

Coral disease prevalence and coral health in the Wakatobi Marine Park, south-east Sulawesi, Indonesia

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This is the first study on coral diseases in the Wakatobi Marine National Park (WMNP), south-east Sulawesi. It aimed to provide baseline knowledge of coral disease prevalence and coral health in this remote region. Results indicate a low disease prevalence of 0.57% with only two known diseases occurring within the sampling unit, white syndrome (0.42%) and tumours (0.15%). They affected 15 taxonomic groups from a total of 32 taxonomic groups. The presence of black-band disease (BBD), skeletal eroding band (SEB) disease and *Porites* ulcerative white spot disease (PUWSD) was identified outside the study area. A large number of corals were affected by previously undescribed conditions (9.7% of colonies). The impact of lesions named as green spot, green band, pigmented spot, and flatworm infestation is not known and represents an important area for future studies.

INTRODUCTION

Wilkinson (2004) estimated that 20% of coral reefs globally have been destroyed and show no immediate prospects of recovery, 24% were under imminent risk of collapse through human pressures and a further 26% were under long-term threat of collapse. Eutrophication, increased sediment flow, over-exploitation of marine species and physical destruction by reef users are major causes of reef destruction (Sebens, 1994). Coral bleaching is another major factor contributing to the decline of coral reefs (Brown, 1997; Hoegh-Guldberg, 1999).

Coral diseases were first described in the early 1970s (Garrett & Ducklow, 1975; Antonius, 1977), but detailed quantitative studies were only conducted in the early 1990s on black-band disease (Edmunds, 1991; Kuta & Richardson, 1996). The rate of discovery of new diseases has increased dramatically with more than 29 syndromes now described (Green & Bruckner, 2000; Weil, 2004).

In this study coral diseases were identified by the presence and characteristics of lesions. Alternative approaches to identifying coral diseases are by etiology and causality.

Causative agents have been reported for only a few coral diseases. These are bleaching of *Oculina patagonica* by the bacterium *Vibrio shiloi* (Kushmaro et al., 1996, 1997), black-band disease by a microbial consortium (Carlton & Richardson, 1995), sea fan disease by the fungus *Aspergillus sydowii* (Smith et al., 1996; Geiser et al., 1998), white pox disease by the bacterium *Serratia marcescens* (Patterson et al., 2002), and the Caribbean coral white plague type II by the bacterium *Aurantimonas corallicida* (Denner et al., 2003), tumours caused by microalgae (Weil et al., 2006) and red-band disease caused by cyanobacteria (Weil et al., 2006).

Reservoirs have only been identified for black-band disease (Cooney et al., 2002) and aspergillosis (Shinn et al., 2000). The only known vector is the fireworm (*Hermodice carunculata*) that harbours the bleaching inducing bacterium *Vibrio shiloi* (Sussman et al., 2003).

Black-band disease (BBD), skeletal eroding band (SEB) and white-band disease (WBD) (Antonius, 1985) are found globally. Diseases found only in the Indo-Pacific are: yellow-band disease (YBD) (Korrubel & Riegl, 1998); the encysting stage of a trematode infecting *Porites compressa* in Hawaii (Aeby, 1991); *Porites* ulcerative white spot disease (PUWSD) (Raymundo et al., 2003); and atromentous necrosis (Bourne, personal communication 2006). These findings suggest that infectious pathogens are a common component of Indo-Pacific reef communities, and disease may be more important in the structuring of Indo-Pacific coral communities than previously thought (Willis et al., 2004).

Coral diseases have been well documented in the Caribbean (e.g. Rodriguez-Martinez et al., 2001; Weil et al., 2002), but little is known about Indo-Pacific coral diseases despite the region encompassing over 80% of reefs worldwide and >90% of the world's coral species (Bryant et al., 1998). The comparatively few reports of coral disease from Indo-Pacific reefs is in contrast to the high proportion (>65%) of records in the Global Disease Database from the Caribbean region, now considered to be a coral disease hotspot (Green & Bruckner, 2000; Weil, 2004).

Preliminary surveys in Australia (Willis et al., 2004), the Philippines (Raymundo et al., 2003) and East Africa reveal significant and damaging new diseases in all locations surveyed. Despite greater species richness in the Indo-Pacific, the number of species affected by disease is proportionally much lower than in the Caribbean (Sutherland et al., 2004).

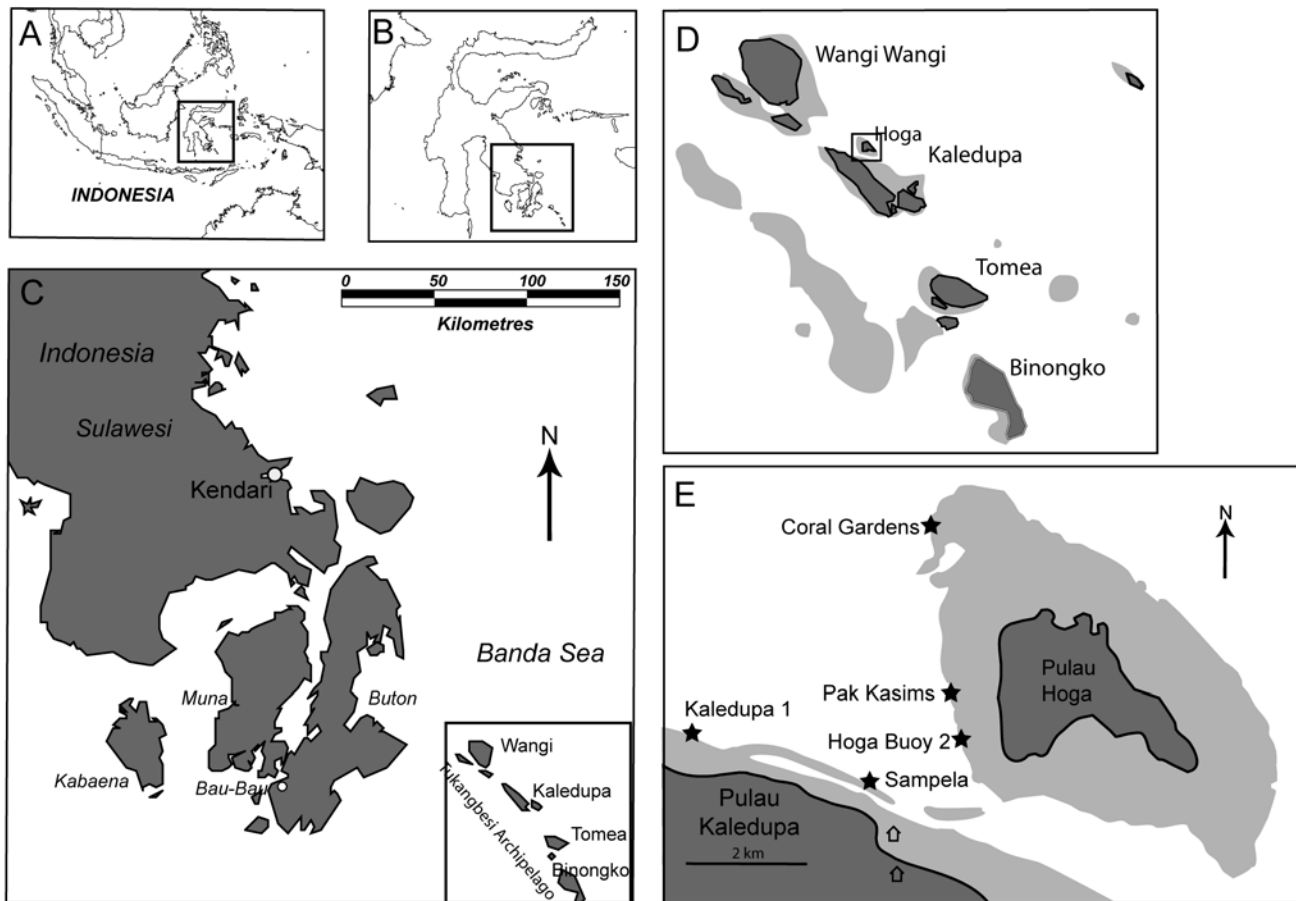


Figure 1. Map of the Wakatobi National Park and survey sites (Coral Gardens, Pak Kasims, Hoga Buoy 2, Sampela and Kaledupa 1).

There is a rising concern that coral diseases will alter both total abundance and species diversity of coral reefs in the Indo-Pacific such as has been found elsewhere in the world (Loya et al., 2001). Diseases may threaten the whole reef framework in the Indo-Pacific as has happened in the Caribbean (Nugues, 2002).

The aim of this study was to examine the frequency of occurrence of coral diseases in a typical Indo-Pacific reef system, the Wakatobi Marine National Park (WMNP). Species–disease associations were studied as well as any differences within and between bioregions by analysing the coral community structure. To date, this is the first attempt to describe coral diseases and their prevalence in the area.

MATERIALS AND METHODS

Study site

The Wakatobi Marine National Park (WMNP) is situated in the Tukang Besi Island region between the Banda and Flores Seas, south-east Sulawesi (3° – 6° S and $120^{\circ}45'$ – $124^{\circ}06'$ E) (Figure 1). It is the second largest marine national park in Indonesia covering an area of 1.39 million hectares.

In total, 396 species of hermatypic scleractinian corals and over 900 fish species are found in the park (Pet-Soede & Erdmann, 2003). Coral damage is widespread but patchy (Pet-Soede & Erdmann, 2003). Three major impact types are blast fishing, cyanide fishing and over-exploitation of marine resources (D. Smith, personal observation 2006).

Coral disease prevalence was determined at five sites located around the islands of Hoga and Kaledupa (Figure 1). The fringing reefs at these sites range in depth from <1 m to approximately 35 m. They are situated between 500 m and 1 km offshore. Sampling was conducted between 29 June and 16 September 2005.

Survey method

Surveys were conducted using belt transects, each covering an area measuring 20×4 m (2 m on each side of the transect line). Three replicate transects were laid on the reef flat (1–3 m depth), crest (3–7 m depth) and slope (8–12 m depth). Transects followed the depth contour of the reef. The first transect was located randomly in order to satisfy assumptions relating to independence of data for statistical analysis and the other two transects were based on the location of the first transect. A gap of 20 m was left between transects.

Each coral colony within a transect was counted and recorded as healthy or diseased. Data were expressed both as incidence (number of diseased colonies per site) and prevalence (proportion, or percentage, of diseased colonies per site). Prevalence of a certain disease was calculated by dividing the number of diseased colonies by the total number of coral colonies. Means and standard errors were calculated from all three transects at each depth at each site. Corals were recorded to genus or family level following the Australian Institute of Marine Science (AIMS) coral disease survey method.

Table 1. Description of five conditions found on corals in the Wakatobi Marine National Park.

Condition	Description
White syndrome	Narrow width of a recently exposed zone, white skeleton and a relatively regular appearance of the tissue front (Willis et al., 2004).
Tumour	A distinctive gross colony surface irregularity lacking zooxanthellar pigmentation.
Green spot	A few centimetres wide, irregular shaped green markings on the colony surface, found only on massive <i>Porites</i> .
Green band	From a few millimetres to a few centimetres wide band situated on the extremities of massive coral colonies, mostly on massive <i>Porites</i> .
Pigmented spots	From a few millimetres to a few centimetres wide regular, round markings of pink colour, found on the surface of massive <i>Porites</i> .

Coral cover of the area was determined by using a point intercept method on a 50 m long transect (points situated 0.5 m apart). A visual estimate of the medium size of coral colonies was made for each transect by the same person.

Disease identification

Photographs of diseased corals were taken and identified using the AIMS coral disease identification cards and photographs compiled by Willis et al. (2004). Photographs were taken of all disease categories and used as a reference in order to keep identification of the diseases consistent. Identification of diseases was further confirmed by Bette Willis at James Cook University, Australia.

White syndrome (WS) was used to describe conditions resulting in white bands of tissue and/or skeleton on corals found in the Wakatobi. In addition to WBI/II and WPI/II found in the Caribbean, white syndrome could potentially encompass white pox (Patterson et al., 2002), patchy necrosis (Rodriguez-Martinez et al., 2001), and even shut down reaction (Antonius, 1977). White syndrome and the other encountered diseases are described in Table 1. The lesions photographed are signs of partial mortality of the colony.

Statistical analyses

Coral community and pathology assemblage data were analysed using principle components analysis (PCA) and redundancy analysis (RDA) (Ter Braak, 1995). The PCA was used to display coral community and pathology assemblage data on PCA biplots for easy visual interpretation. The RDA was used to generate ordination axes and associated eigenvalues for coral community and pathology assemblage data constrained by two environmental variables: site and position (Ter Braak, 1995). Hypotheses that coral community structure and pathology assemblages differed significantly between site and position were tested using a Monte Carlo method, in which the observed eigenvalues for the first two RDA axes were compared with a distribution of 1000

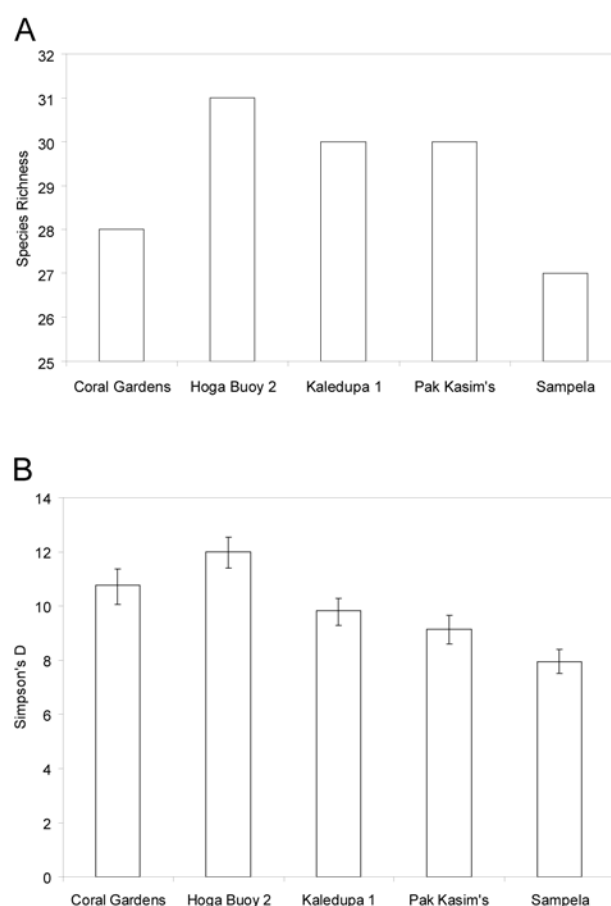


Figure 2. Number of taxonomic groups (A) and diversity (Simpson's D) \pm 95% confidence limits (obtained by bootstrapping) (B) by site.

simulated eigenvalues obtained from random arrangements of coral taxa or pathology data (i.e. data for each coral taxa or pathology randomly shuffled amongst sampling locations). These tests were made using the program ECOM (ECOM v. 1.37, Pisces Conservation Ltd).

Two-way factorial analyses of variance (ANOVA) were used to test hypotheses that site and position can predict coral cover and mean colony diameter using SPSS (SPSS v. 13.0, SPSS Inc.). Percentage coral cover data were arcsine transformed before analysis, and Tukey tests were used for post-hoc multiple comparisons using SPSS. A significance level of 0.05 was used for all tests.

RESULTS

A total of 12,352 coral colonies from 32 taxonomic groups was encountered in an area of 3600 m² (forty-five 20×4 m belt transects). A total of 1627 (13.2%) colonies showed signs of pathology, including bleaching, signs of predation, disease and unknown pathologies. By far the most frequently encountered type of pathology was the previously undescribed category (9.7% of colonies) that included unclassified conditions (3.0%), green spot (2.8%), pigmented spot (1.9%), green-band (1.2%), and flatworm infestation (0.8%). The next most frequent syndrome was coral bleaching (2.0% of colonies), followed by signs of predation (1.0%). Signs corresponding to known coral

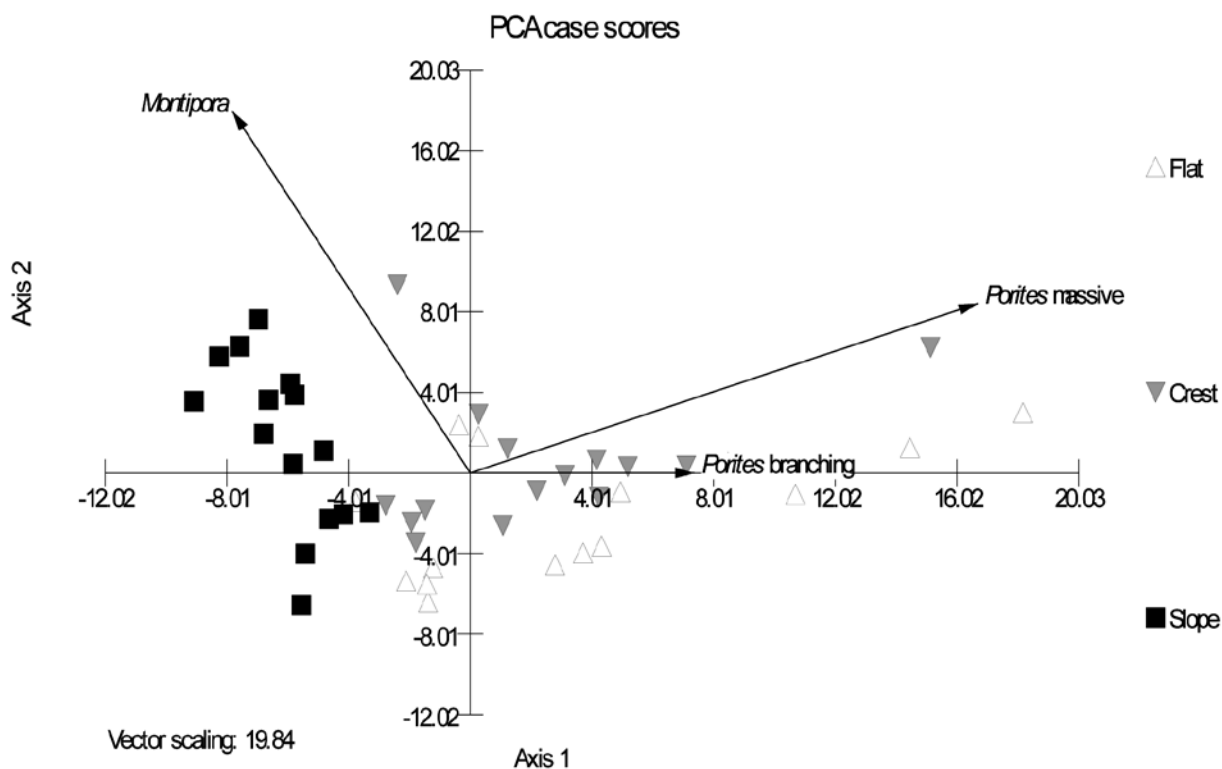


Figure 3. Principal component analysis (PCA) biplot of coral communities.

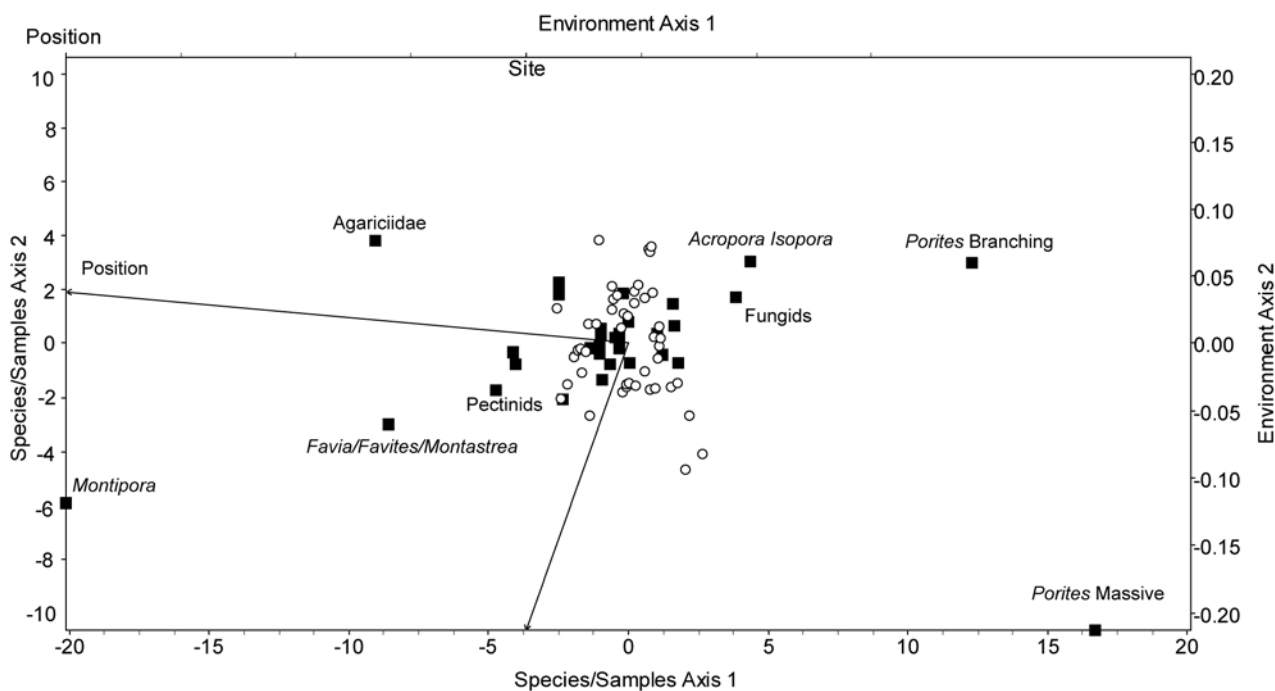


Figure 4. Redundancy analysis (RDA) biplot of coral community data using ‘site’ and ‘position’ as environmental variables. Black squares refer to species scores and white circles refer to location scores generated by the RDA.

diseases including white syndrome and tumours were only observed in 0.6% of colonies encountered. Black-band disease, SEB and *Porites* ulcerative white spot disease were observed at low prevalence in regions away from transects.

Coral community structure

Taxonomic groups of coral most commonly found on transects were massive forms of *Porites* (17% of colonies), followed by *Montipora* spp. (14%) and then the *Favia/Favites/Montastrea* group (11%). Sites of greatest and least coral

Table 2. ANOVA table showing the effect of site and position on arcsine transformed coral coverage. Adjusted R^2 of model=0.738. Tukey post-hoc tests indicate the only differences in coral cover between sites occur between Sampela and all other sites.

Source	SS	df	ms	F	P
Corrected model	0.91	14	0.06	9.87	0.00
Intercept	16.13	1	16.13	2462.48	0.00
Site	0.79	4	0.20	30.21	0.00
Position	0.03	2	0.01	2.02	0.15
Site * Position	0.09	8	0.01	1.66	0.15
Error	0.20	30	0.01		
Total	17.23	45			
Corrected total	1.10	44			

taxonomic group richness and diversity were Hoga Buoy 2 and Sampela respectively (Figure 2).

Although Coral Gardens had relatively low species richness, it had the second greatest value for Simpson's diversity index, suggesting a relatively even distribution of species abundance. The PCA biplot (Figure 3) showed a clear separation in the community of corals between positions (flat, crest and slope) along axis 1.

Reef slopes were characterized by high abundances of *Montipora* spp., whilst shallow reef flats and crests were dominated by massive colonies of *Porites*. Coral Gardens was characterized by unusually high frequencies of colonies of *Acropora isopora* and branching *Porites* spp., and relatively low frequencies of colonies of *Porites* massive, *Favia/Favites/Montastrea* spp. and agariciids. The most diverse site, Hoga Buoy 2, had intermediate frequencies of the five most abundant coral taxa, but high frequencies of some rarer groups including *Acropora* 'bushy', *Acropora* 'staghorn', and dendrophyllids. The least diverse site, Sampela, was characterized by an unusually high frequency of *Stylophora* colonies, and high frequencies of *Montipora* and *Favia/Favites/Montastrea* spp. colonies. Sampela had an unusually low frequency of *Porites* 'branching' colonies, and relatively few fungids. The first ordination axis generated by a redundancy analysis using site and position as environmental variables

Table 3. ANOVA table showing the effect of site and position on log transformed mean coral colony sizes (diameter in cm). Adjusted R^2 of model=0.673. Tukey post-hoc tests indicate that the mean colony size in Coral Gardens is larger than that in all other sites, and the mean colony size in Sampela is smaller than all other sites.

Source	SS	df	ms	F	P
Corrected model	3.57	14	0.25	7.48	0.00
Intercept	596.39	1	596.39	17491.39	0.00
Site	2.77	4	0.69	20.31	0.00
Position	0.20	2	0.10	3.00	0.06
Site * Position	0.60	8	0.07	2.18	0.06
Error	1.02	30	0.03		
Total	600.98	45			
Corrected total	4.59	44			

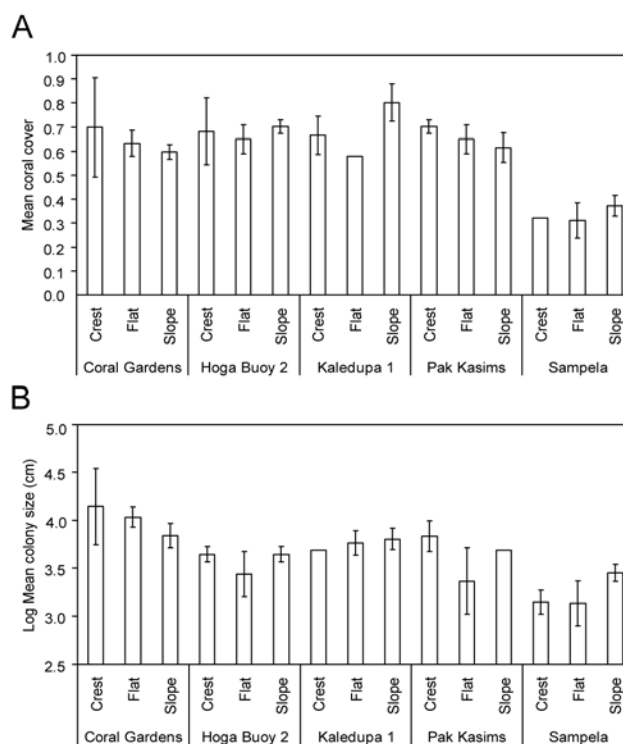


Figure 5. (A) Mean coral cover (proportion) \pm SD by site; (B) mean colony diameter (cm) \pm SD by site.

was strongly related to position (slope, crest, and flat) and axis two was strongly related to site (Figure 4). The observed eigenvalues of axes 1 and 2 were significantly greater than those expected from random arrangements of frequencies of coral colonies amongst sites (axis 1: observed eigenvalue=1119, mean expected eigenvalue=167, $P=0.001$; axis 2: observed eigenvalue=221, mean expected eigenvalue=50, $P=0.002$) showing that site and position explained a significant amount of variation in coral community composition.

Coral cover and colony size

Coral cover ranged from 7% (Sampela, flat) to 65% (Coral Gardens, reef crest). Coral cover was significantly lower in Sampela than other sites, but there was no significant difference in coral cover between positions and no significant interaction effect (Table 2; Figure 5A).

A high value of Cook's distance for one transect (transect 15 on the crest of Coral Gardens with very high coral cover; Cook's distance=0.43) suggested that this point had a disproportionate effect on the outcome of the ANOVA. When the analysis was repeated without this point, the effect of site was still significant ($F_{4,29}=50.3$, $P<0.001$), there was a significant interaction effect ($F_{8,29}=2.6$, $P=0.028$) and a better model fit (adjusted $R^2=0.822$). A Tukey post-hoc test still revealed that the only significant difference between sites occurred between Sampela and the other sites.

Mean diameter of coral colonies was significantly larger in Coral Gardens than other sites, and significantly smaller in Sampela than other sites (Table 3; Figure 5B), but there was no significant effect of position. Again a high value of Cook's distance for transect 15 on Coral Gardens' reef crest, suggested that this point may have an important effect on the results of the ANOVA. When this point was excluded

Table 4. Goodness of fit between the distributions of pathologies amongst taxonomic groups of corals. Expected frequencies are based on a random distribution of colonies showing symptoms of pathologies amongst taxonomic group.

Pathology	Chi-squared	df	P
Unclassifiable	104.8	18	<<0.001
Compromised*	2655.6	25	<<0.001
Bleached	118.8	12	<<0.001
Predated	178.3	7	<<0.001
Diseased	53.8	5	<<0.001

* includes recognizable symptoms of unknown origin including ‘green spot’, ‘green band’, ‘pigmented spot’ and flatworm infestation.

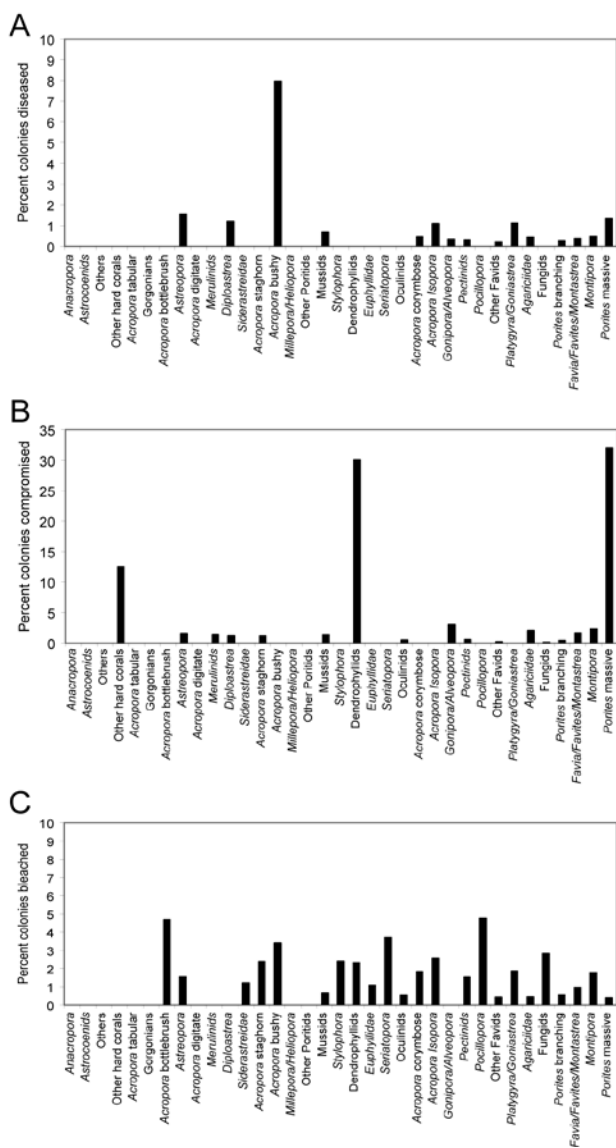


Figure 6. (A) Pathology data arranged by coral taxonomic group. Percentage of diseased (including white syndrome and tumours) colonies; (B) percentage of compromised (includes ‘green spot’, pigmented spot’, green band’ and flatworm infestation) colonies; (C) percentage of bleached colonies.

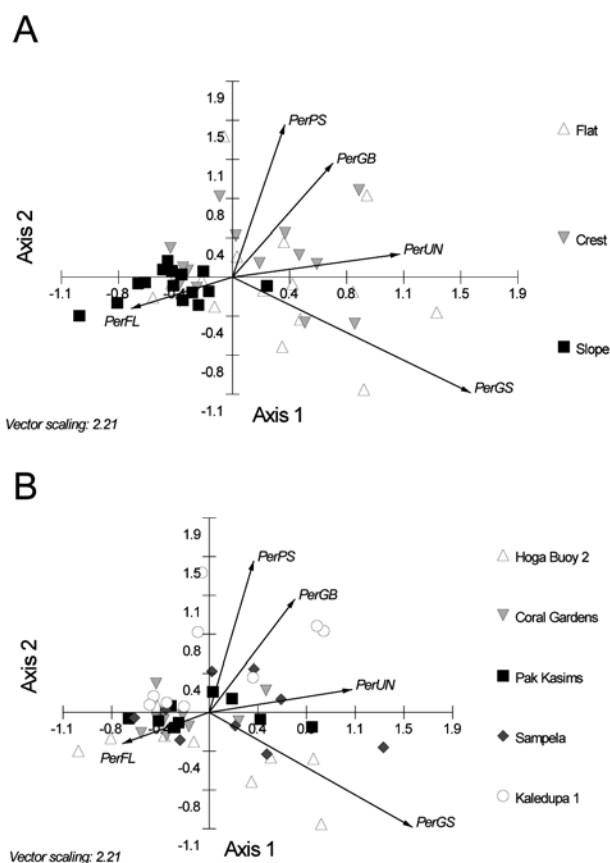


Figure 7. Principal component analysis (PCA) biplot of pathology assemblage data with points labelled by (A) position and (B) site. PerPS, PerUN, PerGB, PerGS and PerFL refer to pathology eigenvectors for the percentage of coral colonies with pigmented spot, unclassifiable signs, green band, green spot and flatworm infestation respectively.

from the analysis, site still had a highly significant effect on mean colony size ($F_{4,29}=22.6, P<0.001$), but as with coral cover, there was a significant interaction effect ($F_{8,29}=2.9, P<0.017$) and a better model fit (adjusted $R^2=0.714$).

Association of pathology with coral species

The frequency of colonies showing signs from each category of coral pathology was strongly dependent on coral taxonomic group (Figure 6A; Table 4).

A considerably higher proportion of colonies of *Acropora* ‘bushy’ suffered signs of known diseases (white syndrome and tumours) than any other coral taxa (8.0% colonies of *Acropora* ‘bushy’ infected versus 1.5% for next highest group, *Astreopora*). Unknown conditions and colonies infested by flatworms were classified as ‘compromised’. Unusually high percentage of colonies of *Porites* massive (32.0%) were compromised (showed signs of unknown conditions: ‘green spot’, ‘green band’ and ‘pigmented spot’). The high percentage of compromised dendrophyllids (30.1%) was due to flatworm infestations; all other taxa displaying these signs typically registered percentages lower than 3% (Figure 6B).

Twenty-three taxa had colonies showing signs of bleaching but at low percentages. In all cases the percentage of colonies showing signs of bleaching was less than 5% (Figure

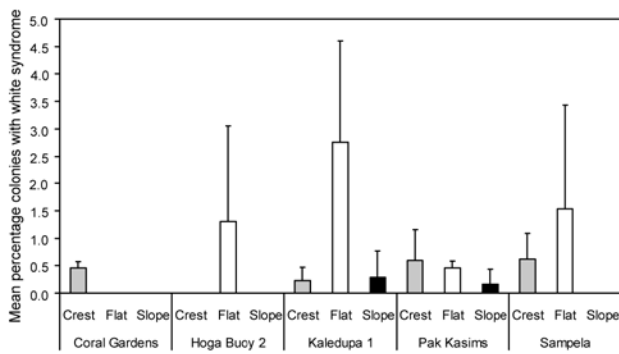


Figure 8. Mean percentage (\pm SD) of coral colonies showing signs of white syndrome.

6C). *Acropora tabularis* showed the greatest prevalence of signs of predation (4.8%) followed by merulinids (4.1%). *Porites* massive, the most abundant coral taxonomic group had a predation percentage of 3%.

Structure of pathology assemblages

The number of pathology types present at each site was very similar (tumours were absent from transects in Hoga Buoy 2, Kaledupa 1 and Sampela). A PCA biplot showed separation of pathology assemblages by position (Figure 7A) and site (Figure 7B).

Reef slopes were almost all situated on the left side of axis 1, and were characterized by relatively high percentages of colonies with flatworm infestations and low percentages of signs of other pathologies. There was little difference between pathology assemblages between sites on reef slopes. In contrast, the pathology assemblages on reef flats and crests varied with site. The flats and crests of Kaledupa 1 were characterized by high percentages of colonies showing signs of pigmented spot and green band, whilst those of Hoga Buoy 2 showed a high percentage of green spot. Observed eigenvalues of axes 1 and 2 generated by a redundancy analysis using site and position as habitat variables were significantly greater than those expected from random arrangements of pathology percentages amongst sites (axis 1: observed eigenvalue=4.9, mean expected eigenvalue=1.2, $P=0.001$; axis 2: observed eigenvalue=2.5, mean expected eigenvalue=0.4, $P=0.001$) showing that site and position explained a significant amount of variation in coral pathology assemblages.

Distribution of known diseases

Signs corresponding to two coral diseases were detected on sampling transects: white syndrome and tumours. Signs of white syndrome were found on 0.42% of all coral colonies, and tumours were found on only 0.15% of all colonies. The large majority of colonies with tumours (78%; $N=18$) were found on one transect on the crest of coral gardens. Colonies with tumours were detected in only three other transects, two in Coral Gardens (both on reef flats), and one in Pak Kasim's (reef slope). The mean percentage of colonies showing signs of white syndrome was higher on reef flats than crests or slopes (Figure 8).

Table 5. Logistic regression model using site and position to predict the presence of coral colonies with white syndrome. Since both site and position are categorical variables, each level of each category is treated as a 'dummy variable'. The model correctly predicts 80% of cases. $-2\log$ likelihood=40.00, Model $\chi^2=15.79$, $df=6$, $P=0.015$.

Variables	B	SE	Wald	df	P
Kaledupa 1			3.99	4.00	0.41
Hoga Buoy 2	-1.42	1.24	1.31	1.00	0.25
Coral Gardens	-2.40	1.43	2.82	1.00	0.09
Pak Kasim's	0.00	1.15	0.00	1.00	1.00
Sampela	-0.67	1.17	0.33	1.00	0.57
Slope			8.40	2.00	0.02
Flat	3.40	1.24	7.51	1.00	0.01
Crest	1.74	1.22	2.02	1.00	0.16
Constant	-2.04	1.23	2.78	1.00	0.10

Colonies with white syndrome were significantly more likely to occur on reef flats than other positions, but there was no significant effect of site (Table 5). Even though Coral Gardens had very low prevalence of white syndrome, site did not predict a significant amount of variation in the presence of white syndrome, and if site was removed from the logistic regression model, the drop in model fit was not significant (change in $-2\log$ likelihood=5.09, $df=4$, $P=0.28$).

DISCUSSION

There is mounting evidence that the emergence of new coral diseases is increasing, and the geographical distribution of diseases and host species range is extending (Goreau et al., 1998; Hayes & Goreau, 1998; Richardson, 1998; Harvell et al., 1999; Porter & Tougas, 2001; Porter et al., 2001; Rosenberg & Ben-Haim, 2002). Information regarding coral diseases in the Indo-Pacific region is limited and little quantitative work has been accomplished.

Coral communities in the study area

Corals within the study site could be clearly separated into shallow and deepwater communities with *Porites* being the most common genus (17%) in shallow water and *Montipora* (14%) in deep water. Raymundo et al. (2003) also found that *Porites* was the dominant genus with 30% coverage in the Philippines. The effect of hydrodynamic exposure might be an important factor in determining the depth preferences of coral genera in the study area. Riegl & Velimirov (1994) stated that this was a major factor influencing coral community structure in the Red Sea. Exposed coral communities were dominated by *Acropora* species and sheltered communities by *Porites* species in the Red Sea. Site and position explained a significant amount of variation in coral community composition. These results were expected, since Hoga Buoy 2 and Coral Gardens are relatively pristine sites and Sampela is the most impacted site. Close to Sampela is a Bajo (sea gypsy) village whose people rely on the surrounding reefs for food for its growing population. All wastewater is released straight into the waters surrounding the village, the major problem on this site being, therefore, light limitation due to sedimentation (Crabbe & Smith, 2005, 2006).

Previously undescribed conditions

The results indicated that 9.7% of colonies showed signs of previously undescribed or unclassifiable conditions. Corals in this category showed signs of green spot, green band, pigmented spot, and flatworm infestation. These corals were categorized as compromised, since there was no clear evidence of their impact on coral health. The pigmentation response is a possible result of reactive oxygen leading to coral pigmentation breakdown under high light or temperature conditions (D. Smith, personal observation, 2006). It is also possible that the pigmentation response might involve an unidentified microbial agent that may trigger the reaction or secondarily infect compromised tissue. Their potential for significant impacts on reef structure may be high, as massive *Porites* in particular seems to be host to multiple lesions. Studies of disease pathogenesis, differential species susceptibility, host defensive capabilities, etiology and causative agents are urgently needed (Raymundo et al., 2005).

Several different types of pigmentation responses have been observed on massive *Porites*. Willis et al. (2004) suggested that pigmentation appeared to be a symptom of a response mounted by the coral to contain invading or competing organisms such as cyanobacteria (Ravindran & Raghukumar, 2002), polychaetes, molluscs, and the intermediate metacercariae stage of the digenetic trematode, *Podocotylodes stenometra* (Aeby, 1991, 1998). The finding of this study may be the same pigmentation response that Raymundo et al. (2005) observed in the Philippines on *Porites*. Bongiorno & Rinkevich (2005) observed a pigmentation response resulting in pink-blue spots on branching *Acropora eurystroma* in the Red Sea and suspected that it was caused by restricted environmental and/or biological stress.

Association of pathology with coral species

The prevalence of disease and other signs was strongly dependent on coral taxonomic group. *Acropora* 'bushy' was a relatively rarely encountered growth form, but it had a higher disease (white syndrome and tumours) prevalence than any other coral taxa. On the Great Barrier Reef (GBR), disease prevalence varied among scleractinian families being greatest in Pocilloporidae (northern GBR) and Acroporidae (southern GBR) (Willis et al., 2004). Results showing a high percentage of green spot, green band and pigmented spot in *Porites* (32% of colonies) reflects the observations of Raymundo et al. (2005) in the Philippines, who also found that *Porites* was the genus most impacted by disease. In this study, *Porites* was mostly found in shallow water suggesting that species living in shallow waters are more prone to different syndromes due to higher temperatures and light exposure experienced in shallow areas.

With the exception of flatworm infestations, most pathologies in the compromised category were found on the flat and crest. Different topographies and current patterns could explain this difference. Kaledupa 1 is affected by strong tidal currents, whereas Hoga Buoy 2 is not. Kaledupa 1 has also been heavily impacted by blast fishing which causes direct damage (in craters) but also more diffuse damage to coral tissue in corals further away from the epi-centre that

may increase susceptibility to disease. Other sites have a quite even disease distribution.

Dendrophylliids had a high prevalence of flatworm infestations found almost entirely on the slope. Similar observations of corals infested by acoelid flatworms have been made from the Red Sea (Ogunlana et al., 2005), the Coral Sea (Winsor, 1990) and the Pacific Micronesian region (Trench & Winsor, 1987). Flatworms have been classified into two genera; *Convolutriloba* and *Waminoa*. *Waminoa* has reportedly infested some corals in relatively large numbers and was often sufficiently conspicuous to make a coral appear spotted. As Trench & Winsor (1987) suggested for *Waminoa*, its coral-infesting form may be a benthic phase in a life cycle involving a pelagic phase. Barneah (personal communication, 2006) found that corals with the worm were devoid of the mucus layer which may seriously interfere with the coral's ability to resist pathogens. It is not sure if the animals feed on the mucus or if they only 'brush' it off. Barneah also suspected that a large number of worms could interfere with the coral's photosynthesis by shading the zooxanthellae.

Observed levels of bleaching (2.0%) and predation (1%) were low. The level of bleaching corresponds to the amount found on the GBR, where Willis et al. (2004) found that it affected less than 1.7% of colonies under 'normal' conditions.

Disease prevalence

There was a low disease prevalence of 0.57% with only two identified diseases, white syndrome (0.42%) and tumours (0.15%) present. These diseases affected 15 taxonomic groups out of a total of 32 taxonomic groups. Observations of black-band disease (BBD), possibly skeletal eroding band (SEB) disease and *Porites* ulcerative white spot disease (PUWSD) described by Raymundo et al. (2003) were made outside the study area.

Compared to other regions in the world, levels of disease in the WMNP appear to be low. In the Philippines, Raymundo et al. (2005) found a mean total disease prevalence of 8.3 ± 1.2% (mean ± SE; N=8 reefs).

In the same study, the prevalence of tumours was 8.96 ± 2.2% respectively, and white syndrome was observed on *Montipora* outside the study area. Earlier at the same study site Raymundo et al. (2003) found a prevalence of 22 ± 7% (mean ± SE) for *Porites* ulcerative white spot disease (PUWSD). In the Caribbean values have tended to be higher: aspergillosis (39% of *Gorgonia* spp. in the Caribbean [Nagelkerken et al., 1997] and 47% of *Gorgonia* spp. in the Florida Keys [Kim & Harvell, 2002]), and Plague Type II (20.1% of *Dichocoenia stokesi* colonies [Richardson et al., 1998]). In contrast, reported prevalence values for BBD are generally lower: 0.2% in the Virgin Islands (Edmunds, 1991), 0.72% in the Florida Keys (Kuta & Richardson, 1996), 8.6% for *Siderastrea siderea* and 2.2% for *Montastraea annularis* in the Caribbean (Bruckner et al., 1997), with the GBR showing the highest prevalence (19%: Miller, 1996). On the GBR, white syndrome and SEB are the two most common diseases (Willis et al., 2004). The GBR total disease prevalence in hard corals ranged from a minimum of 7.2 ± 1.06% to a maximum of 10.7 ± 0.76%, both in the northern sector. In the Solitary Islands, Australia,

Dalton & Smith (2006) found a total disease prevalence of 6.21–13.58%. One of the greatest causes for concern on the Great Barrier Reef is, according to Willis et al. (2004), the 22 to 150-fold increase in the abundance of white syndrome on outer-shelf reefs in the northern and southern sectors of the GBR that have been detected over the last five years. The overall mean disease prevalence of $8.97 \pm 0.79\%$ at eight sites in the northern and southern sectors of the GBR in summer 2003 was higher than the $5.38 \pm 1.2\%$ mean disease prevalence that has been recorded in comparable surveys of 28 Caribbean sites in the past four years (Weil, 2004). Comparisons of white diseases between regions will not be possible until pathogens have been isolated. However, the potential for their presence and impact on coral communities in the Indo-Pacific should be viewed with concern (Willis et al., 2004).

Distribution of known diseases

Tumours were detected only in four transects, three of which were situated on the crest and flat transects of Coral Gardens. It is possible that tumorous growths are more common in shallow water habitats. Coles & Seapy (1998) found a substantially lower UVB absorption rate for tumorous *Acropora valenciennesi* extracts from a shallow reef flat. However, Coles & Seapy (1998) stated that reduced concentrations of UVB absorbing compounds may have been a consequence of tissue degeneration and loss of zooxanthellae accompanying tumour growth.

White syndrome was also observed mostly on crests and flats, suggesting a link to the more extreme light and physical regimes experienced in shallower waters. Willis et al. (2004) found that disease prevalence was higher in sheltered, shallow habitats. The reef flats of the study area, except for Sampela, are exposed to quite high tidal currents. According to Willis (personal communication, 2006), reef flats are stressful environments, which might reduce host resistance. There exists an array of factors contributing to patterns in disease prevalence such as temperature (e.g. Edmunds, 1991; Willis et al., 2004), high nutrient levels and turbidity (Kim & Harvell, 2002).

Effect of environmental factors on disease prevalence

Bell & Smith (2004) state that Sampela reef is impacted by high sedimentation rates and fine sediment particles that settle slowly, while Hoga experiences only fast settling coarse sediment with lower overall sedimentation rates. Crabbe & Smith (2002) found that the sedimentation rate was about four times higher in Sampela ($20.16 \text{ g dry weight m}^{-2} \text{ d}^{-1}$) than in Kaledupa ($5.35 \text{ g dry weight m}^{-2} \text{ d}^{-1}$). There were not significantly more diseased or compromised corals in Sampela.

Taylor (1983), Antonius (1988) and Bruckner et al. (1997) reported an increase in black-band disease activity on reefs that were close to sewage outflows and/or exposed to high sediment deposition; however, this was not quantified. Kim & Harvell (2002) revealed that aspergillosis distribution in the Florida Keys was more prevalent in waters with relatively higher dissolved inorganic nitrogen and slightly lower water clarity.

This study did not compare temporal changes in disease prevalence. There is a need to assess disease dynamics in

the area, only then can ecosystem responses be determined and used as a management tool. No data exist on disease prevalence in the rainy season (December–March) from the area. This would be interesting to measure in order to fully understand the extent of coral disease in the area. Raymundo et al. (2003) found a slight increase in infection of *Porites* during the rainy season. Edmunds (1991) reported a strong seasonality of the black-band disease in the US Virgin Islands, with highest disease frequency at water temperatures of 28°C . Willis et al. (2004) observed an increase in disease prevalence on the GBR in the austral summer.

It has been predicted that there will be an increase in frequency and severity of El Niño Southern Oscillation (ENSO) events in the future (Hoegh-Guldberg, 1999). High sea temperatures may allow certain bacterial pathogens to proliferate or induce virulence leading to disease. High temperatures may also cause physiological stress, leading to decreased immune competence and/or bleaching, and may benefit certain pathogens as well (Raymundo et al., 2003). At least four coral diseases are associated with high water temperature. Black-band disease (Edmunds, 1991; Kuta & Richardson, 1996; Bruckner et al., 1997; Kuta & Richardson, 2002), plague (Dustan, 1977; Richardson et al., 1998), and the newly described dark spots disease (Gil-Agudelo & Garzon-Ferreira, 2001) occur primarily during warm months of the year when water temperature is above 28°C .

The correlation between disease prevalence and environmental parameters is not clear, as suggested by the results of Raymundo et al. (2005) in the Philippines where disease prevalence was higher on reefs with high water quality, and on the Great Barrier Reef where the highest disease prevalence of white syndrome occurred on the outer reefs that were not impacted by human activities (Willis et al., 2004).

Problems and suggestions for future research

A major problem in this study, as in most studies of this kind, was the identification of coral diseases and syndromes. Unfortunately many existing descriptions of coral disease are ambiguous or open to subjective interpretation making comparisons problematic. Using photography as the only tool of disease identification has its limitations. Photography does not detect chronic illness or sub-clinical infections and can therefore only help in the estimation of what epidemiologists define as ‘true disease prevalence’.

In order to confirm prevalence, diagnostic tools to detect disease pathogens should be developed. Only then can issues of ‘true prevalence’ be addressed with certainty and the status of the health of the reef be defined. It would also be important to increase the amount of disease surveys covering large areas in the area. This would allow an estimate of the ‘endemic occurrence of disease’ which is the usual frequency of occurrence of disease in a population in the area, and to be able to detect the ‘epidemic occurrence’ which is an occurrence of an infectious or non-infectious disease to a level in excess of the expected (endemic) level in the area (Thrusfield, 2005).

Using transects as a survey method also has its problems. Disease outbreaks may be ‘clustered’, meaning that the

whole picture of health and disease of an area might be overlooked if only surveyed by using random transects.

Given the limited host-response repertoire of corals, it is likely that in certain cases different causal agents will elicit similar gross lesions (Work & Aeby, 2006). There is clearly a need for detailed studies on etiologies of white syndrome and tumours. In the Wakatobi, many signs interpreted as disease may be stress responses due to localized environmental stressors.

White syndrome may lead to increased rates of physical and biological erosion of the reef structure as well as the reduction of reproductive output and growth, and therefore coral fitness (e.g. Szmant-Froelich, 1985). The same might also be true for the observed pigmentation responses. These syndromes could lead to phase-shifts, for example an algae-dominated reef structure has been observed in the Caribbean as a result of the *Diadema antillarum* epizootic (Liddell & Ohlhorst, 1986; Hughes, 1994). In order to fully understand the impact of disease and compromising factors on coral health a study on disease and reef dynamics should be conducted within the WMNP.

Low disease prevalence in the WMNP may be a reflection of the areas isolation due to relatively cold surrounding ocean currents. These currents protected the WMNP from serious bleaching in 1998. With a prevalence of 0.57% of known disease, it is unlikely that coral diseases in the WMNP would lead to a shift from reefs dominated by corals to those dominated by algae like in the Caribbean (Porter & Meier, 1992; Porter et al., 2001), at least if they persist on their current levels. Destructive fishing methods and over-exploitation of the reefs in the area may be more likely to lead to this shift. The risk of coral disease prevalence could increase in the WMNP with rising sea temperatures associated with global climate change (IPCC, 2002). Pathogen virulence has been found to increase in warm temperatures (Harvell et al., 2002; Rosenberg & Ben-Haim, 2002).

This baseline information will be used for future in-depth studies targeting specific diseases or syndromes to elucidate pathogens, disease progression, and impacts on the reef community in order to better manage the reefs of the Wakatobi.

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REFERENCES

- Aeby, G.S., 1991. Behavioural and ecological relationship of a parasite and its hosts within a coral reef system. *Pacific Science*, **45**, 263–269.
- Aeby, G.S., 1998. A digenean metacercaria from the reef coral, *Porites compressa*, experimentally identified as *Podocotyloides stenometra*. *Journal of Parasitology*, **84**, 1259–1261.
- Antonius, A., 1977. Coral mortality in reefs: a problem for science and management. *Proceedings of the 3rd International Coral Reef Symposium*, **2**, 617–623.
- Antonius, A., 1985. Coral diseases in the Indo-Pacific: a first record. *PSZNI Marine Ecology*, **6**, 197–218.
- Antonius, A., 1988. Distribution and dynamics of coral diseases in the Eastern Red Sea. *Proceedings of the 6th International Coral Reef Symposium*, **2**, 293–298.
- Bell, J.J. & Smith, D., 2004. Ecology of sponge assemblages (Porifera) in the Wakatobi region, south-east Sulawesi, Indonesia: richness and abundance. *Journal of the Marine Biological Association of the United Kingdom*, **84**, 581–591.
- Bongiorni, L. & Rinkevich, B., 2005. The pink-blue spot syndrome in *Acropora eurystoma* (Filat, Red Sea): a possible marker of stress? *Zoology*, **108**, 247–256.
- Brown, B.E., 1997. Disturbances to reefs in recent times. In *Life and death of coral reefs* (ed. C. Birkeland), pp. 354–379. New York: Chapman & Hall.
- Bruckner, A.W., Bruckner, R.J. & Williams, E.H. Jr, 1997. Spread of a black-band disease epizootic through the coral reef system in St Ann's Bay Jamaica. *Bulletin of Marine Science*, **61**, 919–928.
- Bryant, D., Burke, L., McManus, J. & Spalding, M., 1998. *Reefs at risk: a map-based indicator of threats to the World's coral reefs*. Washington, DC: World Resource Institute.
- Carlton, R.G. & Richardson, L.L., 1995. Oxygen and sulphide dynamics in a horizontally migrating cyanobacterial mat: black band disease of corals. *FEMS Microbiology and Ecology*, **18**, 155–162.
- Coles, S.L. & Seapy, D.G., 1998. Ultra-violet absorbing compounds and tumorous growths on acroporid corals from Bandar Khayran, Gulf of Oman, Indian Ocean. *Coral Reefs*, **17**, 195–198.
- Cooney, R.P., Pantos, O., Le Tissier, M.D.A., Barer, M.R., O'Donnell, A.G. & Bythell, J.C., 2002. Characterization of the bacterial consortium associated with black band disease in coral using molecular microbiological techniques. *Environmental Microbiology*, **47**, 401–413.
- Crabbe, M.J.C. & Smith, D., 2002. Comparison of two reef sites in the Wakatobi Marine National Park (SE Sulawesi, Indonesia) using digital image analysis. *Coral Reefs*, **21**, 242–244.
- Crabbe, M.J.C. & Smith, D., 2005. Sediment impacts on growth rates of *Acropora* and *Porites* corals from fringing reefs of Sulawesi, Indonesia. *Coral Reefs*, **24**, 437–441.
- Crabbe, M.J.C. & Smith, D., 2006. Modelling variations in corallite morphology of *Galaxea fascicularis* coral colonies with depth and light on coastal fringing reefs in the Wakatobi Marine National Park (S.E. Sulawesi, Indonesia). *Computational Biology and Chemistry*, **30**, 155–159.
- Dalton, S.J. & Smith, D.A., 2006. Coral disease dynamics at a subtropical location, Solitary Islands Marine Park, eastern Australia. *Coral Reefs*, **25**, 37–45.
- Denner, E.B.M., Smith, G., Busse, H.J., Schumann, P., Narzt, T., Polson, S.W., Lubitz, W., Richardson, L.L., 2003. *Aurantimonas corallitida* gen. nov., sp. nov., the causative agent of white plague type II on Caribbean scleractinian corals. *International Journal of Systematic and Evolutionary Microbiology*, **53**, 1115–1122.
- Dustan, P., 1977. Vitality of reef coral populations off Key Largo, Florida; recruitment and mortality. *Environmental Geology and Water Science*, **2**, 51–58.
- Edmunds, P.J., 1991. Extent and effect of Black Band Disease on a Caribbean reef. *Coral Reefs*, **10**, 161–165.
- Garrett, T. & Ducklow, H., 1975. Coral diseases in Bermuda. *Nature, London*, **253**, 349–350.
- Geiser, D.M., Taylor, J.W., Ritchie, K.B. & Smith, G.W., 1998. Cause of sea fan death in the West Indies. *Nature, London*, **394**, 137–138.
- Gil-Agudelo, D.L. & Garzon-Ferreira, J., 2001. Spatial and seasonal variation of dark spots disease in coral communities of the Santa Marta area (Colombian Caribbean). Proceedings of the International Conference on Scientific Aspects of Coral Reef Assessment, Monitoring, and Restoration. *Bulletin of Marine Science*, **69**, 619–629.

- Green, E.P. & Bruckner, A.W., 2000. The significance of coral disease epizootiology for coral reef conservation. *Biological Conservation*, **96**, 347–361.
- Goreau, T.J. et al., 1998. Rapid spread of diseases in Caribbean coral reefs. *Revista de Biología Tropical*, **46**, 157–171.
- Harvell, C.D. et al., 1999. Emerging marine diseases—climate links and anthropogenic factors. *Science, New York*, **285**, 1505–1510.
- Harvell, C.D., Mitchell, C.E., Ward, J.R., Altizer, S., Dobson, A.P., Ostfeld, R.S. & Samuel, M.D., 2002. Climate warming and disease risks for terrestrial and marine biota. *Science, New York*, **296**, 2158–2162.
- Hayes, R.L. & Goreau, T.J., 1998. The significance of emerging diseases in the tropical coral reef ecosystem. *Revista de Biología Tropical*, **46**, 173–185.
- Hoegh-Guldberg, O., 1999. Climate change, coral bleaching and the future of the world's coral reefs. *Marine and Freshwater Research*, **50**, 839–866.
- Hughes, P., 1994. Catastrophes, phase shifts, and large-scale degradation of a Caribbean coral reef. *Science, New York*, **265**, 1547–1551.
- IPCC, 2002. *Climate change and biodiversity*. Intergovernmental Panel on Climate Change (ed. H. Gitay et al.). Technical Paper V, 86 pp.
- Kim, K. & Harvell, C.D., 2002. Aspergillosis of sea fan corals: disease dynamics in the Florida Keys. In *The Everglades, Florida Bay, and coral reefs of the Florida Keys: an ecosystem sourcebook* (ed. J.W. Porter and K.G. Porter) pp. 813–824. Boca Raton: CRC Press.
- Korrubel, J.L. & Riegl, B., 1998. A new coral disease from the southern Arabian Gulf. *Coral Reefs*, **17**, 22.
- Kushmaro, A., Loya, Y., Fine, M. & Rosenberg, E., 1996. Bacterial infection and coral bleaching. *Nature, London*, **380**, 396.
- Kushmaro, A., Rosenberg, E., Fine, M. & Loya, Y., 1997. Bleaching of the coral *Oculina patagonica* by *Vibrio* AK-1. *Marine Ecology Progress Series*, **147**, 159–165.
- Kuta, K.G. & Richardson, L.L., 1996. Abundance and distribution of black band disease on coral reefs in the northern Florida Keys. *Coral Reefs*, **15**, 219–223.
- Kuta, K.G. & Richardson, L.L., 2002. Ecological aspects of black band disease of corals: relationships between disease prevalence and environmental factors. *Coral Reefs*, **21**, 393–398.
- Liddell, W.D. & Ohlhorst, S.L., 1986. Changes in benthic community composition following the mass Mortality of *Diadema* at Jamaica. *Journal of Experimental Marine Biology and Ecology*, **95**, 271–278.
- Loya, Y., Sakai, K., Yamazato, K., Nakano, Y., Sambali, H. & Woesik, R. van, 2001. Coral bleaching: the winners and the losers? *Ecology Letters*, **4**, 122–131.
- Miller, I., 1996. Black-band disease on the Great Barrier Reef. *Coral Reefs*, **15**, 58.
- Nagelkerken, I. et al., 1997. Widespread disease in Caribbean sea fans: II. Patterns of infection and tissue loss. *Marine Ecology Progress Series*, **160**, 255–263.
- Nugues, M.M., 2002. Impact of a coral disease outbreak on coral communities in St. Lucia: what and how much has been lost? *Marine Ecology Progress Series*, **229**, 61–71.
- Ogunlana, M.V., Hooge, M.D., Tekle, Y.I., Benayahu, Y., Barneah, O. & Tyler, S., 2005. *Waminoa brickneri* n. sp. (Acoela: Acoelomorpha) associated with corals in the Red Sea. *Zootaxa*, **1008**, 1–11.
- Patterson, K.L., Porter, J.W., Ritchie, K.B., Polson, S.W., Mueller, E., Peters, E.C., Santavy, D.L. & Smith, G.W., 2002. The etiology of white pox, a lethal disease of the Caribbean Elkhorn coral, *Acropora palmata*. *Proceedings of the National Academy of Sciences of the United States of America*, **99**, 8725–8730.
- Pet-Soede, C. & Erdmann, M.V.E., 2003. Rapid Ecological Assessment—Wakatobi National Park. *Report from WWF Indonesia Indonesia Marine Program*, Denpasar, Bali, Indonesia.
- Porter, J.W. & Meier, O.W., 1992. Quantification of loss and change in Floridian reef coral populations. *American Zoologist*, **32**, 625–640.
- Porter, J.W. & Tougas, J.I., 2001. Reef ecosystems: threats to their biodiversity. In *Encyclopedia of biodiversity*, vol 5, pp. 73–95. New York: Academic Press.
- Porter, J.W., Dustan, P., Jaap, W.C., Patterson, K.L., Kosmynin, V., Meier, O.W., Patterson, M.E. & Parsons, M., 2001. Patterns of spread of coral disease in the Florida Keys. *Hydrobiologia*, **460**, 1–24.
- Ravindran, J. & Raghukumar, C., 2002. Pink line syndrome (PLS) in the scleractinian coral *Porites lutea*. *Coral Reefs*, **21**, 252.
- Raymundo, L., Harvell, C.D. & Reynolds, T., 2003. *Porites* ulcerative white spot disease: description, prevalence, and host range: a new disease impacting Indo-pacific reefs. *Diseases of Aquatic Organisms*, **56**, 95–104.
- Raymundo, L., Rosell, K.B., Reboton, C.T. & Kaczmarzsky, L., 2005. Coral diseases on Philippine reefs: genus *Porites* is a dominant host. *Diseases of Aquatic Organisms*, **64**, 181–191.
- Richardson, L.L., 1998. Coral diseases: what is really known. *Trends in Evolutionary Ecology*, **13**, 438–443.
- Richardson, L.L., Goldberg, W.M., Carlton, R.G. & Halas, J.C., 1998. Coral disease outbreak in the Florida Keys: plague type II. *Revista de Biología Tropical*, Suppl. 5, 187–198.
- Riegl, B. & Velimirov, B., 1994. The structure of coral communities at Hurghada in the northern Red Sea. *Marine Ecology*, **15**, 213–231.
- Rodriguez-Martinez, R.E., Banaszak, A.T. & Jordan-Dahlgren, E., 2001. Necrotic patches affect *Acropora palmata* (Scleractinia: Acroporidae) in the Mexican Caribbean. *Diseases of Aquatic Organisms*, **47**, 229–234.
- Rosenberg, E. & Ben-Haim, Y., 2002. Microbial diseases of corals and global warming. *Environmental Microbiology*, **4**, 318–326.
- Sebens, K.P., 1994. Biodiversity of coral reefs: what are we losing and why? *American Zoologist*, **34**, 115–133.
- Shinn, E.A., Smith, G.W., Prospero, J.M., Betzer, P., Hayes, M.L., Garrison, V. & Barber, R.T., 2000. African dust and the demise of Caribbean coral reefs. *Geophysical Research Letters*, **27**, 3029–3032.
- Sinderman, C.J., 1990. *Principal diseases of marine fish and shellfish*, vol. 2, 2nd edn. New York: Academic Press.
- Smith, G.W., Ives, L.D., Nagelkerken, I.A. & Ritchie, K.B., 1996. Caribbean sea-fan mortalities. *Nature, London*, **383**, 487.
- Sussman, M., Loya, Y., Fine, M. & Rosenberg, E., 2003. The marine fireworm *Hermodice carunculata* is a winter reservoir and spring-summer vector for the coral-bleaching pathogen *Vibrio shiloi*. *Environmental Microbiology*, **5**, 250–255.
- Sutherland, K.P., Porter, J.W. & Torres, C., 2004. Disease and immunity in Caribbean and Indo-Pacific zooxanthellate corals. *Marine Ecology Progress Series*, **266**, 273–302.
- Szmant-Froelich, A., 1985. The effect of colony size on the reproductive ability of the Caribbean coral *Montastrea annularis*. *Proceedings of the 5th International Coral Reef Symposium*, **4**, 295–300.
- Taylor, D.L., 1983. The black band disease of Atlantic reef corals. 2. Isolation, cultivation, and growth of *Phormidium corallyticum*. *PSZNI Marine Ecology*, **4**, 321–328.
- Ter Braak, C.J.F., 1995. Ordination. In *Data analysis in community and landscape ecology* (ed. R.H.G. Jongman et al.), pp. 91–173. Cambridge: Cambridge University Press.
- Thrusfield, M., 2005. *Veterinary epidemiology*, 3rd edn. Oxford: Blackwell Science.
- Trench, R.K. & Winsor, L., 1987. Symbiosis with dinoflagellates in two pelagic flatworms, *Amphisclops* sp. and *Haplodiscus* sp. *Symbiosis*, **3**, 1–22.
- Weil, E., Urreiztieta, I. & Garzón-Ferreira, J., 2002. Geographic variability in the prevalence of coral and octocoral disease in the wider Caribbean. *Proceedings of the 9th International Coral Reef Symposium*, **2**, 1231–1238.

- Weil, E., 2004. Coral reef diseases in the wider Caribbean. In *Coral health and disease* (ed. E. Rosenberg and Y. Loya), pp. 35–68. Berlin: Springer-Verlag.
- Weil, E., Smith, G. & Gil-Agudelo, D.L., 2006. Status and progress in coral reef disease research. *Diseases of Aquatic Organisms*, **69**, 1–7.
- Wilkinson, C., ed., 2004. *Status of coral reefs of the world: 2004*. Townsville: Australian Institute of Marine Science.
- Willis, B.L., Page, C.A. & Dinsdale, L.A., 2004. Coral disease on the Great Barrier Reef. In *Coral health and disease* (ed. E. Rosenberg and Y. Loya), pp. 69–104. Berlin: Springer-Verlag.
- Winsor, L., 1990. Marine Turbellaria (Acoela) from North Queensland. *Memoirs of the Queensland Museum*, **28**, 785–800.
- Work, T.M. & Aeby, G.S., 2006. Describing gross lesions in corals. *Diseases of Aquatic Organisms*, **70**, 155–160.

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