Functionally diverse reef-fish communities ameliorate coral disease

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Coral reefs, the most diverse of marine ecosystems, currently experience unprecedented levels of degradation. Diseases are now recognized as a major cause of mortality in reef-forming corals and are complicit in phase shifts of reef ecosystems to algal-dominated states worldwide. Even so, factors contributing to disease occurrence, spread, and impact remain poorly understood. Ecosystem resilience has been linked to the conservation of functional diversity, whereas overfishing reduces functional diversity through cascading, top-down effects. Hence, we tested the hypothesis that reefs with trophically diverse reef fish communities have less coral disease than overfished reefs. We surveyed reefs across the central Philippines, including well-managed marine protected areas (MPAs), and found that disease prevalence was significantly negatively correlated with fish taxonomic diversity. Further, MPAs had significantly higher fish diversity and less disease than unprotected areas. We subsequently investigated potential links between coral disease and the trophic components of fish diversity, finding that only the density of coral-feeding chaetodontid butterflyfishes, seldom targeted by fishers, was positively associated with disease prevalence. These previously uncharacterized results are supported by a second large-scale dataset from the Great Barrier Reef. We hypothesize that members of the charismatic reef-fish family Chaetodontidae are major vectors of coral disease by virtue of their trophic specialization on hard corals and their ecological release in overfished areas, particularly outside MPAs.

biodiversity | ecosystem function | marine protected area | coral reef

Coral reefs remain under increasing threat from poor water quality, habitat degradation, and destructive fishing practices (1–3). These disturbances have abetted drastic shifts in reef community structure (4), reduced productivity (5), and lowered resilience (6–8). More recently, outbreaks of infectious diseases have become a significant cause of coral mortality and habitat loss (9). In the Caribbean basin, for example, diseases of coral reef organisms have become the most important factor in the decline of coral reefs throughout that region (10). However, despite a concerted global effort to characterize coral diseases since the early 1990s, the ecological drivers of these phenomena—and the ultimate consequences for coral reef communities—remain poorly understood (11, 12).

Fish are a dominant structuring force on coral reefs, controlling the distribution and abundance of many reef taxa (13–16). However, if subject to sustained heavy fishing, entire functional groups can be lost (17, 18), resulting in a cascade of effects. These can include population increases of species released from predation and competition (19), reduced diversity, and simplified community structure. This, in turn, disrupts numerous weaker, higher-order interactions thought to be directly linked to ecosystem stability (20–22) and which otherwise buffer the effects of agents of mortality, such as pathogens, their vectors, or predators with "boom and bust" population cycles (17, 23, 24). Ultimately, the resilience of coral reefs is compromised (6, 17). In light of evidence that the diversity and abundance of host, vector, or reservoir species can affect the epizootiology of a disease (25–27), we speculate that diverse, less-impacted reef-fish communities can promote coral health.

Hence, to examine whether functionally diverse fish communities play a role in ameliorating coral disease, we surveyed 14 sites [seven marine protected areas (MPAs) and seven adjacent fished sites] across the central Philippines and examined 1,260 m^2 of reef for the disease status of 21,646 coral colonies. We tested the hypothesis that reefs with more intact fish communities had significantly lower levels of coral disease. We used well-managed MPAs to ensure intact fish communities, along with companion sites open to fishing but with otherwise comparable benthic communities, to investigate the potential role of reef fish in coral disease dynamics.

Results and Discussion

Factors varying at spatial scales less than that of the study region (circa 12,000 km^2) appeared responsible for disease prevalence across sites ("site" refers to a portion of a reef that was surveyed; two sites per reef: one MPA and one fished area). Mean prevalence of all six coral diseases recorded (white syndrome, ulcerative white spots, growth anomalies, black band, skeletal eroding band, and brown band) differed widely among sites, from a low of 0.25% to a high of 7.9% (Fig. 1), although the number of diseases per site did not (Wilcoxon $H = -0.808$, $P = 0.4191$). There was a strong spatial component to disease at the regional scale. Sites differed significantly in the prevalence of diseases (two-way ANOVA $F = 13.68$; $P < 0.0001$), with sites closer together more likely to have similar disease prevalence than those farther away ($r = 0.367$, $P = 0.0009$; simple Mantel test). Thus, despite the potentially high connectivity between the sites in this archipelagic marine system, variation in disease prevalence depended more on aspects of transmission operating at the scale of average intersite distances, 10 km–50 km.

Overall, MPAs had a powerful effect on reducing coral disease, with significantly lower disease prevalence than that of unprotected sites ($\bar{x} \pm 1$ SE: 2.8 ± 0.9 vs. 4.5 ± 1.2; two-way ANOVA $F = 5.17$; $P = 0.02$). At all reefs, disease prevalence was lower in MPAs than in fished sites (Fig. 1). These results were not due to differences in percent total coral cover between MPAs and fished sites (57.0 ± 4.5 vs. 57.3 ± 3.4), percent cover of *Porites*, the dominant hard-coral genus and disease host (40.0 ± 3.4 vs. 40.2 ± 4.0), mean total number of coral colonies per transect (342.2 ± 29.1 vs. 356.8 ± 31.4), or physical damage to colonies (0.14 ± 0.02 vs. 0.10 ± 0.02). Moreover, community-level comparisons, as measured by Bray–Curtis similarities in benthic attributes between sites, revealed no differences (global $R = -0.029$, $P = 0.58$) (27). The prevalence of coral disease was instead most strongly reduced on reefs completely protected from fishing; i.e., in MPAs.

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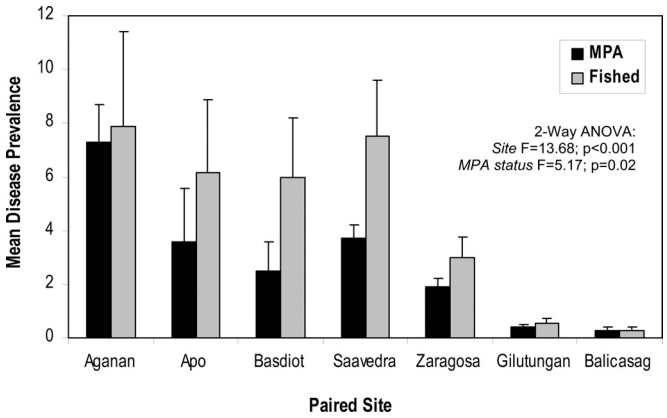

Fig. 1. Mean total disease prevalence for six disease states in 14 sites surveyed in the Central Visayas. Mean ± SE presented; n = 3–6 transects per site.

Because of its manifold potential effects on fish diversity, fishing could be acting in several ways to exacerbate coral disease, and these ways are not necessarily mutually exclusive. Hence, to clarify the relationship between disease and fish diversity per se, we regressed disease prevalence against the average taxonomic distinctness of the fish assemblages at each site. The latter variable summarizes fish diversity as a function of taxonomic relatedness and is allied with trophic and functional diversity (28–30). For example, low distinctness indicates limited taxonomic representation and has been used to indicate reef degradation (31). Disease prevalence was significantly negatively correlated with fish taxonomic distinctness, regardless of a site's protection status, when controlling for spatial effects and coral cover with a partial Mantel test (all sites: $r = -0.753$, $P < 0.001$; Fig. 2*A*). Moreover, six of seven MPAs showed higher taxonomic distinctness than their paired fished sites (paired t test: $t = 2.49$; $P = 0.053$). This relationship suggests an important role, as seen in other systems (24, 32), for high diversity in limiting disease through ecological control of vector species.

To examine this idea, we tested which taxonomic components of fish diversity were linked to variation in coral diseases by separately regressing disease prevalence against densities of the 27 most abundant functional groups/taxa (Table 1). We found no significant associations between coral disease and any functional group/taxon (all sites: $r \leq 0.371$; $P \geq 0.110$). However, when we examined only sites with coral cover ≥50%, a single significant and positive relationship was revealed between disease prevalence and the butterflyfish family Chaetodontidae ($r = 0.680$, $P = 0.028$; Fig. 2*B* and Table 1). This is consistent with a threshold effect of host-coral density in diseases spread through secondary infection (33). Further, within the family, corallivorous species were associated with disease ($r = 0.750$, $P = 0.022$), noncorallivorous species only marginally so ($r = 0.503$, $P = 0.067$), and obligate corallivores explained more variation in disease than did facultative species ($r = 0.686$, $P = 0.039$ vs. $r = 0.587$, $P = 0.049$). Chaetodontids were more abundant at sites with taxonomically depauperate fish assemblages ($r = -0.692$, $P = 0.022$; Fig. 2*C*), most likely because they are not targeted by fishers (34) or are released by other indirect, top-down interactions. These results support the hypothesis that corallivorous butterflyfishes can act as vectors of coral diseases.

To test the generality of these findings in other Indo-Pacific reefs, we examined a second large-scale public-domain dataset (35) from the Great Barrier Reef (GBR), Australia, which permitted comparisons of the abundance of taxonomic components of fish assemblages to counts of diseased coral colonies. Chaetodontids again emerged as the single fish family significantly and positively associated with disease prevalence ($r = 0.513$, $P = 0.0081$; Fig. 2*D* and Table 2) at sites with moderate to high coral cover (≥40%) while controlling for spatial autocorrelation effects and holding coral cover constant.

Although the etiologies of the disease states we documented remain under study and likely involve multiple mechanisms of

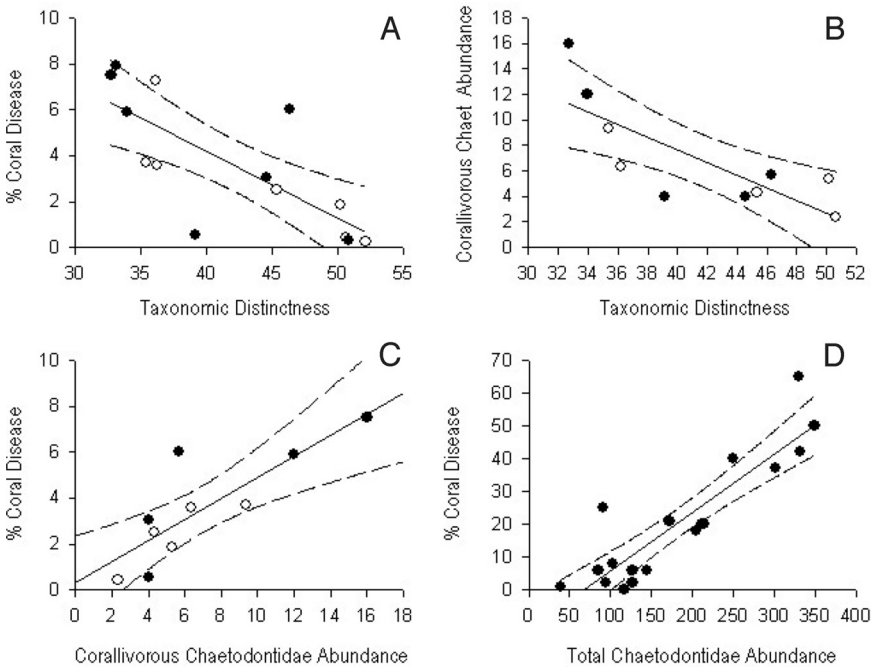

Fig. 2. Simple linear regressions on relationships between coral disease and fish diversity per reef site. Dashed lines represent 95% confidence intervals; closed circles, MPAs; open circles, fished sites. (*A*–*C*) Philippines. (*D*) GBR. (*A*) Average taxonomic distinctness of fish assemblages vs. mean coral disease prevalence, all sites. (*B*) Average taxonomic distinctness vs. chaetodontid abundance, sites with ≥50% live coral cover. (*C*) Chaetodontid abundance vs. mean coral disease prevalence, sites with ≥50% live coral cover. (*D*) Chaetodontid abundance vs. coral disease counts, sites with ≥40% live coral cover.

Table 1. Partial Mantel regressions of prevalence of coral diseases at sites in the Philippines with >50% coral cover on the 10 most abundant taxonomic groups of fishes while holding intersite distances and the percent cover of the dominant coral genus *Porites* constant

Taxon and trophic group	Species, *n*	*r*	*P*
Acanthuridae			
Mixed	27	−0.4238	0.1354
Herbivores	19	−0.2615	0.2621
Planktivores	8	−0.3432	0.2034
Anthiinae			
Herbivores	3	−0.2313	0.2991
Balistidae			
Mixed	7	−0.2925	0.2573
Planktivores	2	−0.2475	0.2939
Invertivores	2	−0.5654	0.0589
Omnivores	3	0.0000	0.9999
Carangidae			
Piscivores	9	−0.1504	0.3851
Chaetodontidae			
Mixed	29	0.6800	0.0275*
Planktivores	2	−0.4222	0.1506
Omnivores	15	0.5025	0.0674
Corallivores	12	0.7497	0.0224*
Facultative corallivores	5	0.5875	0.0486*
Obligate corallivores	7	0.6463	0.0392*
Epinephilinae			
Piscivores	13	−0.3070	0.2258
Labridae			
Mixed	54	−0.2638	0.2707
Corallivores	2	0.2046	0.3189
Invertivores	50	−0.2691	0.2561
Piscivores	2	0.0388	0.4691
Lutjanidae			
Piscivores	12	−0.5040	0.0858
Pomacentridae			
Mixed	58	0.4045	0.1664
Corallivore	1	−0.1228	0.4077
Herbivores	4	0.4870	0.1221
Omnivores	27	0.2224	0.2865
Planktivores	26	0.4547	0.1353
Scaridae			
Herbivores	22	−0.3423	0.2063

*, $P < 0.05$.

transmission, our data suggest a singular mechanism by which high fish diversity can mitigate disease spread in corals. We propose that fishing for highly desired species releases nontargeted fishes, such as corallivorous chaetodontids, from predation- or competition-limited population densities which, in turn, exacerbates coral disease spread in a host-threshold, density-dependent manner. Corallivorous fishes feed preferentially on physically damaged, stressed, or diseased coral tissue (36–38) and increase the rate at which disease spreads from infected to noninfected corals in aquaria (38). These behaviors could transmit coral pathogens via feeding, although spread via fecal contamination (39, 40) and water-borne contamination of feeding-related entry wounds (41) have also been proposed. Regardless, these observations, coupled with the results presented here, raise concern that rarely harvested coral-associated fishes, particularly the common and charismatic butterflyfishes, may be complicit in the demise of reef corals on poorly managed reefs.

Table 2. Partial Mantel regressions of prevalence of coral diseases at sites on the Great Barrier Reef with >40% coral cover on the 10 most abundant taxonomic groups of fishes while holding intersite distances and the hard-coral percent cover constant

Taxon	Trophic group	*r*	*P*
Acanthuridae	Herbivores	0.1494	0.2816
Chaetodontidae	Mixed	0.5130	0.0081*
Lethrinidae	Invertivores	−0.0673	0.4487
Lutjanidae	Piscivores	0.0275	0.4824
Scaridae	Herbivores	−0.0775	0.4021
Serranidae	Piscivores	−0.1893	0.2550
Siganidae	Herbivores	−0.3048	0.1094
Zanclidae	Invertivores	0.2500	0.1558

*, $P < 0.05$.

Our results demonstrate a clear link between functionally diverse, species-rich fish communities and low disease prevalence. We acknowledge that the effect of fishing on coral health is complex and that the mechanisms by which this occurs require further investigation. Nevertheless, we found that effectively managed MPAs had a powerful effect on reducing coral disease by maintaining functionally diverse fish assemblages. Of equal importance, we showed that even among fished reefs, those with greater fish diversity were, on average, less diseased. This indicates that even a moderate reduction in fishing pressure can ameliorate coral disease. Thus, while protecting reefs from overharvesting confers numerous previously recognized benefits (7, 42), it also presents a promising approach to managing coral disease.

Methods

We surveyed seven MPAs and seven adjacent fished reefs in the central Philippines (refer to Fig. S1 and Table S1 for site locations). All MPAs had an active management plan, including a total ban on harvesting, for at least the previous 5 years and had little to no poaching. We avoided reefs obviously impacted by poor water quality and past destructive fishing practices because such stressors may influence disease prevalence regardless of management effectiveness. Surveys took place in May and June 2006 and were conducted sufficiently distant from the MPA boundary (≥50 m) to minimize edge effects.

At each site, we laid three to six 20-m × 1-m belt transects, the number of transects being proportional to the areal extent of the MPA. To minimize variation in estimates of coral cover between sites, we surveyed the reef crest/slope between 3-m and 7-m depths. Within each transect, all coral colonies >2 cm in diameter were identified to genus, counted, and scored for presence/absence of previously described Indo-Pacific diseases (43–45). Disease prevalence was expressed as percent of diseased colonies per transect and averaged over site. Percentages of live hard coral, coral rubble, and dead standing coral were determined by using the line-intercept method (46). An index of physical damage was calculated per transect as (coral rubble + dead standing coral)/(coral rubble + dead standing coral + live hard coral) and averaged over a site. Fish species abundance was quantified by a single observer along three 50-m × 10-m belt transects at each site within the same reef zones as those surveyed for coral.

We used two-way ANOVAs to look for differences between MPAs and fished sites in disease prevalence, density of *Porites*, live hard-coral colony counts, and abundance of fish taxa. Site and management status (i.e., protected vs. fished) were used as factors. Data were transformed to meet the assumptions of ANOVA where necessary. When data did not meet these assumptions, nonparametric analogues were used. To account for spatial autocorrelation, we performed all regressions by using Mantel and partial Mantel tests, assessing significance via randomization (47, 48). Intersite distances were calculated as the shortest over-water distance. Partial Mantels were performed by holding intersite distance constant and permuting the raw values (contra the residuals) as recommended in Legendre (49).

The functional diversity of fish assemblages was assessed at each site by using average taxonomic distinctness (28, 50), defined as the degree to which species in a sample are related taxonomically to each other by measuring the average path length between every pair of species through a taxonomic tree.

This measure is independent of sample size, which circumvents a problem confounding many of the other more commonly used measures of diversity. It is more sensitive to disturbance effects than traditional indices, such as Shannon diversity (51). The master list of coral reef fishes of the Philippines was compiled from a query to FishBase (www.fishbase.org/search.php).

To assess the generality of the results from the Philippines, we also performed partial Mantel regressions on a comparable dataset from the GBR, which provides publicly available information on population trends of corals and reef fishes for 93 reefs spanning 2,000 km of the GBR (34). We could not, however, compare fish taxonomic distinctness or trophic status within taxa between regions because the GBR data are grouped to the family level. Further, recent rezoning of no-take areas within the GBR precluded our making comparisons between different management regimes (52). We used the most current dataset for each reef (2006 or 2007), which included mean site abundance for 10 fish families and diseased coral colony counts.

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Corrections

MEDICAL SCIENCES

Correction for "Adenoviral transfer of HIF-1α enhances vascular responses to critical limb ischemia in diabetic mice," by Kakali Sarkar, Karen Fox-Talbot, Charles Steenbergen, Marta Bosch-Marcé, and Gregg L. Semenza, which appeared in issue 44, November 3, 2009, of *Proc Natl Acad Sci USA* (106:18769–18774; first published October 19, 2009; 10.1073/pnas.0910561106).

The authors note that, in Fig. 3*K*, the labels "AdCA5" and "AdLacZ" in the legend of the bar graph are reversed. The corrected figure and its legend appear below.

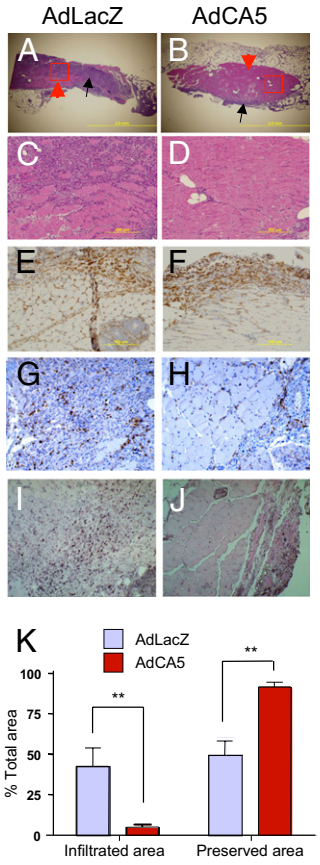

Fig. 3. Histological analysis of ischemic adductor muscles from diabetic mice after therapy. (*A–D*) Hematoxylin-eosin staining of tissue sections on day 7 after femoral artery ligation and treatment with AdLacZ (*A*) or AdCA5 (*B*) (original magnification, 40×). Black arrow indicates area infiltrated with inflammatory cells and red arrow indicates area with preservation of normal muscle tissue histology. Boxed areas are shown at higher power (*C* and *D*; original magnification, 200×). (*E–J*) Immunohistochemical staining of infiltrated areas for macrophages (*E* and *F*), lymphocytes (*G* and *H*), and neutrophils (*I* and *J*) using F4/80, CD3 and myeloperoxidase antibodies, respectively (original magnification, 200×). (*K*) Quantification of infiltrated and preserved areas as percentage of total tissue area with mean ± SEM (n = 4 mice each) shown. **, $P < 0.01$ (Mann–Whitney test).

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ECOLOGY

Correction for "Functionally diverse reef-fish communities ameliorate coral disease," by Laurie J. Raymundo, Andrew R. Halford, Aileen P. Maypa, and Alexander M. Kerr, which appeared in issue 40, October 6, 2009, of *Proc Natl Acad Sci USA* (106:17067–17070; first published September 28, 2009; 10.1073/pnas.0900365106).

The authors note that, in the second sentence of the legend for Fig. 2, "Dashed lines represent 95% confidence intervals; closed circles, MPAs; open circles, fished sites" should instead appear as "Dashed lines represent 95% confidence intervals; closed circles, fished sites; open circles, MPAs." This error does not affect the conclusions of the article. The figure and its corrected legend appear below.

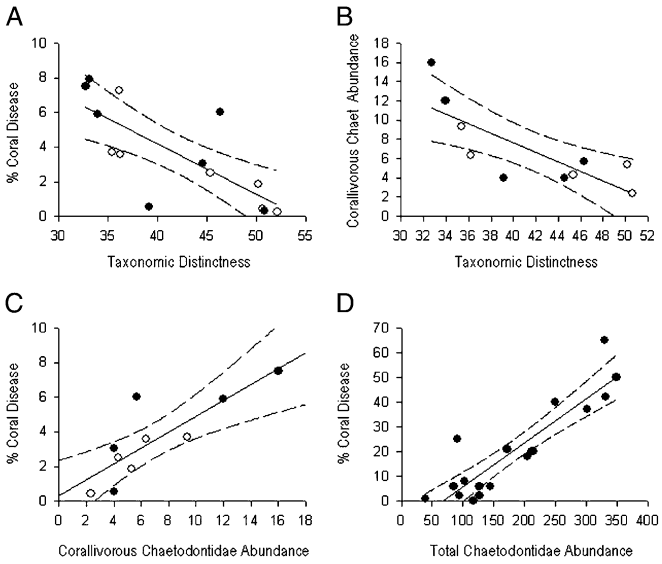

Fig. 2. Simple linear regressions on relationships between coral disease and fish diversity per reef site. Dashed lines represent 95% confidence intervals; closed circles, fished sites; open circles, MPAs. (*A–C*) Philippines. (*D*) GBR. (*A*) Average taxonomic distinctness of fish assemblages vs. mean coral disease prevalence, all sites. (*B*) Average taxonomic distinctness vs. chaetodontid abundance, sites with ≥50% live coral cover. (*C*) Chaetodontid abundance vs. mean coral disease prevalence, sites with ≥50% live coral cover. (*D*) Chaetodontid abundance vs. coral disease counts, sites with ≥40% live coral cover.

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Supporting Information

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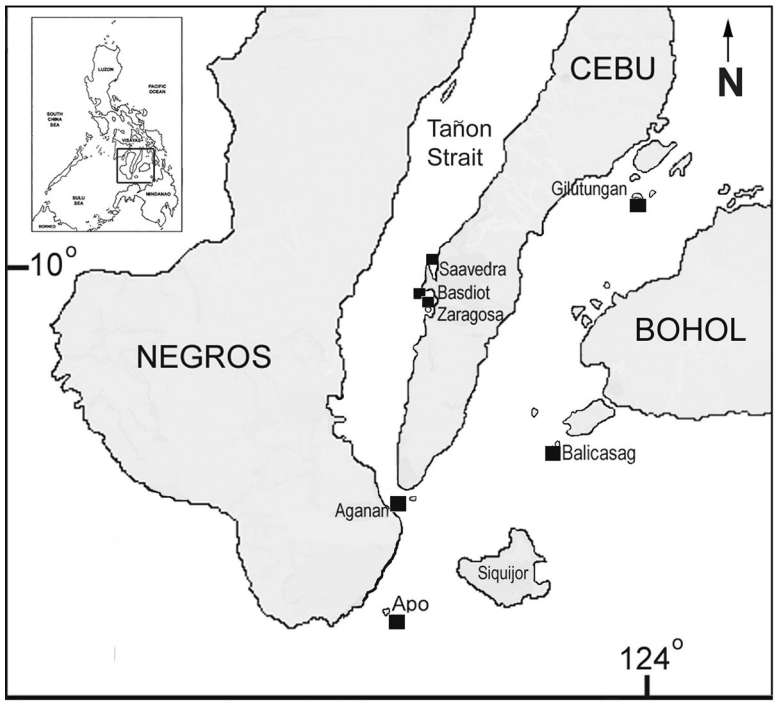

Fig. S1. Map of the Central Visayas, Philippines, showing locations of surveyed reefs. Reefs are marked with a ■.

Table S1. GPS locations of reefs and paired sites within reefs

Reef	Location MPA site	Location fished site
Balicasag	N 09.31.004, E 123.40.496	N 09.30.573, E 123.40.486
Gilutungan	N 10.12.376, E 123.59.076	N 10.12.361, E 123.59.118
Basdiot	N 09.56.267, E 123.22.126	N 09.56.278, E 123.22.121
Saavedra	N 09.59.338, E 123.23.060	N 09.59.349, E 123.23.056
Zaragosa	N 09.53.378, E 123.22.529	N 09.53.375, E 123.22.546
Apo Island	N 09.04.306, E.123.16.189	N 09.04.314, E.123.16.233
Aganan	N 09.20.090, E 123.18.407	N 09.20.026, E 123.18.407