

Coral diseases on Philippine reefs: genus *Porites* is a dominant host

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ABSTRACT: While it is generally assumed that Indo-Pacific reefs are not widely affected by diseases, limited data suggest a number of diseases and syndromes that appear to differ from those currently under study in the Caribbean. This report presents the results of a baseline survey of coral diseases in 2 regions in the Philippines: the Central Visayas and the Lingayen Gulf. Mean prevalence for all diseases observed was $8.3 \pm 1.2\%$ (mean \pm SE; $n = 8$ reefs), with Central Visayas reefs showing higher disease prevalence ($11.6 \pm 2.8\%$; $n = 4$ reefs) than those of Lingayen Gulf ($5.1 \pm 1.4\%$; $n = 4$ reefs). Five diseases and syndromes were described; 3 of these—*Porites* ulcerative white spot disease (PUWS) (prevalence = $8.96 \pm 2.2\%$), tumors (prevalence = $1.0 \pm 0.5\%$) and pigmentation response (prevalence = $0.5 \pm 0.2\%$)—occurred frequently in both regions and targeted the genus *Porites*. Correlation between disease prevalence and number of *Porites* colonies was fairly strong ($r^2 = 43.4$), though not significant, and no correlation was seen between prevalence and either the amount or diversity of hard coral. *Porites* is a major reef-builder in the Indo-Pacific comprising 30% of hard coral colonies on our surveyed reefs, and is generally thought to be a hardy, long-lived genus. Diseases targeting this robust group present an as yet unquantified risk to Philippine reefs and could result in major changes in reef structure.

KEY WORDS: Coral disease · Philippines · *Porites* · Disease prevalence · Host range · Species diversity

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INTRODUCTION

Reef scientists today must protect and manage coral reefs that continue to be subjected to a variety of assaults including over-harvesting, destructive fishing, eutrophication and siltation. Marine diseases have recently been added to this list of impacts. Concern is growing regarding the effects of infectious diseases on reef community structure as a number of studies have suggested that impacts can be considerable. Aronson & Precht (1997) and Aronson et al. (1998) demonstrated that recent changes in reef structure brought about by white band disease are unprecedented for the past 3800 yr. The well-documented epizootic that killed the major herbivore *Diadema antillarum* provided compelling evidence of the structuring force that a disease can impose, par-

ticularly when combined with anthropogenic stressors such as overfishing (Hughes 1994). Data suggest that the number of diseases affecting marine species is increasing via both host and range shifts (Harvell et al. 1999, Ward & Lafferty 2004). Climate change is predicted by some to increase host susceptibility and host range, pathogen survival and disease transmission (Ritchie et al. 2001, Harvell et al. 2002), while others predict fewer impacts with density-dependent modes of transmission as host populations decline (Lafferty & Holt 2003). Clearly, the effects of environmental factors are complex and synergistic and as yet are poorly understood.

The Western Atlantic is considered a hotspot for coral reef diseases. Few reefs within this region remain unaffected (Richardson 1998, Bruckner 2002). Consequently, this region has been the focus of disease-

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related research and much of our current understanding of disease etiology, causal agents and impacts to reef communities comes from research on these reef systems. However, outbreaks are not limited to the Caribbean (Antonius 1985, 1988, Alder & Braley 1988, Coles 1994, Littler & Littler 1996) and the increase in diseases may be a world-wide phenomenon that is unquantified and under-reported. In addition, while both Black Band and White Band diseases have a circumglobal distribution, host species show much variation between oceans (Gladfelter 1982, Antonius 1985, 1988, Coles 1994). However, most diseases appear to have a more limited geographic range. Aspergillosis is widely dispersed throughout the Caribbean (Nagelkerken et al. 1997) but has not been reported elsewhere. *Porites* ulcerative white spot disease (PUWS) affects reefs in the Philippines and possibly elsewhere in the Indo-Pacific (Raymundo et al. 2003), but has not been described in Caribbean poritids. However, the signs of PUWS are similar to another syndrome ('white spot syndrome') reported in the Caribbean affecting massive corals (Global Coral Reef Alliance web page; cited in Bruckner 2002). These observations raise numerous questions that can guide future research. How do host range and disease etiology differ between oceans? Which diseases reported from widely dispersed geographical regions are caused by the same pathogen? How do differences in reef diversity and human impacts affect the severity of disease outbreaks?

The coral reefs of the Indo-Pacific are the most diverse in the world. A total of 581 species of scleractinian corals have been reported from this region. In contrast, the Western Atlantic has 62 reported species (Veron 2000). This diversity could be predicted to lower the spread of infectious diseases that are limited in host range as the probability of pathogens encountering a host would be reduced in a high-diversity community. Alternatively, more diverse reefs could harbor a greater diversity of pathogens. Indo-Pacific reefs show the highest global diversity in most taxonomic groups (Spalding et al. 2001), although microbial community diversity has not been documented. Information regarding diseases in the Indo-Pacific region is very limited and little quantitative work has been accomplished. Antonius (1985) reported the first survey of Philippine reefs, finding 20 species in 8 families with white band infections and 2 species with both white band and black band diseases. No other diseases were described in this first survey. Several syndromes and anomalies from the region have been described qualitatively (see Bruckner 2002 and Sutherland et al. 2004 for summaries) and species exhibiting these disease signs have been identified. However, the majority of these

reports describe single point-in-time observations and should serve only as a starting point for a more in-depth investigation.

A few diseases have been documented quantitatively. Preliminary work on a new disease, PUWS, quantified its host range, prevalence, incidence and seasonality in the central Philippines (Raymundo et al. 2003). Pink line syndrome (here referred to as pigmentation response) is widespread throughout the Indo-Pacific and thought to be caused by the cyanobacterium *Phormidium valderianum* (Ravindran & Raghukumar 2002), but the pathogen has not yet been isolated from diseased tissues, nor has it satisfied Koch's postulates. A transfection study carried out in laboratory aquaria suggested that the condition is not infectious (Azcona 2005). Two bacterial pathogens (*Vibrio shiloi*, Kushmaro et al. 1998; and *V. coralliilyticus*, Ben-Haim et al. 2003) have been found to cause bleaching in the corals *Oculina patagonica* and *Pocillopora damicornis*, respectively. Finally, skeletal eroding band affects a number of coral genera in Papua New Guinea, Australia, the Red Sea and the Indian Ocean, and has been found to be caused by the boring activities of the ciliate *Halofolliculina corallasia* (Antonius & Lipscomb 2000).

This paper reports the results of a recent baseline survey to identify diseases affecting hermatypic corals in the Philippines. Philippine reefs are among the most impacted in the world, with an estimated 98% of these reefs suggested to be at medium to high risk from threats ranging from destructive fishing to eutrophication (Burke et al. 2002). Two regions differing in geographic position within the archipelago were selected for preliminary rapid assessment surveys (Fig. 1). The Lingayen Gulf is a large bay on the western margin of Luzon Island that opens to the South China Sea. In contrast, the Central Visayas region is in the central part of the country, which has local water circulation within the semi-enclosed Mindanao Sea and was the site for the original surveys by Antonius (1985). Both regions experience intense fishing pressure, which can include illegal methods such as dynamite and cyanide. Erosion, agricultural runoff and eutrophication from sewage also impact local areas in both regions (Udarbe-Walker & Magdaong 2003, authors' pers. obs.). Marine protected areas (MPAs) have been established within each region. These provided sampling sites for what was considered the best coral cover/reef health in the area. The objectives of this survey were to quantify diseases that were previously recorded within the country, to develop a species list of hosts affected by each disease observed and to describe any apparently 'new' syndromes or potential diseases. This baseline information will be used for future in-depth studies targeting specific diseases or syndromes, to

elucidate pathogens, disease progression and impacts to the reef community.

MATERIALS AND METHODS

Prior to surveys, reefs were selected in each region that represented contrasting states of disturbance: relatively undisturbed vs. impacted by human activity. 'Relatively undisturbed' is defined here as a reef actively and continually protected from harvesting (i.e. MPAs), high hard coral cover relative to dead coral/rubble, with clear water and no obvious nearby sources of nutrient or silt input. At each site, both shallow and deep transects were laid at approximately equal distances apart (distance between transects was estimated using the same number of fin kicks between each transect; shallow transects ranged from 2 to 4 m; deep transects ranged from 5 to 10 m), except for the Agan-an and Looc sites. These 2 sites represent different sections of an extensive fringing reef along Dumaguete City, Negros Island. The reef is very shallow and 2 sampling depths were not possible, so 2 different sections were sampled to maintain the same replication as on other reefs. At each depth, three 2×20 m belt transects were laid. All diseased colonies within this belt were noted and the number of diseased vs. healthy colonies per species was counted so that disease prevalence could be calculated. In spite of the very high diversity of most reefs, this was possible largely because *Porites* appeared to be the only genus that was regularly observed to be affected by disease. Therefore, we concentrated our efforts to quantify healthy vs. diseased colonies on this genus alone. In addition, the total number of *Porites* colonies (diseased and healthy) and the total number of hard coral colonies were counted for a 2×10 m segment of each transect, to calculate the percent contribution of the genus *Porites* to the hard coral community at each site and to determine overall prevalence. Our initial efforts focused on diseases previously documented from the country: black and white band diseases and PUWS. In addition, pigmentation response and skeletal anomalies, hereafter referred to as tumors, were also observed. We define a tumor as 'an abnormal tissue proliferation', as described in Sutherland et al. (2004), which in corals also involves the underlying skeleton. This appears to be the most appropriate term to apply for the distinctive gross colony surface irregularities we regularly observed on massive *Porites*. Aside from these diseases, new signs of disease, either biotic or abiotic in origin, were described and photographed when encountered and the species displaying them were identified.

Two other types of data were collected within these belt transects. The Line-Intercept method was used to

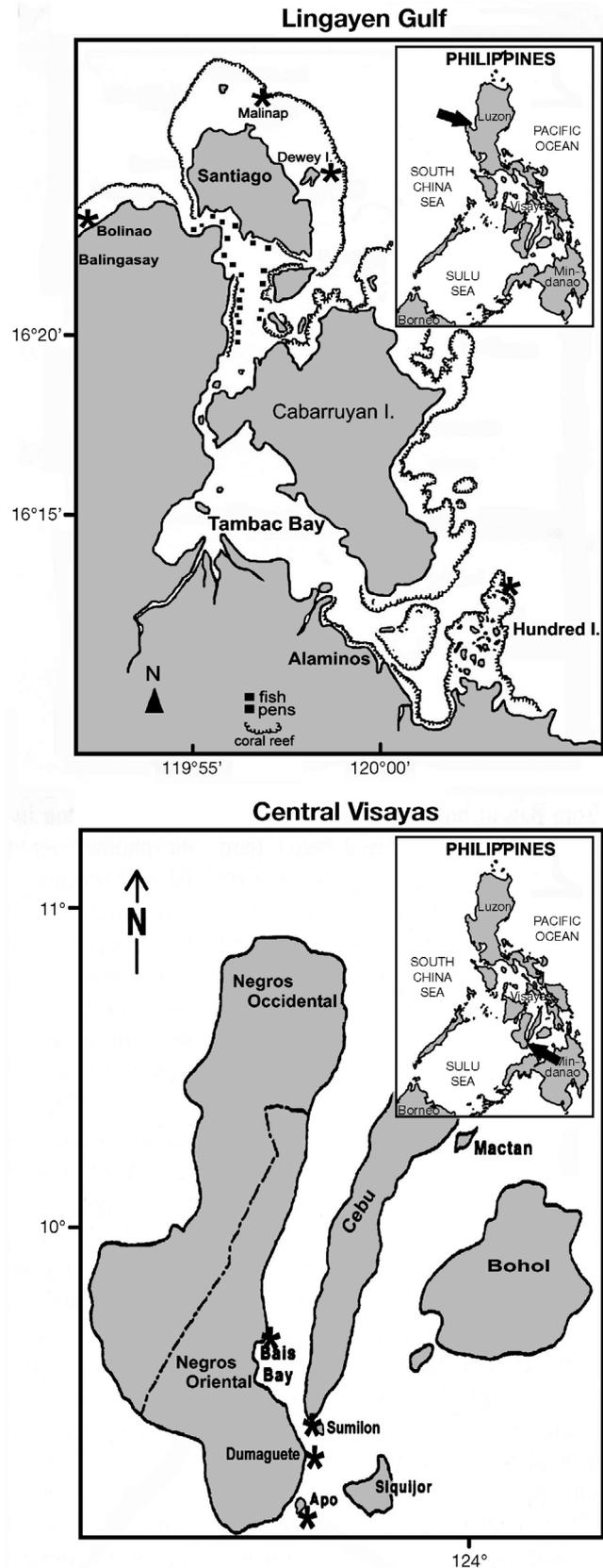


Fig. 1. Map of the Philippines showing the respective study sites. *: individual reefs surveyed

obtain a detailed description of substrate composition and the following substrate categories were noted: live hard coral, soft coral, recently killed hard coral, weathered rock with turf algae, rubble, sand, silt, canopy-forming algae and other encrusting organisms (such as sponges, ascidians, gorgonians). Additional information on poaching, pollution sources and other human stressors was obtained by interviewing local fishers and managers. Hard coral diversity was obtained along five 2 m segments bisecting each transect at 5 m intervals. Species were identified *in situ* where possible, or small pieces were collected for identification in the laboratory when necessary. Identification of specimens was based on Veron (2000) and Wallace (1999). Species diversity was determined using Simpson's index of concentration by combining species lists for all transects within a reef. Simpson's index is useful for such sampling as it does not depend on sample size or require a specific type of abundance distribution (Rosenzweig 1995). In order to determine if the number of segments we sampled per reef ($n = 30$) was a sufficient representation of diversity, we counted the number of new species added per segment. In all reefs, the number of new species approached zero prior to the thirtieth segment, so we assumed that our sample number was adequate.

Disease prevalence was calculated as follows (see explanation above):

$$\frac{\text{number of diseased colonies per transect}}{\text{total number of colonies per transect}} \times 100$$

This was computed per depth and per reef for all diseases, and per species per reef for PUWS. To examine a possible connection between disease prevalence and *Porites* host abundance, the relative abundance of each species was calculated per reef as the total number of colonies per *Porites* species/total number of all colonies and this also allowed a calculation of *Porites* spp. abundance relative to all other species. A 2-way ANOVA was used to detect differences in prevalence between the 2 regions (Lingayen Gulf and Central Visayas) and the 2 depths surveyed (shallow and deep). Regression analysis was used to examine connections between disease prevalence and live hard coral cover, hard coral diversity and *Porites* spp. abundance.

RESULTS

A summary of the survey sites is presented in Table 1. Most sites in both regions had reduced water quality from sewage, silt and runoff, and were heavily fished. Information regarding the use of cyanide was obtained from interviews with local fishers. The Lingayen Gulf (LG) reefs, in particular, were stressed from continual nutrient input and particulate material from numerous fish cages that lined the coast. Balingasay Marine Reserve was the only reef unaffected by these fish cages, as it was located around the northern point of the gulf, facing the South China Sea (Fig. 1). In the Central Visayas (CV), North Bais Bay showed the

Table 1. Site descriptions of reefs surveyed for disease. LHC = live hard coral; RU = rubble; DC = dead coral. Percent substrate composition values are mean \pm SE. Simpson's index values close to 0 signify higher diversity and those approaching 1 signify lower diversity

Survey site	Simpson's index	Percent substrate composition			Possible stressors
		LHC	RU	DC	
Central Visayas					
Apo Is. ^a	0.179	37.90 \pm 16.4	3.13 \pm 1.9	2.30 \pm 0.5	Tourist-diving coral breakage (minimal)
North Bais Bay	0.022	37.55 \pm 10.4	28.85 \pm 8.8	14.0 \pm 4.1	Agricultural runoff, cyanide, silt, overfishing
Agan-an/Looc	0.074	45.13 \pm 6.74	2.88 \pm 2.9	11.83 \pm 4.4	Sewage, overfishing, cyanide fishing, silt
Sumilon Is. ^a	0.125	31.26 \pm 7.4	5.48 \pm 4.6	2.09 \pm 2.1	Anchor damage, tourist diving, poaching
Lingayen Gulf					
Balingasay ^a	0.067	31.88 \pm 7.38	2.92 \pm 1.5	3.17 \pm 1.1	Seasonal heavy waves
Malilnep	0.151	33.71 \pm 3.8	9.17 \pm 4.8	20.83 \pm 6.5	Silt, overfishing, sewage, cyanide
Victory, Dewey Is. ^a	0.033	38.13 \pm 9.7	6.29 \pm 1.9	6.33 \pm 3.8	Silt, overfishing, sewage, cyanide
Governor Is.	0.069	42.42 \pm 4.1	11.29 \pm 4.3	9.67 \pm 2.0	Dynamite fishing, silt, cyanide, overfishing
^a Marine protected area					

highest hard coral diversity and high live hard coral cover, but also high rubble and dead coral values. This reef receives agricultural and river runoff and is known to contain large amounts of fertilizers, pesticides, effluent from a sugar processing plant and silt. Both the best-managed marine reserves, Apo Island (CV) and Balingasay (LG), had high water quality, with lower coral diversity and the lowest values for dead coral and rubble. Although our original intention was to select an equal number of sites that varied distinctly in water quality and overall reef health (i.e. healthy vs. impacted or degraded reefs), Apo Island and Balingasay appeared to be the only sites that truly fit the description of reefs with minimal impacts from human activities. The other marine reserves, Sumilon Is. (CV) and Victory (LG), showed a very depauperate and sparse fish population, suggesting some poaching in spite of protection. Victory, in particular, was extremely silty, but as it was a shallow, lagoonal platform reef with a high percentage of apparently healthy faviids and other massives, we suspected that the reef had developed under somewhat silty conditions. The pre-existing silty condition has recently been greatly exacerbated, however, from the nearby fish cages.

We identified a number of diseases and syndromes present on our surveyed reefs; these are described in Table 2. Fig. 2 presents photographs of each disease. Mean total disease prevalence for all diseases observed on all reefs was $8.28 \pm 1.16\%$ (mean \pm SE). Our surveys revealed no confirmed infections of either white band or black band diseases previously recorded by Antonius (1985). PUWS, tumors and pigmentation response were the most commonly observed conditions and affected the genus *Porites* almost exclusively.

The present survey expanded the previously recorded host range of PUWS (Raymundo et al. 2003). A total of 10 branching *Porites* species were infected with PUWS (Table 2) compared with the 6 species previously listed as susceptible. The massive species *P. lobata*, *P. lutea*, *P. australiensis*, *P. solida*, and *P. mayeri* were identified in the laboratory from our transects,

Table 2. Description of diseases and syndromes affecting corals in the Philippines

Disease/Syndrome	Gross description	Host species
<i>Porites</i> ulcerative white spot disease (PUWS)	Small (3 to 5 mm) white lesions found either singly or in small clusters. Lesions may contain bleached tissue which either progresses to tissue necrosis and death, or polyp recovery. Advanced infections involve lesion coalescence and spreading; dead skeleton is colonized by algae. Colony death can occur within 1 yr. Commonly found	Massive <i>Porites</i> <i>P. cylindrica</i> , <i>P. attenuata</i> , <i>P. nigrescens</i> , <i>P. tuberculosa</i> , <i>P. horizontallata</i> , <i>P. annae</i> , <i>P. latistella</i> <i>P. lichen</i> , <i>P. rus</i> , <i>P. sillimaniana</i> , <i>Goniastrea minuta</i> , <i>Favia stelligera</i>
Tumors	Irregular patches of skeleton either raised or depressed relative to surrounding healthy tissue. Surface texture distinct from healthy tissue, with distinct margin between the two. Tumor tissue discolored. Microscopic examination showed deformed corallites and skeletal irregularities. Fairly commonly found	Massive <i>Porites</i>
Pigmentation response	Found along margins of dead or necrotic tissue, colonized by turf algae. Margins highly pigmented with pink, blue or yellow band 2 to 4 mm wide produced by host colony. Pink exuded material may accumulate along the innermost lesion/dead skeleton border, accompanied by thickening of walls of affected corallites. Fairly commonly found	Massive <i>Porites</i>
Necrotic syndrome	Irregular patches of necrotic tissue with mucous discharge. Within the patch, the tissue is bleached and discolored. Patches bordered by 2 mm chalky, white band. Patches found near colony margin, around crevice in colony, or facing an infected neighboring colony. Commonly found on 1 reef	Massive <i>Porites</i>
White syndrome	White band of exposed skeleton from recently killed tissue. Tissue death appears to begin in center of foliose colony, spreading toward margin; older exposed skeleton is algae-colonized. Rarely found	<i>Montipora informis</i>

but we were unable to verify *in situ* which of these species were infected, due to difficulties with both field identification and sampling. In addition, 2 faviid species (*Goniastrea minuta* and *Favia stelligera*) were observed to exhibit PUWS-like lesions on 2 different reefs (one in each region), suggesting that host range is not limited to the genus *Porites*. Mean prevalence of

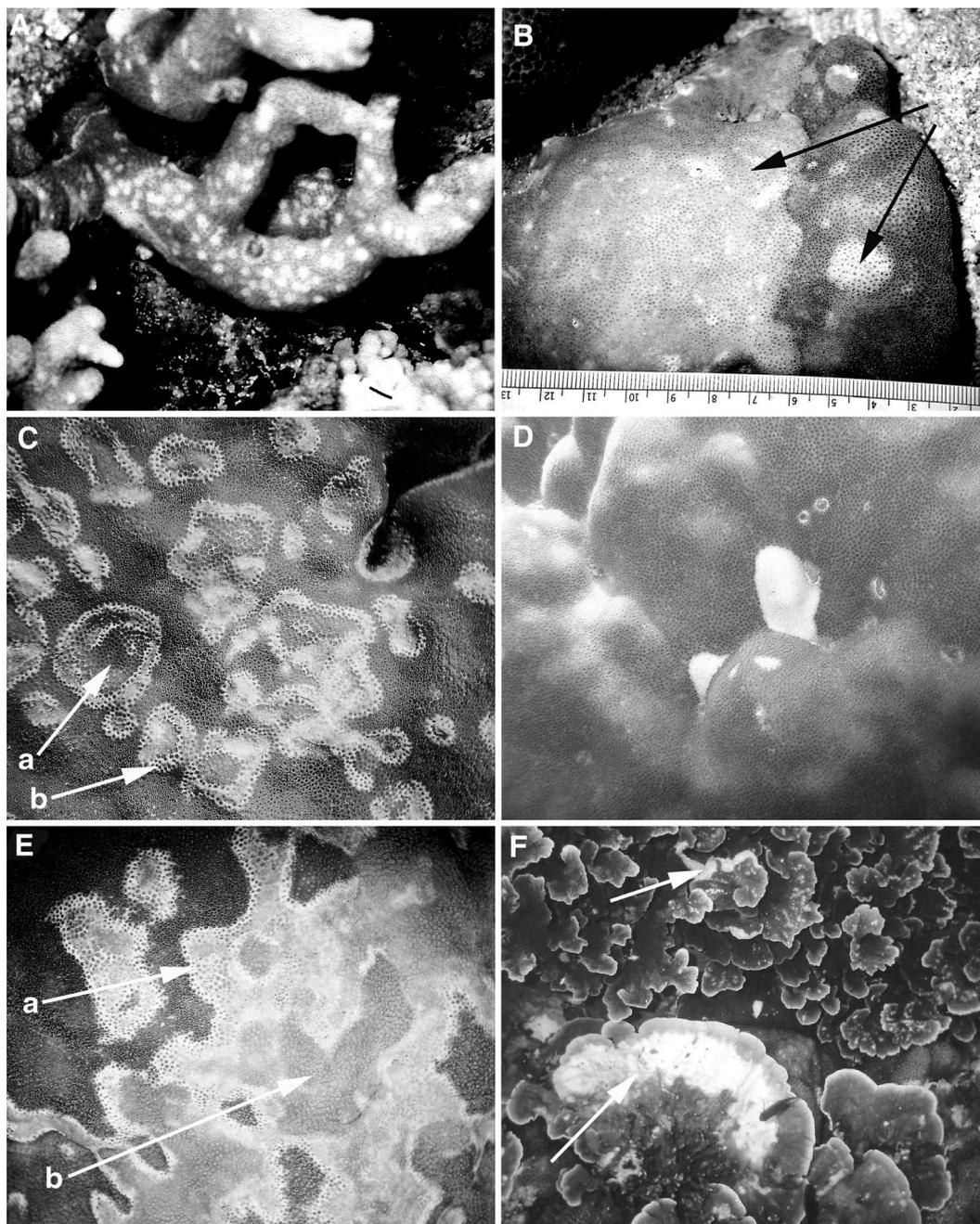


Fig. 2. Diseases affecting Philippine corals. (A) *Porites* ulcerative white spot disease on *Porites attenuata*. (B) Tumors; arrows at affected portions of massive *Porites* colony. (C) Pigmentation response on massive *Porites*; (a) arrow pointing to highly pigmented tissue, (b) arrow pointing to increased skeletalization around affected corallites. (D) White blotch on massive *Porites*. (E) Necrotic syndrome on massive *Porites*; (a) arrow pointing to white, chalky band, (b) arrow pointing to necrotic tissue. (F) White syndrome; arrows pointing to 2 affected patches on *Montipora informis*. Photo credits: L. Raymundo, L. Kaczmarzky

PUWS within the genus *Porites* on surveyed reefs was $8.96 \pm 2.2\%$.

Tumors were found in both regions, possibly affecting all massive *Porites* species we encountered, and mean prevalence within the genus on all reefs was $1.01 \pm 0.5\%$. Colonies affected by both PUWS and

tumors were occasionally observed. Pigmentation response is an ill-defined condition, commonly seen in massive *Porites* and possibly associated with the cyanobacterium *Phormidium valderianum* (referred to as pink line syndrome by Ravindran & Raghukumar 2002). It is characterized by irregular patches of dead

skeleton on massive *Porites*, bordered by a highly pigmented margin. Such margins may also border cavities created by certain boring organisms and competitors. Pigmentation response has been described as a generalized stress response of the host coral to what appears to be many varied agents, and therefore would be considered an example of an abiotic disease. The response may vary in intensity and color; we regularly observed margins of pink, blue and yellow on different colonies. However, a distinctive feature of this response is the production (apparently from the coral itself) of a bright pink material along a lesion border that may be accompanied by increased skeletalization of affected corallites, which causes a visible thickening of corallite walls (Fig. 2). Although few colonies per reef manifested these patches, we observed it regularly on reefs in both regions (mean prevalence within the genus on all reefs was $0.5 \pm 0.2\%$). The prevalence of these 3 diseases is presented in Fig. 3.

On a single reef in LG, we observed yet another condition affecting massive *Porites*. 'Necrotic Syndrome' was observed on Governor Is. (Table 2) affecting 25.3% of the large massive *Porites* (large massives comprised approximately 38% of total live coral cover on this reef). Extensive necrotic patches occurred along colony margins, crevices in the colony surface or on colony surfaces facing a similar patch on a neighboring colony. These patches were characterized by discoloration and abundant mucous production, and the presence of a chalky, white material in a band between healthy and diseased tissue (Fig. 2). A similar condition was subsequently observed on 3 colonies in the Agan-an reef the following year.

A suite of disease signs consistent with those of white syndrome reported from the Great Barrier Reef (Willis et al. 2004) affected colonies of *Montipora informis* at the Apo site (Fig. 2). This syndrome was not quantified, as the reef portion where it was observed was outside our transected area, but colonies displaying disease signs have been observed on several different occasions. Therefore, we mention it in this report.

Between-reef differences in prevalence were significant (ANOVA; $F = 10.777$, $p < 0.0001$). Dumaguete and Sumilon Is. showed much higher prevalence than all other reefs; the 2 best protected marine reserves, Balingasay and Apo Is. showed very little disease (Fig. 4A,B). Prevalence was significantly lower on LG reefs ($5.09 \pm 1.4\%$), than on CV reefs ($11.6 \pm 2.79\%$; ANOVA; $F = 9.139$, $p = 0.0041$, $df = 1$). There was no

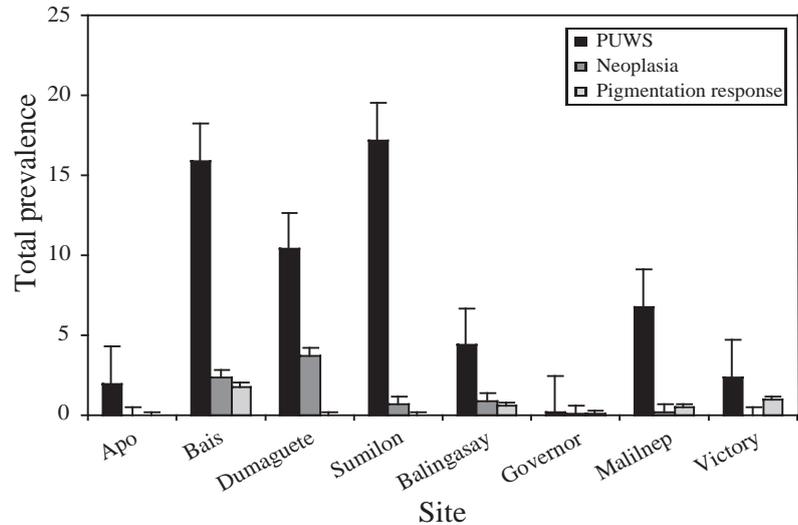


Fig. 3. Mean prevalence of the 3 most common diseases/syndromes observed on surveyed reefs. Mean \pm SE; $n = 3$ transects per depth, 2 depths per reef

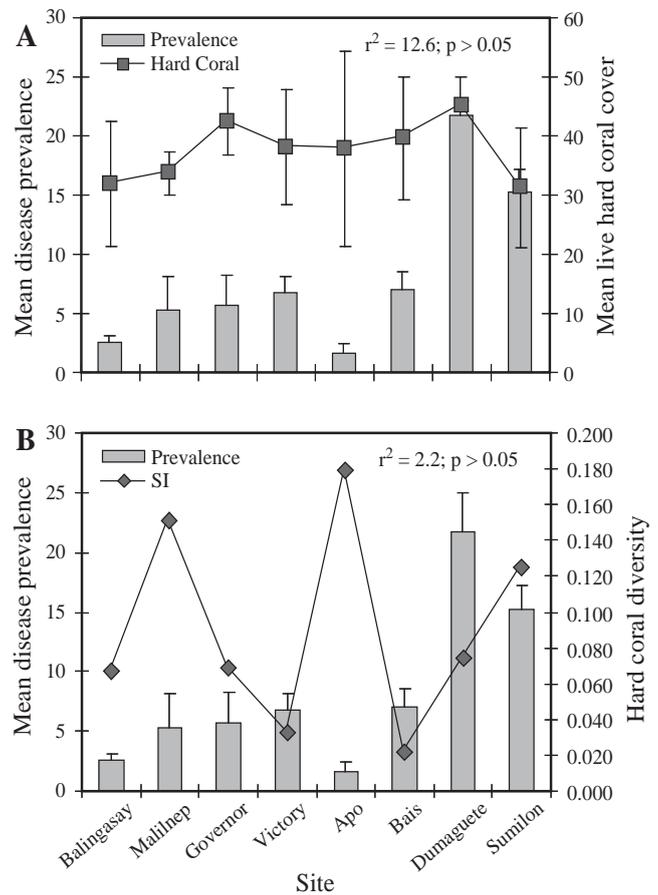


Fig. 4. (A) Mean total disease prevalence vs. mean hard coral cover per reef. (B) Mean total disease prevalence vs. total hard coral diversity per reef. ($N = 8$ reefs, 2 depths per reef; $n = 3$ transects per depth; mean \pm SE). Simpson's index of concentration provides a value between 0 and 1; the lower values signify higher diversity

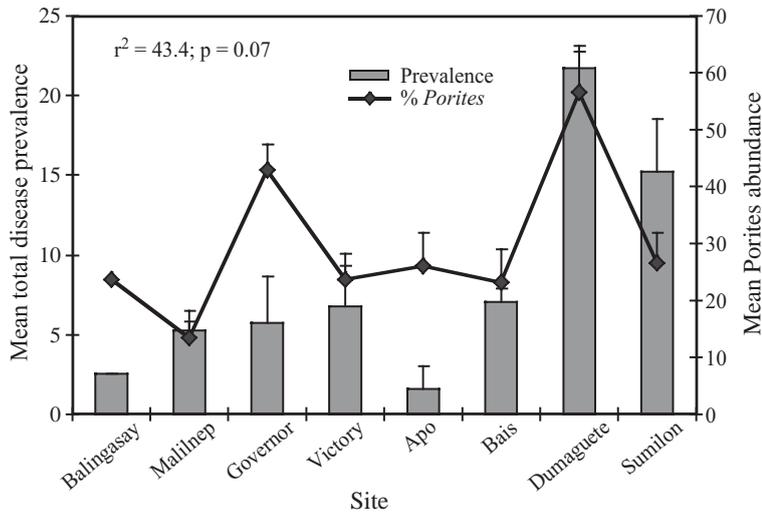


Fig. 5. Disease prevalence and *Porites* spp. abundance per reef. Prevalence calculated per reef as the number of diseased colonies per species/total number of colonies per species $\times 100$; relative abundance calculated per reef as the total number of colonies per species/total number of *Porites* spp. colonies $\times 100$. N = 8 reefs; Mean + SE

difference in prevalence between shallow and deep transects (ANOVA; $F = 2.157$, $p > 0.05$). It was not possible to do a statistical analysis of prevalence between 'good' and 'impacted' reefs as we had too few reefs that we considered to be relatively healthy.

The genus *Porites* was particularly vulnerable to all diseases we observed; indeed, 'white syndrome' was the only disease noted that did not affect the genus and only rarely was any other genus observed to be diseased. *Porites* is a major contributor to reef structure on most reefs, comprising a mean of $29.9 \pm 3.2\%$ (range =

4.3 to 68.5%) of the total hard coral colonies along our transects. As expected, given the apparent susceptibility of *Porites* to the observed diseases, there was a fairly strong positive correlation between disease prevalence and the abundance of *Porites* colonies per reef (Fig. 5; $r^2 = 43.4$, $p = 0.076$). Though data suggested a weak relationship between the amount of live coral cover and disease prevalence (Fig. 4A), this relationship was not statistically significant ($r^2 = 12.6$, $p = 0.389$). No relationship between total disease prevalence and hard coral diversity was suggested (Fig. 4B; $r^2 = 2.2$, $p = 0.727$). Prevalence of PUWS among species of *Porites* present on our transects was highly variable, and not strongly correlated with species abundance (Fig. 6; $r^2 = 14.7$, $p > 0.05$). However, we observed that the rarer species were almost never infected, while the 2 most common *Porites*, *P. cylindrica* and the massive *Porites* (*australiensis*, *lutea*, and *lobata*), did not show the highest disease prevalence.

P. deformis and *P. sillimaniani*, while relatively uncommon, showed the highest prevalence for PUWS.

DISCUSSION

Our preliminary surveys have provided baseline information on the status of coral disease affecting reefs in the Philippines, a country within the global center of coral reef biodiversity. Our data represent one of the first attempts to quantify disease impacts in

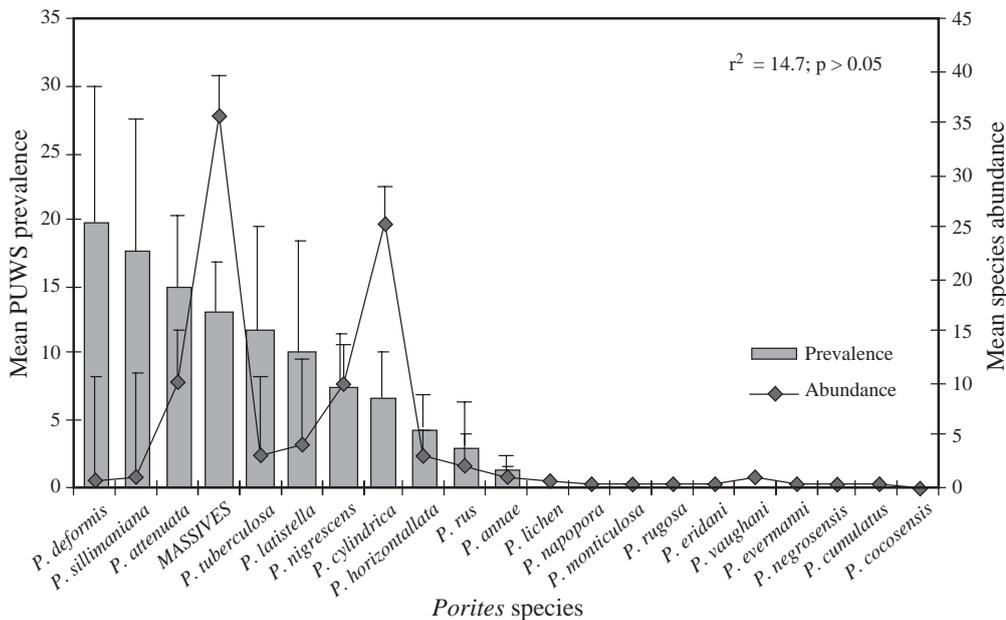


Fig. 6. Total disease prevalence and relative colony abundance for *Porites* spp. Prevalence calculated per reef as the number of diseased *Porites* colonies/total number of *Porites* $\times 100$; relative abundance calculated as the total number of *Porites* colonies/total number of colonies $\times 100$. N = 8 reefs; mean + SE

the Indo-Pacific. Our results verified the presence of one described disease, PUWS, outside the previously recorded range of the Central Visayas region and increased the host range of this disease from the previous report (Raymundo et al. 2003). Our surveys also suggested the presence of a number of other potential diseases: namely, tumors and white blotch, which have not been previously recorded or adequately described. White syndrome and pigmentation response have been reported elsewhere in the region and represent the first records from the Philippines.

Our results were comparable with previous work on PUWS prevalence in Central Visayas reefs (Raymundo et al. 2003). Surveys in 1998 and 1999 also showed highest prevalence in Sumilon and Bais, with no infected colonies in Apo and Agan-an. Mean PUWS prevalence in 1998 was 22% ($n = 8$ reefs in the Central Visayas; Raymundo et al. 2003), as opposed to our current value of 9% ($n = 8$ reefs in Central Visayas and Lingayen Gulf). However, past surveys were accomplished during the 1997–98 El Niño bleaching event, which may have contributed to the higher prevalence. Four years later, Apo Is. is still relatively unimpacted by disease. Differences in PUWS prevalence among the branching species of *Porites* suggests differences in susceptibility between species as prevalence was not strongly correlated with species abundance. Rarer species were almost never infected as would be predicted with an infectious disease. However, the 2 most abundant groups, massive *Porites* (*australiensis*, *lutea* and *lobata*) and *Porites cylindrica*, did not show the greatest PUWS prevalence. Prevalence was greatest among 2 relatively rare species, *P. deformis* and *P. sillimaniani*, though between-site variability was very high. This suggests that certain species may be more susceptible to the disease than others.

Although one of our objectives was to examine the effect of reef health on disease prevalence, the rarity of truly healthy reefs within our sampling regions made it impossible to conduct such a comparison. A larger-scale survey is needed to elucidate relationships between disease prevalence and reef health, water quality, hard coral cover and hard coral diversity. Another related question is the relationship between disease prevalence and the pattern of water circulation or flushing to which a particular reef is exposed. One theory put forth to explain high disease prevalence among Caribbean reefs is that the entire region is structured as a single large basin within which water locally circulates, affected by deposited African dust (Garrison et al. 2003). Thus, there appears to be a low correlation between reef health and disease outbreaks and apparently healthy reefs show high prevalence of disease (Goreau et al. 1998, Jordan-Dahlgren et al. 2005). The Philippines, being an archipelago with

internal local circulation patterns but bounded by the South China Sea and Pacific Ocean, provide a uniquely appropriate location for studying disease prevalence on reefs affected by local circulation vs. reefs which are continually or seasonally flushed by open ocean water. Our preliminary surveys, including the semi-enclosed reefs of the Central Visayas which are influenced by local circulation, and the more exposed and seasonally flushed reefs of Lingayen Gulf that drain into the South China Sea, suggested such an influence. Sea surface temperatures were similar for both regions in April 2003, averaging 27 to 29°C, but they had risen to 30 to 32°C by May in the Central Visayas, during our surveys (SEAWiFs images: <http://oceancolor.gsfc.nasa.gov>). Lingayen Gulf temperatures were not available for May, but they were generally cooler (29 to 31°C) along the outer margins of the country. Furthermore, all but one (Balingasay) of the Lingayen Gulf survey sites were heavily impacted by silt, agricultural runoff, eutrophication, blast fishing and overfishing, and yet consistently showed lower disease prevalence (5%) than did Central Visayas reefs (11%), which generally had higher water quality and were less impacted by human activity.

Both tumors and pigmentation response were seen in massive *Porites* within a broad range of size classes, but we could not determine the exact host range of each of these without sampling each colony underwater for later identification. 'pink line syndrome' (Ravindran & Raghukumar 2002) is actually a misnomer. We observed several different types of hyperpigmentation along the margins of variably sized dead patches of massive *Porites*, possibly reflecting pigmentation differences in the host coral. In addition, microscopic examination clearly revealed that the line is composed of coral skeleton, produced by the host coral; it is not a microbial band as is seen in the band diseases. It is widely assumed to be a generalized response to a diverse array of stressors, but the absence of such a response around predation scars, tumor patches and PUWS lesions suggests that not all damage to the colony surface results in such a response. It seems reasonable to speculate that unidentified chemical and/or microbial agent(s) may trigger this response or secondarily infect compromised tissue. Harvell (pers. comm.) suggests that such margins may represent a hypermelanization response to a pathogen, similar to what is observed in diseased gorgonian sea fans infected with Aspergillosis (Petes et al. 2003).

Threats to biodiversity are considered one of the major predicted impacts of new diseases and those infecting multiple species (Kim et al. 2003). We expected a relationship between diversity and disease prevalence, as high prevalence of a disease with multiple hosts can

lower diversity (Kuta & Richardson 2002). In contrast, a high-diversity reef could be predicted to be more resistant to an infectious disease, if host species are less abundant. However, we found only a very weak relationship between the two ($r^2 = 2.2$, $p > 0.05$), possibly because of our small sample size. Our diversity calculations did not vary widely between sites (Table 1, Fig. 4B), and the reef with the lowest diversity, Apo Is., had the healthiest reef community and highest water quality. Although water quality has historically been very high (i.e. no eutrophication, silt, agricultural runoff, etc.), the marine reserve at Apo is dominated by large colonies of the massive coral *Galaxea fascicularis*, which suffered high mortality during the 1998 El Niño bleaching event. Recovery of this species has been slow (Raymundo & Maypa 2003), and soft corals have colonized much of the open substrate. This accounts for the low diversity of hard corals, though *Porites* spp. are major components of the reef flat community and would be predicted to show disease signs if pathogens were present.

Porites is generally considered a hardy, slow-growing, robust genus. It survives well when transplanted (Clark & Edwards 1995), massive species show high recovery from bleaching events (Wilkinson 1998, 2000, Sheppard 1999), and it is a dominant component of Indo-Pacific reefs, comprising an average of 30% of live hard coral cover on our reefs. It is alarming, therefore, that all of the diseases we consistently observed appeared to target this genus, both those which suggest a biotic cause (i.e. a pathogen, such as PUWS) and those considered to be abiotic (i.e. caused by environmental stressors, such as pigmentation response). The potential for significant impacts on reef structure may be quite high, as this prominent genus, massive *Porites* in particular, is host to multiple diseases. Long-term monitoring is essential to determine what impacts these diseases will have on the future structure and diversity of these reefs. In-depth studies of disease pathogenesis, differential species susceptibility, host defensive capabilities, etiology and causative agents are also urgently needed.

This preliminary survey has documented that Indo-Pacific reefs are currently impacted by a number of diseases and syndromes. Detailed information about these syndromes is almost non-existent. PUWS is the only disease that has been quantified in Philippine reefs and much about the disease's etiology and pathogenesis is still unknown. As Philippine reefs are among the most impacted in the world, coral diseases pose further threats to these unique systems and a further challenge to reef management.

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