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 implicated 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.

Results and Discussion

Factors varying at spatial scales less than that of the study region (circa 12,000 km2) 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 (F ± 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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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. 2A). 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 $\geq 50\%$, a single significant and positive relationship was revealed between disease prevalence and the butterflyfish family Chaetodontidae ($r = 0.680$, $P = 0.028$; Fig. 2B 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. 2C), 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 ($r = 0.513$, $P = 0.008$; Fig. 2D and Table 2) at sites with moderate to high coral cover ($\geq 40\%$) 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
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 affected 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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