. 2009). On protected inshore reefs in Australia, increases in the prevalence of atramentous necrosis were linked to seasonal sediment runoff and reduced salinity following monsoonal rain events (Haapkylä et al. 2011), while seasonal fluctuations of seawater temperatures and light were associated with recurrent outbreaks of black band disease (Sato et al. 2009). Thus evidence so far suggests that environmental factors will override benefits provided by protected areas that diminish some diseases. Nevertheless, the results of this study indicate that reserves may improve coral reef resilience in a changing climate by reducing the synergistic impacts of diseases associated with anthropogenically driven injury.

In summary, no-take marine reserves played a significant role in mitigating coral disease on heavily fished inshore fringing reefs in Australia, suggesting an additional conservation tool for reducing coral diseases promoted by physical injury. However, it is stressed that line fishing and injuries were inevitably concentrated in a very narrow band of reef slope habitat on the fringing reefs studied here, and it is likely that this spatial concentration of fishing effort contributed to the clear detection of increased prevalence of coral diseases in non-reserve compared to reserve sites. While protected areas have been proposed and implemented in many ecosystems throughout the world, this is the first study to link disease prevalence to the direct effect of injury caused by human activities.

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Supplemental Material

Ecological Archives

The Appendix is available online: http://dx.doi.org/10.1890/14-1952.1.sm