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Spatio-temporal coral disease dynamics in the Wakatobi Marine National Park, South-East Sulawesi, Indonesia

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ABSTRACT: In the present study we investigated inter-annual coral disease dynamics, *in situ* disease progression rates, and disease-associated coral tissue mortality in the Wakatobi Marine National Park (WMNP) situated in the coral triangle in South-East Sulawesi, Indonesia. In 2005, only 2 known syndromes were recorded within the sampling area transect surveys: white syndrome (WS; 0.42% prevalence) and growth anomalies (GA; 0.15% prevalence), whilst 4 diseases were recorded in 2007: WS (0.19%), *Porites* ulcerative white spot disease (PUWS; 0.08%), GA (0.05%) and black band disease (BBD; 0.02%). Total disease prevalence decreased from 0.57% in 2005 to 0.33% in 2007. In addition to prevalence surveys, *in situ* progression rates of 4 diseases were investigated in 2007: BBD on *Pachyseris foliosa*, *P. rugosa* and *Diploastrea heliopora*, WS on *Acropora clathrata*, and brown band (BrB) and skeletal eroding band (SEB) diseases on *Acropora pulchra*. BrB and WS had the highest progression rates, 1.2 ± 0.36 and 1.1 ± 0.07 cm d^{-1}, respectively, indicating that diseases may have a significant impact on local *Acropora* populations. BBD had the lowest progression rate (0.39 ± 0.14 cm d^{-1}). WS caused the most severe recorded total tissue mortality: 53 923 cm^2 over a period of 36 d. Sedimentation and coral cover were studied and a highly significant drop in coral cover was observed. This study provides the first documentation of spatio-temporal coral disease dynamics from Indonesia. Despite low total disease prevalence, progression rates comparable to the ones observed in the Caribbean and Australia indicate that diseases may threaten the reef framework in some locations and add to the degradation of coral reefs in a region already at high risk from anthropogenic impacts.

KEY WORDS: Coral Triangle · Indo-Pacific · Disease prevalence · Disease progression rates · Tissue mortality · Sedimentation

INTRODUCTION

Coral diseases are one of the main factors contributing to the global deterioration of coral reefs (Weil et al. 2006). Coral diseases may contribute to coral–algal phase-shifts through mortality of key reef-building corals and consequent changes to the reef framework (Nugues 2002). Coral disease prevalence studies can reveal trends in disease over time as well as predict possible changes to coral communities by identifying the coral taxa most affected by disease (Lafferty et al. 2004). Rates of disease spread and tissue loss are also important concerns in understanding the impacts of diseases on coral populations (Willis et al. 2004).

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Diseases have been well-studied in the Caribbean, which is considered a 'disease hot spot' due to the fast emergence and high virulence of coral reef diseases/syndromes, their widespread geographic distribution, wide host range, and frequent epizootic events with significant coral mortalities (Epstein et al. 1998, Hayes & Goreau 1998, Green & Bruckner 2000, Weil et al. 2002a, Weil 2004). However, much less is known about coral diseases in the Indo-Pacific region (Weil et al. 2006).

Examples of disease outbreaks from the Caribbean are numerous. One of the most devastating diseases has been white band disease that was first reported by Gladfelter (1982). The large-scale die-off of key reef-building corals *Acropora palmata* (now IUCN red-listed) and *A. cervicornis* has been attributed to the combined impacts of white band disease, white pox (Patterson et al. 2002) and hurricane damage (Woodley 1989, Hughes 1994). Another example of a coral disease outbreak in the Caribbean is the rapidly spreading disease white plague II (WPII), which has destroyed 75% of the key Caribbean reef-building coral *Dichocoenia stokesi* in 7 yr and shifted the population structure in a way which suggests that the remaining *D. stokesi* population is no longer reproducing (Richardson & Voss 2005).

Despite the fact that no major disease outbreaks have yet been reported from the Indo-Pacific, an increasing number of coral diseases have now been observed at several locations: Australia (Willis et al. 2004), the Philippines (Raymundo et al. 2003), Hawaii (Aeby 2005), US Pacific remote islands (Vargas-Ángel 2009) and Indonesia (Haapkylä et al. 2007). Diseases have been monitored on the Great Barrier Reef (GBR) since 1998 revealing a 20-fold increase in white syndrome (WS) between 1998 and 2003 (Willis et al. 2004). The overall disease prevalence was 8.97 ± 0.79% in northern Cooktown/Lizard Island and southern Capricorn Bunker sectors of the GBR in 2004 (Willis et al. 2004). Raymundo et al. (2005) reported a total disease prevalence of 8.3 ± 1.2% (n = 8 reefs) in the Philippines, and prevalences of *Porites* ulcerative white spot (PUWS) and growth anomalies (GA) were 53.7 and 39.1%, respectively, in 2002–2003 (Kaczmarsky 2006). These studies indicate that infectious pathogens may be a common component of Indo-Pacific coral communities, and may play a greater role in structuring these communities than previously thought (Willis et al. 2004).

Spatio-temporal dynamics of coral diseases are often driven by environmental factors. Anomalously high temperature and other environmental stresses can influence the severity and dynamics of infectious coral diseases by increasing host susceptibility and pathogen virulence (Harvell et al. 2002, Lafferty & Holt 2003). The frequency of temperature anomalies, which is predicted to increase in most tropical oceans, can therefore increase the susceptibility of corals to disease, leading to outbreaks where corals are abundant (Bruno et al. 2007). Other environmental factors that can increase disease susceptibility include sedimentation (Voss & Richardson 2006), turbidity (Bruckner & Bruckner 1997) and nutrients (Bruno et al. 2003).

Little is known about the impacts of coral diseases within the coral triangle region of SE Asia, which is regarded as a global marine biodiversity hotspot (Roberts et al. 2002). Wilkinson (2008) reported that coral reefs in Indonesia have continued to show an overall decline in condition since 2004; however, the role of disease in this decline remains poorly understood. Haapkylä et al. (2007) documented the occurrence of coral disease in the Wakatobi Marine National Park (WMNP). In the present study we describe inter-annual coral disease dynamics and investigate the impact of disease on coral assemblages by recording the *in situ* progression rates and tissue mortality caused by 4 coral diseases in the WMNP.

MATERIALS AND METHODS

Study site. The WMNP is the second largest marine national park in Indonesia and covers an area of 1.39 million ha. It is situated in the Tukangbesi Island region between the Banda and Flores Seas, South-East Sulawesi (3–6° S, 120° 45′–124° 06′ E) (Fig. 1). Indonesian coral reefs are among the most diverse in the world (Allen 2007). The WMNP is situated in a global biodiversity hotspot with 396 species of hermatypic scleractinian corals belonging to 68 genera and 15 families (Turak 2003). 10 species of non-scleractinian or ahermatypic hard coral species and 28 soft coral genera are also found in the park (Pet-Soede & Erdmann 2004). Coral reef habitats of the WMNP are mostly in a healthy state, but incidences of declining coral cover and reduced reef predators related to increased fishing pressure are cause for concern (McMellor 2007, Unsworth et al. 2007).

Five sites located around the islands of Hoga and Kaledupa were surveyed in 2005 and 2007 (Fig. 1). These sites represent a typical Indonesian fringing reef. Sampela is the only site situated close to a local village. The fringing reefs at these sites range in depth from <1 m to approximately 35 m and are situated between 500 m and 1 km offshore. Sampling was conducted between 29 June and 16 September 2005 and between 30 June and 4 September 2007.

Inter-annual study. Surveys were conducted using belt transects (English et al. 1997) covering an area of 4 × 20 m (2 m on each side of the transect line) in both

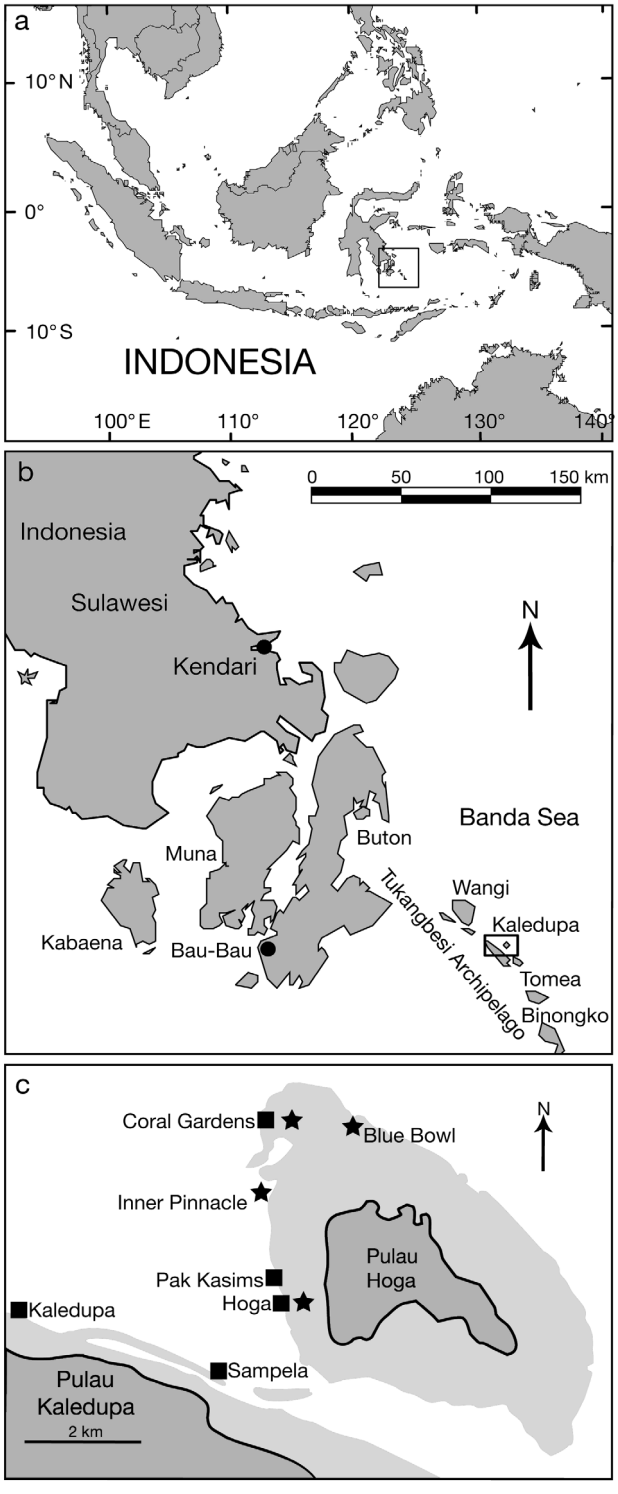

Fig. 1. Location of survey sites. (a) South-East Sulawesi (boxed); (b) Wakatobi National Marine Park (WMNP) includes Tukangbesi Archipelago: Wangi, Kaledupa, Hoga (boxed), Tomea and Binongko Islands; (c) survey sites used in 2005 and 2007 around Hoga and Kaledupa Islands. ■: inter-annual survey sites; ★: sites where disease progression rates and tissue mortality were observed; light grey shading: reef flats around Hoga and Kaledupa Islands

years. Three replicate transects were laid in 3 reef zones: flat (1 to 3 m depth), crest (3 to 7 m depth) and slope (8 to 12 m depth) and each transect was treated as 1 replicate in the analysis. A total of 45 transects were surveyed in both years. Transects followed the depth contour of the reef. The first transect was located randomly to satisfy assumptions about the independence of data for statistical analysis. The additional transects were located at randomly derived distances from the first transect, but always at >20 m to ensure independence and to detect site-specific trends and variances. Each coral colony within the belt was counted to genus or family levels and recorded as healthy or diseased according to disease survey methods described by Willis et al. (2004). Prevalence of each disease was calculated by dividing the number of diseased colonies by the total number of coral colonies. Due to the prevalence of disease among different coral taxa, this was believed to be the appropriate method. This method has been previously used in Indo-Pacific disease studies by e.g. Page & Willis (2008), Raymundo et al. (2005) and Vargas-Ángel (2009). Means and standard errors (SE) were calculated from all 3 transects at each reef zone at each site. Coral cover was estimated by the same observer by using the 0.5 m point intercept transect method in 2005 and the line intercept transect method in 2007 (English et al. 1997).

Disease identification. A disease is defined as any deviation or alteration from the normal structure or function of any body part or organ manifested by a characteristic set of clinical signs of known or unknown cause (Dorland 1982). A lesion represents any functional and morphologic change in tissues during disease (Work & Aeby 2006).

Coral diseases were identified by the presence and characteristics of lesions. Photographs of diseased corals were taken and identified using the Australian Institute of Marine Science (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. Lesions that did not correspond to any of the disease categories were classified as 'undescribed' and abnormally pigmented lesions on corals were classified as 'pigmentation responses'. Samples of brown band disease (BrB) and skeletal eroding band (SEB) were collected and examined microscopically to verify the presence of ciliates that characterize these diseases.

Disease progression rates. The progression rates of black band disease BBD, SEB, BrB and WS were investigated by taking photographs at a fixed angle and including a flexible measuring tape. The diseases were studied in 5 different sites: Blue Bowl, Coral Gardens, Pak Kasims, Inner Pinnacle and Hoga (Fig. 1). The

observation time varied between 5 and 38 d depending on the disease. Progression rates of SEB and BrB on branching *Acropora pulchra* were investigated on randomly selected branches of separate coral colonies (SEB, n = 15; BrB, n = 4) (see Table 3). A cable tie was secured onto the exposed skeleton a short distance behind the disease front to avoid interfering with disease progression. The distance from the cable tie to the nearest live tissue at each observation time was measured from photographs using the software Canvas^{TM}X (System version 10.5.5). The difference between the last and the first measurement was used as a measure of linear disease progression and divided by the number of days between measurements to calculate a daily progression rate. Using the software Canvas^{TM}X (System version 10.5.5), 3 independent measurements extending from a stable reference point on the intersection between healthy and diseased coral tissue/freshly exposed skeleton were recorded for each colony with BBD (foliaceous *Pachyseris foliosa*, n = 19; hemispherical *Diploastrea heliopora*, n = 1; laminar *Pachyseris rugosa*, n = 1) and WS (laminar *Acropora clathrata*, n = 6) after each observation time (see Table 3). The mean rate of disease spread between survey times was determined for each colony by calculating the difference between the respective measurements for each survey date (i.e. subtracting the length of Measurement 1 in the image from July 4 from Measurement 2 on July 10) and averaging the 3 resultant differences. The average measurements from each colony were divided with the number of days between survey dates to calculate an average daily disease progression rate. Finally, the mean rate-of-spread of disease for each affected species between each successive survey period was calculated using the data from all individual colonies within each species.

Tissue loss due to coral disease. To determine the tissue loss due to BBD and WS, the surface area of dead coral tissue was measured for each survey date by using the software Canvas^{TM}X (System version 10.5.5). The average tissue losses were calculated as described above for the disease progression. The surface area of the *Diploastrea heliopora* colony was estimated assuming a hemispherical colony shape: $A = 4\pi r^2/2$ where A = surface area, r = radius.

The single colony of *Diploastrea heliopora* on which we measured tissue loss had a radius of 50 cm. The surface area of *Acropora pulchra*s impacted by SEB and BrB was calculated by using the formula for cylinder area: $A = 2\pi(r) \times h$ where A = surface area, r = radius and h = height (i.e. dead coral tissue).

The diameter of *Acropora pulchra* varies between 7 and 15 mm (Wallace 1978). We used the mean of the smallest and the largest diameter (7.5 mm) to obtain a range of tissue loss caused by disease.

Environmental parameters. Sedimentation rates were assessed using 4 standard sediment traps (English et al. 1997) deployed at each depth within all sites for a 10 d period. Sediment and water were filtered, dried and weighed with rates expressed as mg dry weight (DW) cm^{-2} d^{-1}.

Statistical analyses. Three-way permutational ANOVAs (Anderson 2001, McArdle & Anderson 2001) of disease prevalence, WS and GA prevalence, and coral cover were performed using PERMANOVA (version 1.6). Sedimentation values were $\log_{10}$-transformed and a 2-way nested ANOVA was conducted by using MINITAB (version 13.20). Tukey tests were used for post-hoc multiple comparisons. We used $\alpha = 0.05$ for all tests. The distributions of coral diseases within each coral taxa in each site in each year were compared using non-metric multi-dimensional scaling (nMDS) based on Bray-Curtis similarity measures. Difference between years, transects and reef zones were tested using ANOSIM, which is a non-parametric permutation procedure. After identification of the transects and years which differed the most (ANOSIM pairwise test output), SIMPER analysis was run on the data matrix. SIMPER decomposes Bray-Curtis dissimilarities between all pairs of samples to identify those species that contribute most to differences (Clarke & Warwick 2001). All multivariate analyses were conducted using PRIMER (version 6.1.10).

RESULTS

A total of 12 271 colonies were encountered in 2005 and 12 752 colonies in 2007 in an area of 3600 m^2 (45 belt transects of 4 × 20 m). These colonies represented 32 coral taxa. Total disease prevalence dropped significantly from 0.57% in 2005 to 0.33% in 2007 ($F_{1,60}$ = 4.84, $p < 0.05$) (Fig. 2, Table 1). In 2005 the most frequent type of lesion was the undescribed category (9.7% of all colonies with lesions that did not correspond to any of the disease categories), while pigmentation responses were most frequent in 2007 (3.42% of all colonies had non-normally pigmented lesions).

In 2005, only 2 known syndromes occurred within the sampling area transects: WS (0.42%) and GA (0.15%) (Haapkylä et al. 2007). In 2007, the prevalence of these 2 syndromes was lower: 0.19% for WS and 0.05% for GA. In addition 2 other syndromes were identified in the sampling area transects: PUWS (0.08%) and BBD (0.02%).

There was a significant interaction between reef zone and overall disease prevalence ($F_{20,60}$ = 2.35, $p < 0.01$) (Table 1) with a significant difference between Hoga crests and slopes in 2005, and Sampela flats and slopes in 2007. The prevalence of WS was significantly lower in 2007 ($F_{1,60}$ = 10.02, $p < 0.01$). There was a

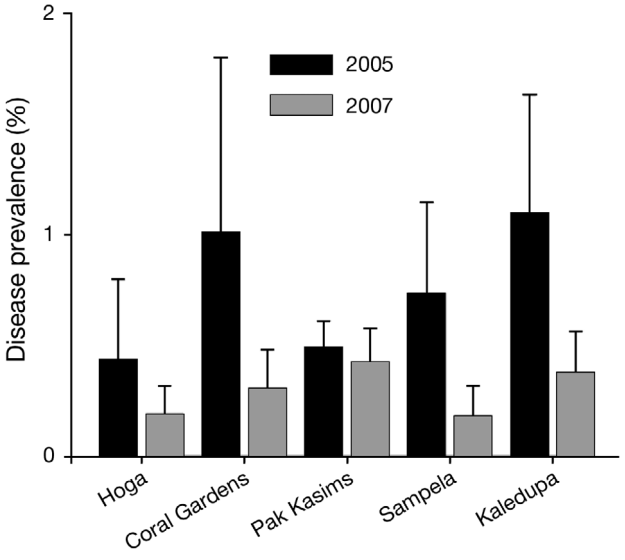

Fig. 2. Mean disease prevalence (± SE) for 5 sites (Kaledupa, Sampela, Hoga, Pak Kasims and Coral Gardens; see Fig. 1) in 2005 and 2007 in the WMNP

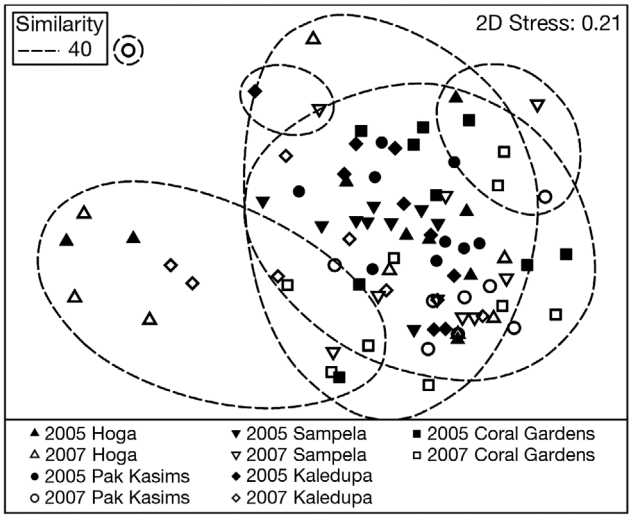

Fig. 3. Non-metric multi-dimensional scaling with superimposed Bray-Curtis similarity clusters (ovals) at the 40% similarity level illustrates the distribution of coral diseases within coral assemblages within each site in 2005 and 2007. Sampling used 5 sites: Kaledupa, Sampela, Hoga, Pak Kasims and Coral Gardens (see Fig. 1)

significant reef zone interaction with the presence of WS ($F_{20,60}$ = 2.49, p < 0.01) (Table 1) with a significant difference between Coral Gardens flat and slope and Hoga crest and slope in 2005, and between Hoga crest and flat and Sampela crest and flat in 2007. No significant differences were found between years or reef zones for the prevalence of growth anomalies (Table 1).

nMDS overlaying years and sites (Fig. 3) together with ANOSIM showed a significant difference in the distribution of diseases within the coral assemblage between years (R = 0.23, p < 0.001) and zones (R = 0.17, p < 0.001), and a weak significant difference between sites (R = 0.07, p < 0.01). The difference between zones is present at all sites (p < 0.001), whilst the difference (p < 0.05) between sites is only present between Hoga–Coral Gardens, Hoga–Sampela, Coral Gardens–Pak Kasims, and Coral Gardens–Sampela. SIMPER analysis found that the overall dissimilarity between the 2 years was mostly the result of differences in the abundance of disease on massive *Porites* (5.34% contribution), *Montipora* (4.97% contribution) and Dendrophyllids (4.68% contribution) (Table 2).

In 2005, 13 coral taxa were diseased compared to only 5 taxa in 2007 (Fig. 4). *Montipora* was the most common coral genus in both years and suffered very little from disease. WS was most prevalent on massive *Porites* in 2005 on Acroporids in 2007. PUWS was observed for the first time in 2007 (Fig. 4). Diseases were more common on flats and crests in 2005, but no clear link was found between disease prevalence and depth in 2007 (Fig. 5).

The *in situ* disease dynamics study of 4 diseases revealed that BrB and WS had the fastest progression rates (1.2 ± 0.36 cm d^{-1}, n = 4) and (1.1 ± 0.07 cm d^{-1}, n = 6) respectively (Table 3). BBD lesions progressed at the slowest rate (0.39 ± 0.14 cm d^{-1}, n = 21) (Table 3).

Table 1. Three-way nested permutational multivariate analysis of variance (PERMANOVA) for disease prevalence, white syndrome, growth anomalies and coral cover in the WMNP between 2005 and 2007 at 5 sites (Hoga, Sampela, Pak Kasims, Kaledupa and Coral Gardens; see Fig. 1), over 3 habitats (reef flat, slope and crest). *Significant differences are taken as those with a Monte Carlo (MC) permutational p-value < 0.05; NS: not significant

Source	df	Total disease prevalence		White syndrome		Growth anomalies		Coral cover	
		F	p (MC)	*F*	p (MC)	*F*	p (MC)	*F*	p (MC)
Year	1	4.84	<0.05*	10.02	<0.01*	0.78	NS	81.48	<0.0001*
Site (Year)	8	1.61	NS	1.53	NS	1.96	NS	6.89	<0.0001*
Reef zone (Year × Site)	20	2.35	<0.01*	2.49	<0.01*	1.22	NS	5.62	<0.0001*
Residual	60								
Total	89								

Table 2. SIMPER analysis (Primer v.6.1.5) to determine 4 most similar and dissimilar (decreasing similarity and dissimilarity from top to bottom) coral taxa between 2005 and 2007 in terms of abundance

Coral taxon	Average similarity 2005	Coral taxon	Average similarity 2007	Coral taxon	Average dissimilarity 2005/2007
Porites (massive)	28.37	*Porites* (massive)	38.29	*Porites* (massive)	5.34
Montipora	4.83	*Montipora*	2.80	*Montipora*	4.97
Favia/Favites/Montastrea	3.52	Dendrophyllids	2.46	Dendrophyllids	4.68
Agariciidae	2.49	*Porites* (branching)	1.07	*Favia/Favites/Montastrea*	3.95

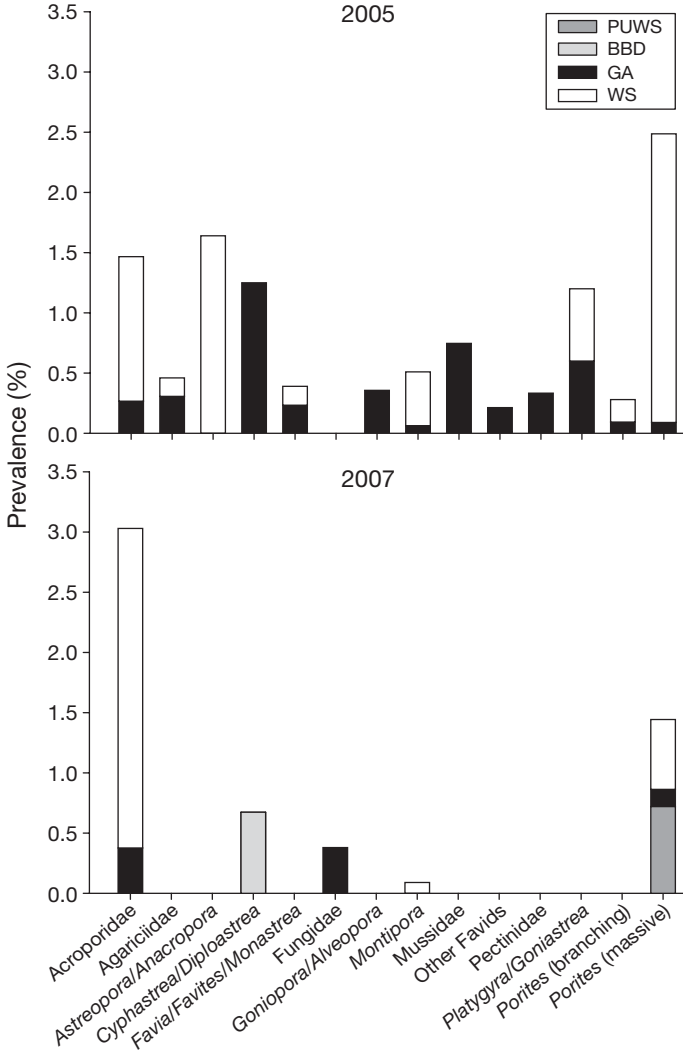

Fig. 4. Prevalence of 4 disease categories: white syndrome (WS), growth anomalies (GA), black band disease (BBD) and *Porites* ulcerative white spot syndrome (PUWS) in scleractinian taxa in 2005 and 2007. Prevalence (per taxa) is calculated relative to the total number of colonies examined in the respective taxa in each year

WS caused the greatest total tissue mortality; a total of 53 923 cm^2 over a period of 36 d and BBD the second most severe mortality; a total of 16 783 cm^2 over a period of 38 d.

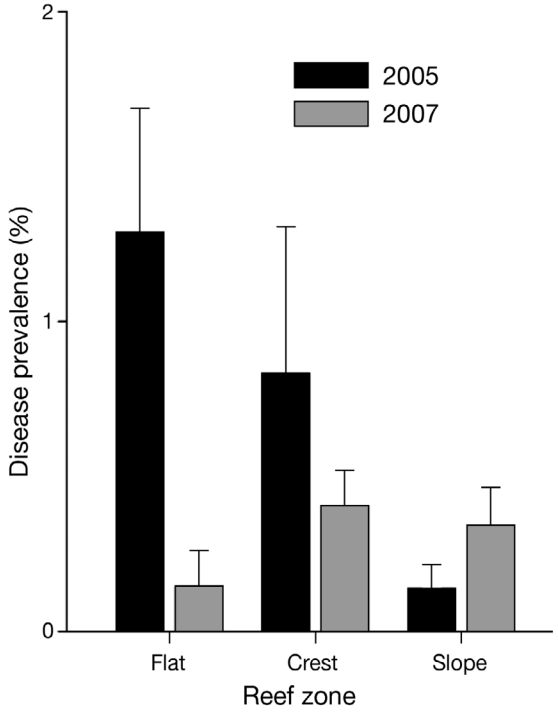

Fig. 5. Mean distribution of diseases per reef zone (± SE) for 5 sites (Kaledupa, Sampela, Hoga, Pak Kasims and Coral Gardens; see Fig. 1) in 2005 and 2007 in the WMNP

A highly significant drop in coral cover was observed between years ($F_{1,60}$ = 81.48, p < 0.0001), sites ($F_{8,60}$ = 6.89, p < 0.0001) and reef zones ($F_{20,60}$ = 5.62, p < 0.0001) (Table 1, Fig. 6). In 2005, all sites were significantly different except for Pak Kasims and Coral Gardens which had similar coral cover. There were no significant differences between sites in 2007. Blue Bowl, considered a pristine site and studied for the first time in 2007, had by far the highest coral cover (74.7%); it was not included in any of the PERMANOVAs, which included only the 5 sites of the inter-annual study. Amongst these sites, Hoga had the highest coral cover in both years (44.78% in 2005 and 24.46% in 2007) whereas Sampela had the lowest cover in 2005 (12.33%) and Kaledupa the lowest in 2007 (8.8%) (Fig. 6).

Table 3. Summary of coral disease progression rates. Where noted, error is SE. BBD: black band disease; WS: white syndrome; SEB: skeletal eroding band; BrB: brown band; WNMP: Wakatobi National Marine Park; GBR: Great Barrier Reef

Disease	Location	Coral host	Mean progression rate (cm d^{-1})[a]	Source
BBD	Blue Bowl, WNMP, Indonesia	*Pachyseris foliosa* (n = 19)	0.13 ± 0.02	Present study
BBD	Inner Pinnacle, WNMP, Indonesia	*Pachyseris rugosa* (n = 1)	0.63	Present study
BBD	Pak Kasims, WNMP, Indonesia	*Diploastrea heliopora* (n = 1)	0.42	Present study
BBD	GBR, Australia	*Acropora muricata*	0.41–0.99	Boyett et al. (2007)
BBD	W Caribbean	Up to 21 species	0.33–1	Summarized in Weil (2004)
WS	Pak Kasims, WNMP, Indonesia	*Acropora clathrata* (n = 1)	1.16	Present study
WS	Coral Gardens, WNMP, Indonesia	*Acropora clathrata* (n = 5)	1.03 ± 0.28	Present study
WS	Solitary Islands, Australia	*Acropora solitaryensis*	0.039–0.52	Dalton & Smith (2006)
White plague I	Florida, USA	Up to 21 species	0.31	Summarized in Weil (2004)
White plague II	W Caribbean	Up to 39 species	2	Richardson et al. (1998)
Yellow band	Curaçao, Caribbean	*Montastraea annularis*	0.6 cm mo^{-1}	Cervino et al. (2001)
Dark spot syndrome	Curaçao, Caribbean	*Siderastrea siderea*, *Stephanocoenia michelinii*	4 cm mo^{-1}	Cervino et al. (2001)
SEB	Hoga, WNMP, Indonesia	*Acropora pulchra* (n = 15)	0.5 ± 0.1	Present study
SEB	GBR, Australia	*Acropora muricata*	0.03–0.33	Page & Willis (2008)
BrB	Hoga, WNMP, Indonesia	*Acropora pulchra* (n = 4)	1.2 ± 0.36	Present study
BrB	GBR, Australia	*Acropora muricata*	max. 2.1 ± 0.35	Boyett (2006)

[a]Except where stated otherwise

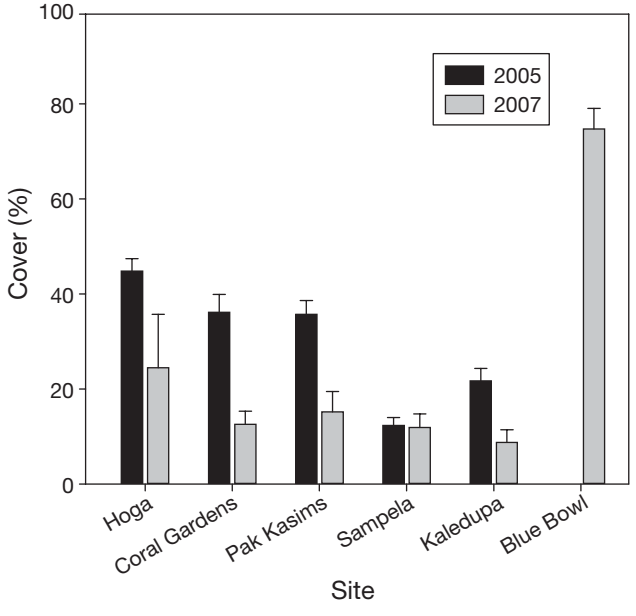

Fig. 6. Mean coral cover (± SE) for 6 sites (Kaledupa, Sampela, Hoga, Pak Kasims, Coral Gardens and Blue Bowl; see Fig. 1) in 2005 and 2007 in the WMNP. Blue Bowl was not part of the PERMANOVA

Sedimentation was significantly different between sites ($F_{5,38}$ = 27.00, p < 0.0001) and reef zones ($F_{12,38}$ = 2.04, p < 0.05). Blue Bowl (more than 15 mg cm^{-2} d^{-1}) was significantly different from all the other sites except for Sampela (11.5 mg cm^{-2} d^{-1} on the slope). All the other sites had a sedimentation rate of less than 5 mg cm^{-2} d^{-1} (Fig. 7).

DISCUSSION

Our study represents the first description of inter-annual coral disease dynamics, disease progression and tissue mortality in Indonesia. Inter-annual variability of diseases was high. There was a significant decrease in disease prevalence from 0.57% in 2005 to 0.33% in 2007. The number of diseased coral taxa decreased from 13 in 2005 to only 5 in 2007. The overall disease prevalence in the WMNP is low and similar to the 0.21% overall prevalence recently found in the remote Pacific islands (Vargas-Ángel 2009). Despite the observed decrease in prevalence, 2 new syndromes were observed in our 2007 surveys: PUWS and BBD. PUWS

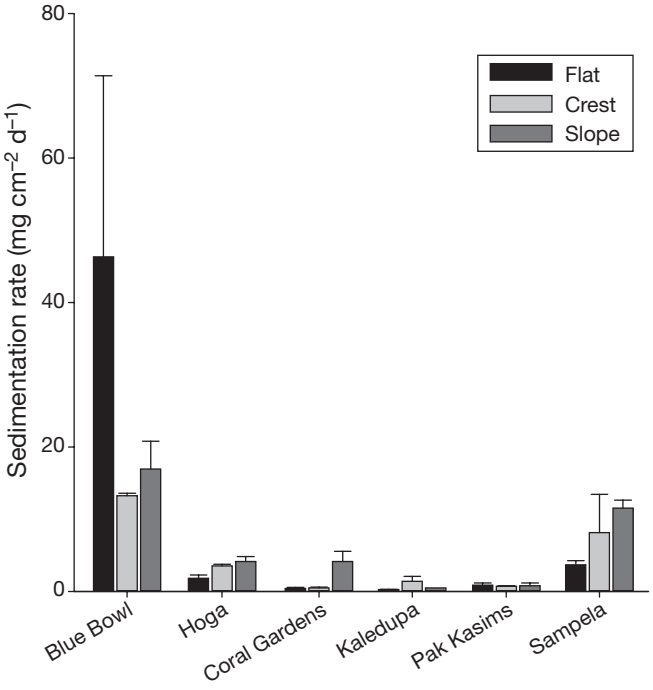

Fig. 7. Mean sedimentation rate (± SE) for 6 sites (Kaledupa, Sampela, Hoga, Pak Kasims, Coral Gardens and Blue Bowl; see Fig. 1) in 2005 and 2007 in the WMNP

has previously been detected in the Philippines where it had a high prevalence (Raymundo et al. 2003, 2005).

Of the diseases found in the present study, PUWS, BrB, SEB and WS (which is a collective term for Indo-Pacific white diseases), are found only in the Indo-Pacific (Raymundo et al. 2003, Willis et al. 2004), whereas GA and BBD are both found globally (Sutherland et al. 2004).

This study reveals for the first time the presence of BrB in Indonesia. SEB was found outside the study area in 2005 (Haapkylä et al. 2007). BrB and SEB are both characterised by dense aggregations of ciliates on the surface of the coral: SEB harbouring *Halofolliculina corallasia* (Antonius & Lipscomb 2001) and BrB a ciliate belonging to the class Oligohymenophorea, subclass Scuticociliata (Bourne et al. 2008). The prevalence of BrB on the GBR is less than 1% and more common on the southern GBR (Willis et al. 2004). SEB was the most prevalent coral disease on the GBR in 2004 to 2006 accounting for 40 to 60% of disease cases recorded in each year (Page & Willis 2008). Moreover, it was found affecting 38% of corals in the Red Sea (Winkler et al. 2004). Another ciliate of the genus *Halofolliculina* was found from the Caribbean (Cróquer et al. 2006a), although it appears to be a different species to the one found in the Indo-Pacific (Cróquer et al. 2006b). Therefore SEB is considered an Indo-Pacific disease and its Caribbean variation is called Caribbean ciliate infection (Rodríguez et al. 2009).

Our results show a significantly lower prevalence of WS in 2007 but no significant difference in GA prevalence. In 2007, diseases were less common in shallow reef zones than in 2005. Only one previous study on depth and disease prevalence exists from the Indo-Pacific where Raymundo et al. (2003) found that PUWS prevalence was not depth dependent in the Philippines. In the Caribbean white plague-infected corals were most common between 8 and 18 m depth (Dustan 1977). A similar depth pattern was found in Venezuela (Cróquer et al. 2003).

There was a highly significant drop in coral cover at all sites in the WMNP between 2005 and 2007 (Fig. 3, Table 1). Kaledupa, considered a pristine site in 2005, had the lowest coral cover of all sites (8.8%) whereas Blue Bowl, dominated by foliaceous corals, was studied for the first time in 2007 and had the highest coral cover (74.7%). The overall decrease in coral cover may predominantly be due to anthropogenic exploitation (McMellor 2007, 2008). Regular monitoring of coral disease could reveal the potential influence of localized coral disease outbreaks on the decline in coral cover.

The major driver of change in the distribution of disease in the coral assemblage between 2005 and 2007 is, according to the SIMPER analysis (Table 2), the differences in the abundance of diseases on massive *Porites* (5.34% contribution), *Montipora* (4.97% contribution) and Dendrophyllids (4.68% contribution). *Porites* and *Montipora* are the 2 major coral genera in the area. Massive *Porites* was the only taxa showing symptoms of 3 syndromes (PUWS, GA and WS). It is a dominant component of Indo-Pacific reefs. Diseases could potentially have larger impacts on massive *Porites* and the reef structure because of its slow growth rates (around 1 cm yr^{-1}) (Pätzold 1984). In 2007, acroporids represented only 4.6% of the total number of corals but they were found to be the most diseased coral taxa (3% of all acroporids were diseased). Acroporids are subject to a number of coral diseases such as BBD (Page & Willis 2006), BrB (Willis et al. 2004), SEB (Page & Willis 2008) on the GBR and white band (Aronson & Precht 2001) and white pox (Patterson et al. 2002) diseases in the Caribbean. However, fast growth rates of *Acropora* may compensate for the mortality. Yap & Gomez (1985) recorded a growth rate of 0.3 to 2.3 cm mo^{-1} for *A. pulchra* in the Philippines.

A positive relationship between host density and disease prevalence has been clearly demonstrated in many host–pathogen systems (Anderson & May 1979, Altizer et al. 2003, Lafferty 2004, Rudolf & Antonovics 2005), and is considered a hallmark of the infectious process (Lafferty & Gerber 2002). Host density is most often associated with greater rates of horizontal transmission (Getz & Pickering 1983, Holt & Pickering 1985,

Altizer & Augustine 1997), leading to localized increase in prevalence. In addition, host density can be positively related to the density of disease vectors (Rosenberg & Falkovitz 2004, Williams & Miller 2005). High coral cover has been linked to a high prevalence of WS on the Great Barrier Reef of Australia (Bruno et al. 2007). High cover of *Pachyseris foliosa* in the Blue Bowl may have facilitated the spread of BBD infections.

Sedimentation may also be a driver of coral disease. Voss & Richardson (2006) observed a link between high sedimentation rate and BBD in the Caribbean and proposed that sediments may act as vectors of coral disease. Sedimentation rates of <10 mg cm^{-2} d^{-1} are typical for reefs not subject to human disturbance (Rogers 1990); the rates obtained in our study were within this range except for the Blue Bowl, a pristine site, where the rate exceeded 15 mg cm^{-2} d^{-1} (over 40 mg cm^{-2} d^{-1}on the flat). The bowl-like topography of the Blue Bowl and the close proximity of a large sandy reef flat may have enhanced the accumulation of sediments at the site. High sedimentation rate in the Blue Bowl may have contributed to the occurrence of BBD on the slope at this site.

Warmer water temperatures have been linked to higher disease prevalence and progression rates in diseases such as WS (Willis et al. 2004, Bruno et al. 2007) and BBD (Boyett et al. 2007). Prevalence, progression rates and tissue mortality due to coral disease may have been higher at the study sites in the warmer wet season when water temperatures exceed 30°C. As a consequence, further studies in the wet season are needed to evaluate seasonal ranges in disease prevalence and dynamics in the WMNP.

Our study of disease progression rates and tissue mortality accentuates the fact that conclusions about impacts of coral disease should not solely be based on the results obtained from transect monitoring. SEB, WS and BBD all had faster progression rates in the WMNP than those reported from the GBR (Table 3). In our study, BrB (1.2 ± 0.36 cm d^{-1}) had the highest progression rate and WS (1.1 ± 0.07 cm d^{-1}) the second highest. The fastest progression rates in the Caribbean were recorded for WPII infections with similar progression rates to BrB on the GBR where the progression rate of BrB was up to 2.1 cm d^{-1} in the austral summer (Boyett et al. 2007).

In this study, WS caused more severe total tissue loss (53 923 cm^2) than BBD (16 783 cm^2). BrB was observed only for 5 d compared to 36 d for WS and 38 d for BBD which resulted in less tissue mortality through time despite its fast progression rate. Coral diseases may have important impact locally in re-structuring reefs by impacting key reef-building corals.

In conclusion, despite a low overall disease prevalence, we documented fast coral disease progression rates and high tissue mortality rates for coral diseases in the WMNP; our research suggests that coral diseases may contribute to the decline of coral cover in this region. Further effort should be dedicated to understanding coral disease dynamics in Indonesia to better inform management and conservation approaches for reef ecosystems in this hotspot of biodiversity.

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LITERATURE CITED

Aeby GS (2005) Outbreak of coral diseas in the Northwestern Hawaiian islands. Coral Reefs 24:481

Allen GR (2007) Conservation hotspots of biodiversity and endemism for Indo-Pacific coral reef fishes. Aquat Conserv Mar Freshw Ecosyst 18:541–556

Altizer SM, Augustine DJ (1997) Interactions between frequency-dependent and vertical transmission in host–parasite systems. Proc R Soc Lond B Biol Sci 264:807–814

Altizer S, Nunn CL, Thrall PH, Gittleman JL and others (2003) Social organization and parasite risk in mammals: integrating theory and empirical studies. Annu Rev Ecol Evol Syst 34:517–547

Anderson MJ (2001) A new method for non-parametric multivariate analysis of variance. Austral Ecol 26:32–46

Anderson RM, May RM (1979) Population biology of infectious diseases: Part I. Nature 280:361–367

Antonius A, Lipscomb D (2001) First protozoan coral-killer identified in the Indo-Pacific. Atoll Res Bull 481–493:1–21

Aronson RB, Precht WF (2001) White-band diseases and the changing face of Caribbean coral reefs. Hydrobiologia 460:25–38

Bourne DG, Boyett HV, Henderson ME, Muirhead A, Willis BL (2008) Identification of a ciliate (Oligohymenophorea: Scuticociliatia) associated with brown band disease on corals of the Great Barrier Reef. Appl Environ Microbiol 74:883–888

Boyett HV (2006) The ecology and microbiology of black band disease and brown band syndrome on the Great Barrier Reef. Master's thesis, James Cook University, Townsville

Boyett HV, Bourne DG, Willis BL (2007) Elevated temperature and light enhance progression and spread of black band disease on staghorn corals of the Great Barrier Reef. Mar Biol 151:1711–1720

Bruckner AW, Bruckner RJ (1997) The persistence of black band disease in Jamaica: impact on community structure. Proc 8th Int Coral Reef Symp 1:601–606

Bruno JF, Petes LE, Harvell CD, Hettinger A (2003) Nutrient enrichment can increase the severity of coral diseases. Ecol Lett 6:1056–1061

Bruno JF, Selig ER, Casey KS, Page CA and others (2007) Thermal stress and coral cover as drivers of coral disease outbreaks. PLoS Biol 5:e124, doi:10.1371/journal.pbio.0050124

Cervino J, Goreau TJ, Nagelkerken I, Smith GW, Hayes R (2001) Yellow band and dark spot syndromes in Caribbean corals: distribution, rate of spread, cytology and effects on abundance and division rate of zooxanthellae. Hydrobiologia 460:53–63

Clarke KR, Warwick RM (2001) Change in marine communities: an approach to statistical analysis and interpretation, 2nd edn. PRIMER-E, Plymouth

Cróquer A, Pauls SM, Zubillaga AL (2003) White plague disease outbreak in a coral reef at Los Roques National Park, Venezuela. Rev Biol Trop 51:39–45

Cróquer A, Bastidas C, Lipscomb D (2006a) Folliculinid ciliates: a new threat to Caribbean corals? Dis Aquat Org 69:75–78

Cróquer A, Bastidas C, Lipscomp D, Rodríguez-Martínez RE, Jordan-Dahlgren E, Guzman HM (2006b) First report of folliculinid ciliates affecting Caribbean scleractinian corals. Coral Reefs 25:187–191

Dalton SJ, Smith SDA (2006) Coral disease dynamics at a subtropical location, Solitary Islands Marine Park, eastern Australia. Coral Reefs 25:37–45

Dorland RB (1982) The protective mechanism of action of amines in diphtheria-toxin treated vero cells. Can J Microbiol 28:611–617

Dustan P (1977) Vitality of reef coral populations off Key Largo, Florida: recruitment and mortality. Environ Geol Water Sci 2:51–58

English S, Wilkinson C, Baker V (1997) Survey manual for tropical marine resources, 2nd edn. Australian Institute of Marine Science, Townsville

Epstein PR, Sherman K, Spanger-Siegfried E, Langston A, Prasad S, McKay B (1998) Marine ecosystems: emerging diseases as indicator of change. Health Ecological and Economic Dimensions (HEED), NOAA Global Change Program, Washington, DC

Getz WM, Pickering J (1983) Epidemic models—thresholds and population regulation. Am Nat 121:892–898

Gladfelter WB (1982) White-band disease in *Acropora palmata*: implications for the structure and growth of shallow reefs. Bull Mar Sci 32:639–643

Green EP, Bruckner AW (2000) The significance of coral disease epizootiology for coral reef conservation. Biol Conserv 96:347–361

Haapkylä J, Seymour AS, Trebilco J, Smith D (2007) Coral disease prevalence and coral health in the Wakatobi Marine Park, South-East Sulawesi Indonesia. J Mar Biol Assoc UK 87:403–414

Harvell CD, Mitchell CE, Ward JR, Altizer S, Dobson AP, Ostfeld RS, Samuel MD (2002) Climate warming and disease risks for terrestrial and marine biota. Science 296: 2158–2162

Hayes RL, Goreau NI (1998) The significance of emerging diseases in the tropical coral reef ecosystem. Rev Biol Trop 46:173–185

Holt RD, Pickering J (1985) Infectious disease and species coexistence — a model of Lotka-Volterra form. Am Nat 126:196–211

Hughes TP (1994) Catastrophes, phase-shifts and large-scale degradation of a Caribbean coral reef. Science 265: 1547–1551

Kaczmarsky LT (2006) Coral disease dynamics in the central Philippines. Dis Aquat Org 69:9–21

Lafferty KD (2004) Fishing for lobsters indirectly increases epidemics in sea urchins. Ecol Appl 14:1566–1573

Lafferty KD, Gerber LR (2002) Good medicine for conservation biology: the intersection of epidemiology and conservation theory. Conserv Biol 16:593–604

Lafferty KD, Holt RD (2003) How should environmental stress affect the population dynamics of disease? Ecol Lett 6: 654–664

Lafferty KD, Porter JW, Ford SE (2004) Are diseases increasing in the ocean? Annu Rev Ecol Evol Syst 35:31–54

McArdle BH, Anderson MJ (2001) Fitting multivariate models to community data: a comment on distance-based redundancy analysis. Ecology 82:290–297

McMellor S (2007) A Conservation Value Index to facilitate coral reef evaluation and assessment. PhD thesis, University of Essex, Colchester

McMellor S (2008) Reef status in the Wakatobi Marine National Park, Indonesia 2002–2007. In: Wilkinson C (ed) Status of coral reefs of the world: 2008. Global Coral Reef Monitoring Network and Reef and Rainforest Research Centre, Townsville

Nugues MM (2002) Impact of a coral disease outbreak on coral communities in St. Lucia: What and how much has been lost? Mar Ecol Prog Ser 229:61–71

Page C, Willis B (2006) Distribution, host range and large-scale spatial variability in black band disease prevalence on the Great Barrier Reef, Australia. Dis Aquat Org 69: 41–51

Page CA, Willis BL (2008) Epidemiology of skeletal eroding band on the Great Barrier Reef and the role of injury in the initiation of this widespread coral disease. Coral Reefs 27:257–272

Patterson KL, Porter JW, Ritchie KB, Polson SW and others (2002) The etiology of white pox, a lethal disease of the Caribbean Elkhorn coral, *Acropora palmata*. Proc Natl Acad Sci USA 99:8725–8730

Pätzold J (1984) Growth rhythms recorded in stable isotopes and density bands in the reef coral *Porites lobata* (Cebu, Philippines). Coral Reefs 3:87–90

Pet-Soede L, Erdmann M (eds) (2004) Rapid Ecological Assessment Wakatobi National Park. November 2003. Report from WWF Indonesia Marine Program, Denpasar, Bali

Raymundo LJH, Harvell CD, Reynolds TL (2003) *Porites* ulcerative white spot disease: description, prevalence, and host range of a new coral disease affecting Indo-Pacific reefs. Dis Aquat Org 56:95–104

Raymundo LJ, Rosell KB, Reboton CT, Kaczmarsky L (2005) Coral diseases on Philippine reefs: genus *Porites* is a dominant host. Dis Aquat Org 64:181–191

Richardson LL, Voss JD (2005) Changes in a coral population on reefs of the northern Florida Keys following a coral disease epizootic. Mar Ecol Prog Ser 297:147–156

Richardson LL, Goldberg WM, Kuta KG, Aronson RB and others (1998) Florida's mystery coral-killer identified. Nature 392:557–558

Roberts CM, McClean CJ, Veron JEN, Hawkins JP and others (2002) Marine biodiversity hotspots and conservation priorities for tropical reefs. Science 295:1280–1284

Rodríguez S, Cróquer A, Guzmán HM, Bastidas C (2009) A mechanism of transmission abnd factors affecting coral susceptibility to *Halofolliculina* sp. infection. Coral Reefs 28:67–77

Rogers CS (1990) Responses of coral reefs and reef organisms to sedimentation. Mar Ecol Prog Ser 62:185–202

Rosenberg E, Falkovitz L (2004) The *Vibrio shiloi/Oculina patagonica* model system of coral bleaching. Annu Rev Microbiol 58:143–159

Rudolf VHW, Antonovics J (2005) Species coexistence and pathogens with frequency-dependent transmission. Am Nat 166:112–118

Sutherland KP, Porter JW, Torres C (2004) Disease and immunity in Caribbean and Indo-Pacific zooxanthellate corals.

Mar Ecol Prog Ser 266:273–302

Turak E (2003) Coral reef surveys during TNC SEACMPA RAP of Wakatobi National Park, Southeast Sulawesi. Final Report to The Nature Conservancy, Bali

Unsworth RKF, Smith DJ, Powell A, Hukon F (2007) The ecology of Indo-Pacific grouper (Serranidae) species and the effects of a small scale no take area on grouper assemblage, abundance and size frequency distribution. Mar Biol 152:243–254

Vargas-Ángel B (2009) Coral health and disease assessment in the U.S. Pacific remote island areas. Bull Mar Sci 84:211–227

Voss JD, Richardson LL (2006) Coral diseases near Lee Stocking Island, Bahamas: patterns and potential drivers. Dis Aquat Org 69:33–40

Wallace CC (1978) The coral genus *Acropora* (Scleractinia:Astocoeniina:*Acropora*) in the central and southern Great Barrier Reef Province. Mem Queensl Mus 18: 273–319

Weil E (2004) Coral reef diseases in the wider Caribbean. In: Rosenberg E, Loya Y (eds) Coral health and disease. Springer-Verlag, Berlin, p 35–68

Weil E, Urreiztieta I, Garzón-Ferreira J (2002a) Geographic variability in the prevalence of coral and octocoral disease in the wider Caribbean. Proc 9th Int Coral Reef Symp 2:1231–1238

Weil E, Smith G, Gil-Agudelo DL (2006) Status and progress in coral reef disease research. Dis Aquat Org 69:1–7

Wilkinson C (ed) (2008) Status of coral reefs of the world: 2008. Global Coral Reef Monitoring Network and Reef and Rainforest Research Centre, Townsville

Williams DE, Miller MW (2005) Coral disease outbreak: pattern, prevalence and transmission in *Acropora cervicornis*. Mar Ecol Prog Ser 301:119–128

Willis BL, Page CA, Dinsdale AD (2004) Coral disease on the Great Barrier Reef. In: Rosenberg E, Loya Y (eds) Coral health and disease. Springer-Verlag, Berlin, p 69–104

Winkler R, Antonius A, Renegar DA (2004) The skeleton eroding band disease on coral reefs of Aqaba, Red Sea. PSZN I: Mar Ecol 25:129–144

Woodley JD (1989) The effects of Hurricane Gilbert on coral reefs at Discovery Bay. In: Bacon PR (ed) Assessment of the economic impacts of Hurricane Gilbert on coastal and marine resources in Jamaica. UNEP-Regional Seas Rep Stud 110, United Nations Environment Programme, Nairobi, p 71–73

Work TM, Aeby G (2006) Systematically describing gross lesions in corals. Dis Aquat Org 70:155–160

Yap HT, Gomez ED (1985) Growth of *Acropora pulchra* III. Preliminary observations on the effects of transplantation and sediment on the growth and survival of transplants. Mar Biol 87:203–209

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