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Distribution and drivers of coral disease at Ningaloo reef, Indian Ocean

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ABSTRACT: We investigated the prevalence and potential drivers of coral disease across Ningaloo Reef on the Western Australian coast. Coral disease assessment surveys were undertaken at 2 spatial scales, the first over a small area of reef (Bill's Bay, 2.5 × 5.0 km), where human use is high and where several anoxic events have caused significant coral mortality, and the second over a broader area (spanning ~200 km of the Ningaloo coast). Throughout Ningaloo, 2.3% of coral colonies showed signs of disease, although disease prevalence varied at both broad and local scales, ranging from 1.1 to 7% along the coast, and from 0.1 to 3.1% locally—all within the range of values recorded in other Indo-Pacific regions. Seven diseases were identified, the most common being 'skeletal eroding band' (which affected ~1% of colonies). At a broad spatial scale, prevalence of skeletal eroding band was positively related to the number of coral colonies exhibiting *Drupella* spp. feeding scars, whilst black-band disease (BBD) was positively associated with density of coral colonies. At the local scale, severity of anoxic events and occurrence of *Drupella* spp. feeding scars were positively related to prevalence of BBD, whilst other cyanobacterial bands were associated only with *Drupella* spp. scars. We saw no strong indication that human activities, measured as density of people or vessels in the water, were related to disease prevalence. Positive relationships amongst disease, anoxic events and *Drupella* spp. feeding suggest that natural stressors are potential drivers of disease at Ningaloo.

KEY WORDS: Ningaloo Marine Park · Coral Bay · *Drupella* · Climate change · Coral disease

INTRODUCTION

Coral reefs worldwide are facing increasing pressure from overfishing, reduced water quality and a range of threats associated with climate change (Hughes et al. 2003, Wilkinson 2008). Disease has recently emerged as a serious threat to coral reefs, having contributed significantly to declines in hard and soft corals and to dramatic changes in the composition of coral communities (Hughes 1994, Aronson & Precht 2001, Kim & Harvell 2004). The Caribbean is recognised as a 'hot spot' for both coral disease and disease research, and until recently, disease was believed to be less severe in the Indo-Pacific (Green & Bruckner 2000, Harvell et al. 2007). More recent research, however, has indicated that coral disease may be a more important factor in coral loss on Indo-Pacific reefs than previously acknowledged (Goldberg & Wilkinson 2004, Willis et al. 2004, McClanahan et al. 2009).

Very little is known about diseases affecting corals in the Indian Ocean (reviewed in Sutherland et al. 2004, Willis et al. 2004), particularly those corals in the eastern Indian Ocean and along the West Australian coast (Table 1). The few studies undertaken in the eastern Indian Ocean include an assessment of white syndrome on Christmas Island and in the Cocos (Keeling)

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Islands (Hobbs & Frisch 2010), and observations of disease off the Pilbara coast (Page & Stoddart 2010). Unpublished records of disease also exist for the Abrohlos Islands south of Ningaloo Reef (L. Smith pers. comm.) and the Rowley Shoals (S. Long pers. comm.). Clearly, greater efforts are required to understand the diseases impacting corals in the eastern Indian Ocean and in Western Australia.

Ningaloo Reef, which stretches over 300 km along the Western Australian coast, is Australia's largest fringing coral reef (Department of Conservation and Land Management 2005). The reef, while reserved as a marine park, is subject to natural pressures, including outbreaks of corallivorous *Drupella* spp. gastropods and anoxic water events. Densities of *D. cornus*, for example, have been unusually high at Ningaloo and, in the 1980s, caused a reduction in coral cover of up to 85% at some sites (Ayling & Ayling 1987). Bill's Bay, in the southern section of the reef, has been subject to several natural anoxic events associated with coral

Table 1. Prevalence of coral disease on Indo-Pacific reefs. Only studies in which health of all corals was assessed and disease prevalence was calculated as the percentage of coral colonies affected by disease are included. Disease prevalence presented for all diseases, unless disease type is specified. Sp: Spring, Su: Summer, A: Autumn, W: Winter. NS: not specified

Location	Year of study	Study season	Disease prevalence (% coral colonies)	Study area (no. sites × area in m^2)	Source
All diseases					
Ningaloo Reef, Australia	2009–2010	A, Su	2.3 (0.1 to 7.0)	28 × 120	Present study
Great Barrier Reef—Heron Is., Lizard Is., No Name Reef; Australia	2003	Su	9.0 (7.2 to 10.7)	8 × 120	Willis et al. (2004)
Great Barrier Reef—Heron Is., Australia	2007–2009	Su, W	1.9 to 4.2	645 m^2 to 720 m^2	Haapkylä et al. (2009)
Solitary Islands Marine Park, Australia	2003–2004	A, W, Sp	9.0	6 × 113	Dalton and Smith (2006)
Wakatobi Marine National Park, Sulawesi, Indonesia	2005–2007	W, Sp	0.3 to 0.6	5 × 240	Haapkylä et al. (2009)
Central Visayas & Lingayen Gulf, Philippines	2003	Sp	8.3	8 × 120	Raymundo et al. (2005)
Central Visayas, Philippines	2006	Sp, Su	0.3 to 7.9	14 × 120	Raymundo et al. (2009)
Guam	NS	NS	0.2 to 12.6	15 × 120	Myers & Raymundo (2009)
Guam & Commonwealth of the Northern Mariana Islands	2006–2007	NS	2.4	66 × 100–300	Vargas-Angel & Wheeler (2008)
American Samoa	2006–2007	NS	3.9	62 × 100–300	Vargas-Angel & Wheeler (2008)
American Samoa	2004–2005	Su, W	0.18	7 × 50–100	Aeby et al. (2008)
Palau	2005	W	2.1	8 × 120	Page et al. (2009)
Northwestern Hawaiian Islands,USA	2006–2007	NS	2.0	64 × 100–300	Vargas-Angel & Wheeler (2008)
Pacific Remote Island Areas, USA	2006–2007	NS	2.0	80 × 100–300	Vargas-Angel & Wheeler (2008)
Main Hawaiian Islands, USA	2006–2007	NS	0.9	54 × 100–300	Vargas-Angel & Wheeler (2008)
Northwestern Hawaiian Islands, USA	2003	Su	0.5	73 × 100	Aeby (2006)
Northern Line Islands	2005	A	2.5 to 6.3	10–12 × 80	Dinsdale et al. (2008)
Palmyra Atoll, Line Islands	2007	NS	0.39	58 × 50	Williams et al. (2007)
Specific diseases					
Christmas Island, Australia	2008	Su, A	White syndrome: 13.0	10 × 450	Hobbs & Frisch (2010)
Cocos Islands, Australia	2008	Su, A	White syndrome: 0.9	9 × 450	Hobbs & Frisch (2010)
Central Visayas, Philippines	2002–2003	A, W, Sp, Su	*Porites* ulcerative white spot disease: 7.5 (max. 53.7) Tumor: 10.3 (max. 39.1) Black band disease: max. 7.8	28 × 120	Kaczmarsky (2006)
Great Barrier Reef—Lizard Is.; Australia	1994	Su	Black band disease: 2.8 (1.3 to 4.9)	4 × 70–100	Dinsdale (2000)
Great Barrier Reef—Heron Is., Lizard Is., No Name Reef; Australia	2004	Su, A	Black band disease: 0 to 0.7	19 × 120	Page & Willis (2006)
Great Barrier Reef, Australia	2004–2006	Su	Skeletal eroding band: 1.2 to 2.3	18 × 120	Page & Willis (2008)
Gulf of Aqaba, Red Sea	2000	Su	Skeletal eroding band: 29.0	4 × 3	Winkler et al. (2004)
Gulf of Mannar, India	2003	W, Sp	Red band disease: 19.6	5 × 1425	Chellaram et al. (2004)
Northern Persian Gulf	2000	NS	Yellow band disease: <5.0	Unspecified	M. R. Shokri Bousjein[a]
Main Hawaiian Islands, USA	2007	Su	Skeletal eroding band: 0 to 1.1	9 × 120	Palmer & Gates (2010)

[a]Data cited as pers. comm. in Goldberg & Wilkinson (2004)

spawning. Anoxic events occur when atypically calm wind and sea conditions coincide with mass coral spawning, and the respiratory demand of the spawn slick deplete available oxygen in the water column and sediments (Simpson et al. 1993). The first recorded event in 1989 led to extensive mortality of corals in the inner bay, with several similar though less devastating events occurring in 2002, 2005 and 2008 (Simpson et al. 1993, van Schoubroeck & Long 2007, Halford & Perret 2009). Ningaloo is a popular destination for tourists, and, although townships along the Ningaloo coast are small (<2000 people), activity in the marine park can be high (~180 000 visitors in 2008; Jones et al. 2009). Since the intensity of both natural and anthropogenic stressors varies along Ningaloo Reef, this region is optimal for investigating the impacts of these factors on coral disease levels.

The present study aimed to quantify the prevalence of diseases affecting coral taxa across Ningaloo Reef at both reef-wide and local scales. To improve our understanding of reef interactions at Ningaloo, and to guide conservation management of coral reefs in general, we also investigated the potential drivers of disease.

To provide context to our findings we include a summary (Table 1) of disease prevalence from other studies in the Indo-Pacific region, focusing on those for which all corals were assessed for disease presence.

MATERIALS AND METHODS

Study area. Surveys were conducted within the Ningaloo Marine Park and Muiron Islands Marine Management Area off the coast of Western Australia. Ningaloo Reef is a fringing barrier coral reef, enclosing a shallow lagoon, that extends ~300 km south from the Muiron Islands (Fig. 1a). Prevalence of disease was first assessed within Bill's Bay (23.14° S, 113.76° E), a focal point for tourist activity and an area where anoxic events have caused coral mortality (Simpson et al. 1993). In May 2009, 13 sites were surveyed within Bill's Bay, adjacent to the town of Coral Bay. Sites were aligned in a grid-like pattern to enable sampling of the inner, middle and outer regions of the approx. 2.5 × 5 km bay (Fig. 1b). In January 2010, variation in disease prevalence was assessed at a broader spatial scale along ~200 km of the Ningaloo coast. Fifteen sites were surveyed between the Muiron Islands (21.66° S, 114.37° E) in the north and the Pelican Sanctuary Zone (23.33° S, 113.78° E) in the south (Fig 1a). All sites were located in shallow (2 to 4 m) water within the lagoon or back reef.

Survey design. At each site, three 20 × 2 m belt transects (from 5 to 40 m apart) were surveyed in accordance with standard protocols (Weil et al. 2008). Every coral colony within each belt transect was examined and categorised as healthy, bleached and/or traumatised or as being affected by a specific disease category based on Raymundo et al. (2008). Each coral was identified to the lowest taxonomic level practicable. *Acropora* spp. and *Porites* spp. were further separated into morphological groups based on Veron (2000). Lesions that were explainable by field signs (e.g. feeding scars from *Drupella* spp. or other predators, and lesions caused by competitive interactions or physical damage) were also recorded.

Data analysis. Because no disease cases were recorded from non-scleractinian corals, these corals were excluded from further calculations and analyses of disease prevalence. The distribution of disease across coral taxa and morphologies within the most abundant coral taxa was investigated using chi-squared tests for homogeneity. Data for all sites, including surveys of both Bill's Bay in 2009 and Ningaloo Reef in 2010, were pooled for this analysis.

Variation in the prevalence of disease at both broad and local scales was examined using 1-way ANOVA. Disease prevalence data were square-root transformed to meet the assumptions of normality (assessed using the Kolmogorov-Smirnov test) and homoscedasticity (assessed using Cochrane's test). Fisher's post hoc tests were used to identify sites with significantly different ($p < 0.05$) disease prevalence.

The relationship between disease prevalence and potential drivers of disease was examined using best-subset analysis, which considers and compares all possible combinations of predictor variables. Analyses were carried out using prevalence of all diseases, as well as the 3 most common diseases at Ningaloo—viz. skeletal eroding band, black band disease and non-black cyanobacterial band—as dependent variables. Disease prevalence was expressed as the mean percentage of coral colonies affected by disease per site (number of diseased coral colonies/total number of coral colonies × 100). For both the broad- and local-scale spatial analyses, predictor variables considered were (1) the abundance of scleractinian colonies at each site, as disease occurrence is commonly related to density of hosts (e.g. Anderson & May 1979, Hobbs & Frisch 2010, Williams et al. 2010), and (2) the number of colonies with *Drupella* spp. feeding scars, as coral disease has previously been linked to *Drupella* spp. (Antonius & Reigl 1997) and these corallivores have been a major cause of coral loss at Ningaloo (Ayling & Ayling 1987, Forde 1994, Osborne & Williams 1995). The broad-scale analysis also investigated relationships between disease and human use patterns across Ningaloo, as humans may act as disease vectors, or their activities may damage corals, leaving them susceptible to disease (summarised in

Page et al. 2009). Moreover, if disease is related to human activities, appropriate management can alleviate this pressure and may limit disease occurrence. An index of human use for each site was calculated as counts of both people and vessels within different management zones of the marine park. We present these as separate indices, although they were collected simultaneously from aerial surveys undertaken 2 to 4 times annually between 2005 and 2009 by the Department of Environment and Conservation's Exmouth District. Data from the relevant management zones were used to calculate mean number of people and vessels per flight per 100 ha. At the local scale we also investigated whether sites that had been repeatedly stressed by anoxic events at Bill's Bay (in 1989, 2002, 2005 and 2008) had a higher prevalence of coral disease. To assess spatial variation in coral mortality following anoxic events, each site was assigned a disturbance score between 1 and 0 where scores of 1 represented extensive coral bleaching and mortality, whilst scores of 0 represented negligible effect. Scores from each of the 4 anoxic events occurring between 1989 and 2008 were tallied to provide an index of cumulative anoxic event effect at each site (van Schoubroeck & Long 2007).

Models for predicting the prevalence of each disease were compared using Akaike's Information Criterion (AIC) and Akaike's weight (AICw), with the best models being those with the lowest AIC value and least number of variables. Models were considered to be significantly different when they differed by >2 AIC units. Support for single predictor variables was assessed by summing AICw across models containing that variable (Burnham & Anderson 2002).

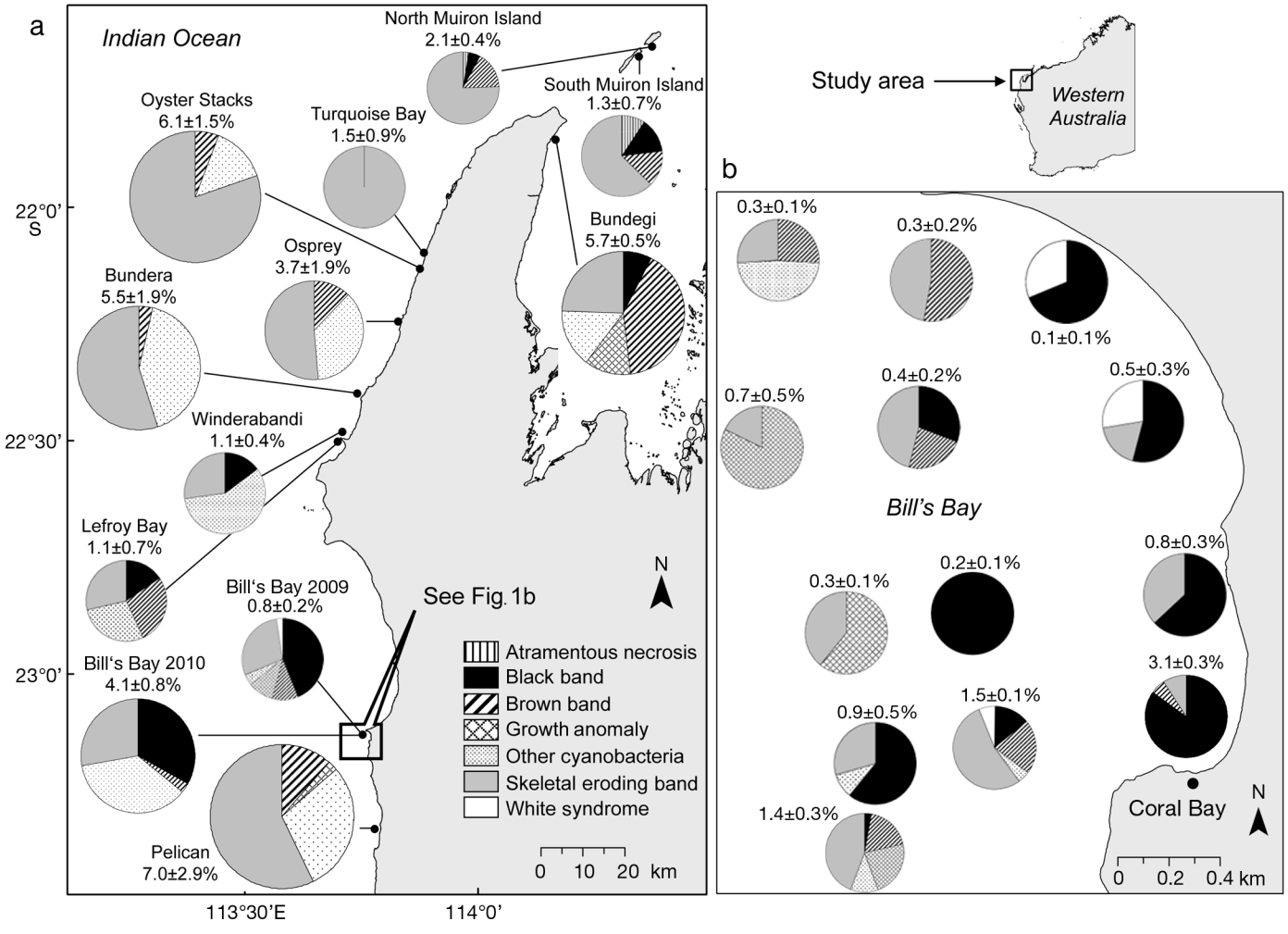

Fig. 1. Prevalence of disease in corals across (a) Ningaloo Reef in January 2010 and (b) Bill's Bay in May 2009. Disease prevalence (%) expressed as mean ± SE of diseased colonies, calculated from 3 transects per site. In (a), size of pie graphs represents relative prevalence of diseased corals across sites and mean prevalence was calculated for Bill's Bay sites surveyed in May 2009 (n = 3 transects × 13 sites) and in January 2010 (n = 3 transects × 5 sites)

Best-subset analyses were carried out using R version 2.12.1 with the package 'leaps: regression subset'. Bi-variate plots for each disease and predictor variable were used to assess the spread of data and relationships between variables. Disease prevalence data were square-root transformed to meet the assumptions of normality (assessed using the Kolmogorov-Smirnov test), but predictor variables were not transformed.

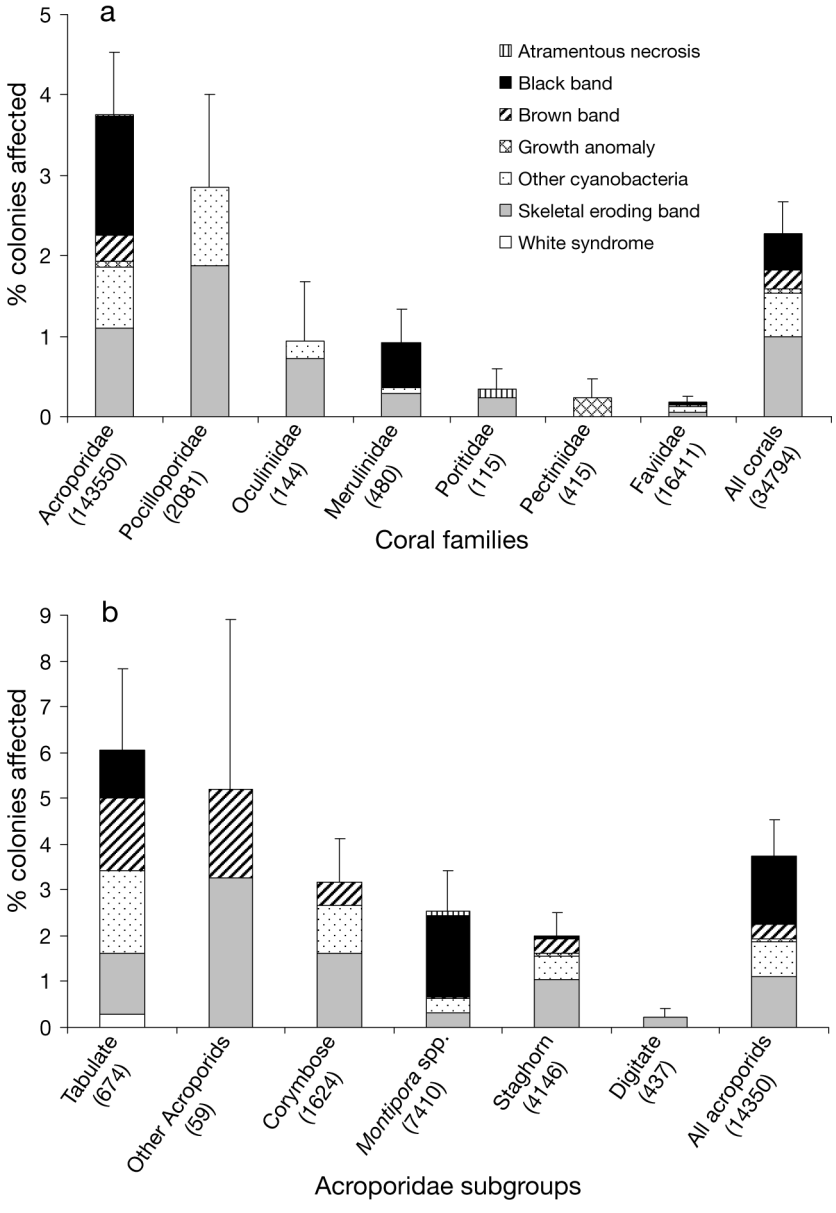

Fig. 2. Prevalence of 7 disease categories (a) across several scleractinian coral families and (b) in the Acroporidae (by morphology and genus) at Ningaloo Reef. Mean prevalence was calculated relative to mean number of coral colonies examined at sites surveyed across reef, including Bill's Bay. Mean number of colonies per site for each (a) family and (b) morphologic group across all sites is shown in parentheses with each category. Error bars: SE calculated from 28 sites. 'All corals' refers to all scleractinian corals

RESULTS

Disease types and prevalence

Seven diseases were recorded from the scleractinian corals at Ningaloo Reef: black band disease (BBD); brown band disease (BrB); white syndrome (WS); non-black cyanobacterial bands (OtCy), the presence of cyanobacteria being verified via examination of histological sections of diseased tissue; skeletal eroding band (SEB); growth anomalies (GAs); and atramentous necrosis (AN). The prevalence of these diseases (pooled) was relatively low, with disease affecting 2.28 ± 0.39% (mean ± SE) of all corals (Fig. 2a). The most common disease recorded was SEB, which affected ~1% of all corals. The 6 other diseases were each considerably less prevalent than SEB (OtCy 0.53%, BBD 0.44%, BrB 0.23%, GA 0.07%, WS <0.01% and AN <0.01%; Fig. 2a).

Susceptibility of corals

Within the order Scleractinia, the susceptibility of families to disease varied significantly (χ^2 = 514.10, df = 7, $p < 0.001$), from 0.2% (faviids) to 3.7% (acroporids) of corals showing signs of disease (Fig. 2a). The family Acroporidae was susceptible to the greatest number of diseases (7), most commonly BBD and SEB (Fig. 2). Prevalence of disease was also high among corals from the family Pocilloporidae, although only 2 diseases were observed on these corals (SEB and OtCy; Fig. 2a).

Within the Acroporidae, disease susceptibility varied greatly among different groups (χ^2 = 204.23, df = 5, $p < 0.001$; Fig. 2b). Disease was most common in species with tabulate morphologies (6.0%) and in the pooled group of 'other acroporids' (5.2%), which included *Isopora, Astreopora,* and caespitose and bottlebrush growth forms. Conversely, colonies with digitate morphologies were >5 times less likely to show signs of disease (<1% of colonies) than tabulate colonies.

No disease cases were recorded from any of the non-scleractinian coral taxa observed within the survey area, including the alcyoniids and fire corals (*Millepora* spp.).

Spatial variation in disease prevalence and potential drivers of variation

At large spatial scales disease prevalence varied ~7-fold across Ningaloo Reef ($F_{10,34}$ = 2.41, p = 0.028; Fig. 1a). The percentage of diseased colonies was greatest at Pelican Sanctuary, Oyster Stacks, Bundegi and Bundera, and lowest at Lefroy Bay (Fig. 1a). Disease prevalence also varied among sites at smaller spatial scales. Within Bill's Bay in 2009, disease prevalence varied >30-fold, with 0.1 to 3.1% of corals being affected per site ($F_{12,26}$ = 3.75, p = 0.023). Sites with the highest percentage of diseased colonies occurred in the south-eastern corner of the bay closest to the town of Coral Bay site, while lowest rates of disease prevalence were found in the central to northern sections of the bay (Fig. 1b). SEB was the most frequently observed disease, occurring at all sites across Ningaloo Reef and, in 2009, at 85% of sites in Bill's Bay (Fig. 1a,b). Two diseases were detected, but infrequently recorded: during the large scale survey AN was only seen at the Muiron Islands (Fig. 1a), and WS was only observed at 3 sites in Bill's Bay in 2009 (Fig. 1b).

A positive relationship was detected across Ningaloo Reef between the prevalence of all diseases and coral colonies exhibiting *Drupella* spp. feeding scars ($F_{1,9}$ = 6.24, p = 0.033, r^2 = 0.41; Table 2). *Drupella* spp. feeding scars were also included in 6 of the 7 best-subset models, accounting for 0.72 of AICw, implying that this is the best single variable predictor of total disease prevalence across Ningaloo Reef. Similarly, a positive relationship was seen between *Drupella* spp. feeding scars and the most common coral disease, SEB. No single-variable model was selected for predicting prevalence of OtCy, but a combination of all 4 variables accounted for 79% of data variation (Table 2). The density of coral colonies was positively related to BBD and accounted for ~30% of the variation of this disease across Ningaloo Reef sites. Human use, measured as either people or vessel density, was generally a poor predictor of disease prevalence (Table 2).

Table 2. Best-subset models for predicting disease along Ningaloo. Values in parentheses are the standardised beta coefficients for variables included in models. Corals: density of scleractinian corals per site; *Drupella*: number of coral colonies with *Drupella* spp. feeding scars at a site; People: mean density of people per 100 ha per observation flight; Vessels: mean density of vessels per 100 ha per observation flight; All: prevalence of all diseases; SEB: skeletal eroding band; OtCy: non-black cyanobacterial bands; BBD: black band disease; R^2: coefficient of determination; AIC: Akaike's Information Criterion; AICw: AIC weights. Models presented are those with the lowest AIC value and any other models within 2 AIC units

Best subsets	R^2	AIC	AICw
All			
Drupella (0.7), People (–0.4)	0.55	48.6	0.20
Drupella (0.6)	0.41	49.5	0.13
Drupella (0.6), Vessels (–0.3)	0.40	49.7	0.12
Corals (–0.6)	0.40	49.7	0.11
Drupella (0.4), Corals (–0.4)	0.50	49.8	0.11
Drupella (0.6), People (–0.3), Corals (–0.2)	0.56	50.3	0.09
Drupella (0.7), Vessels (–0.1), People (–0.3)	0.55	50.6	0.07
SEB			
Drupella (0.7), Vessels (–0.3)	0.68	35.7	0.2
Drupella (0.5), Corals (–0.4)	0.68	35.8	0.19
Drupella (0.8)	0.58	36.7	0.12
Drupella (0.6), Corals (–0.2), Vessels (–0.2)	0.71	36.9	0.11
Drupella (0.8), People (–0.2)	0.62	37.5	0.08
Drupella (0.7), Vessels (–0.4), People (0.1)	0.68	37.7	0.07
Drupella (0.5), Corals (–0.4), People (–0.1)	0.68	37.7	0.07
OtCy			
People (0.7), Vessels (–0.7), Corals (–0.6), *Drupella* (0.5)	0.79	21.3	0.42
BBD			
Corals (0.4)	0.31	–9.0	0.24
Vessels (–0.4)	0.20	–7.2	0.1
Corals (0.5), Vessels (0.2)	0.33	–7.3	0.1
Corals (0.5), People (0.1)	0.33	–7.2	0.1
Corals (0.5), *Drupella* (–0.1)	0.32	–7.0	0.09

Table 3. Best-subset models for predicting disease at Bill's Bay. Values in parentheses are the standardised beta coefficients for variables included in models. Anoxia: severity of anoxic events. See Table 2 for remaining definitions. Models presented are those with the lowest AIC value and any other models within 2 AIC units

Best subsets	R^2	AIC	AICw
All			
Anoxia (0.9), *Drupella* (0.6), Corals (–0.2)	0.44	33.1	0.24
Anoxia (0.9), *Drupella* (0.5)	0.34	33.2	0.23
Anoxia (0.5)	0.23	33.3	0.22
Anoxia (0.6), Corals (–0.3)	0.32	33.7	0.18
SEB			
Anoxia (0.2)	0.04	3.6	0.25
Corals (–0.1)	0.02	3.9	0.21
Drupella (–0.1)	0.01	4.0	0.2
Anoxia (0.3), Corals (–0.2)	0.07	5.1	0.11
Anoxia (0.3), *Drupella* (0.2)	0.05	5.5	0.1
OtCy			
Drupella (0.5)	0.30	–36.5	0.41
Drupella (0.5), Corals (–0.1)	0.30	–34.6	0.15
Drupella (0.6), Anoxia (0.1)	0.30	–34.5	0.15
BBD			
Anoxia (1.0), *Drupella* (0.6), Corals (–0.3)	0.60	25.0	0.41
Anoxia (1.0), *Drupella* (0.6)	0.50	25.9	0.26

At Bill's Bay a weak relationship was noted between disease prevalence and cumulative anoxia impacts ($r^2 = 0.23$) (Table 3). The severity of anoxic events was also a weak predictor of SEB prevalence, although these relationships accounted for <10% of the variation in data (Table 3). *Drupella* spp. feeding scars were the best single variable predictor of OtCy, and combined with anoxic events, were also the best predictor of BBD, accounting for 50% of the variation in disease prevalence (Table 3).

DISCUSSION AND CONCLUSIONS

Our assessment of coral disease at Ningaloo Reef, one of the first comprehensive and quantitative studies of this problem in the Indian Ocean, indicates that the prevalence of all diseases affecting corals at Ningaloo Reef (2.3%) is low and within the range of values recorded in many other Indo-Pacific regions (Table 1). The prevalence of SEB, the most common coral disease recorded at Ningaloo Reef, was similar to the prevalence of this disease in the Great Barrier Reef and Hawaii but was substantially lower than data recorded in the Red Sea (Table 1), which suggests that levels of SEB in the Red Sea are much higher than in other Indo-Pacific regions. Disease prevalence at Ningaloo Reef was also within the range of values recorded in some Caribbean studies (for example <1 to 4% by Weil et al. 2000 and 2.3 to 4.3% by Weil & Croquer 2009) but substantially lower than values for specific diseases in highly susceptible Caribbean taxa. For example, surveys of Venezuelan reefs in 2000 recorded up to 25% of colonies with white plague disease (Croquer et al. 2003).

Consistent with other locations throughout the Indo-Pacific, disease at Ningaloo Reef was seen to predominantly affect corals in the family Acroporidae (Willis et al. 2004, Page et al. 2009, Díaz & Madin 2010, Hobbs & Frisch 2010). Characteristics of this family, including investment in energy towards growth and dominance of reef substratum, rather than resistance to disease (Jackson & Hughes 1985, Palmer & Gates 2010), are likely to contribute to the susceptibility of this family to disease globally (Gladfelter 1982, Patterson et al. 2002, Willis et al. 2004, Hobbs & Frisch 2010). Acroporids are major reef builders, their diversity of growth forms contributing significantly to structural complexity and diversity of reefs (Lirman 1999, Graham et al. 2006, Wilson et al. 2008), including fish and invertebrates which closely associate with *Acropora* corals (Munday et al. 1997, Vytopil & Willis 2001, Pratchett 2005). At Ningaloo Reef, the threat to *Acropora* corals from disease is currently low; however, given the vulnerability of this genus to disease and a variety of other stressors (Precht et al. 2002), the potential loss of *Acropora* corals from reefs is of global concern because such a loss could have far-reaching effects on the abundance and distribution of many reef-associated animals (Pratchett et al. 2009).

Across Ningaloo Reef, disease prevalence correlated well with the presence of *Drupella* spp. feeding scars, although this correlation was primarily driven by a strong relationship between SEB and *Drupella* spp. feeding scars. At a finer spatial scale, occurrence of *Drupella* spp. feeding scars was positively related to both OtCy and BBD. Correlations between disease and *Drupella* spp. outbreaks have also been identified in the Red Sea (Antonius & Reigl 1997), as have positive associations between disease prevalence and other corallivores (Williams & Miller 2005, Dalton & Godwin 2006, Nugues & Bak 2009, Raymundo et al. 2009). Corallivores may play a role as vectors transmitting disease; for example, the fireworm *Hermodice carunculata* transmits *Vibrio shiloi*—a pathogen causing coral bleaching—in the Mediterranean coral *Oculina patagonica* (Sussman et al. 2003). Predation by corallivorous fish, gastropods or the crown-of-thorns starfish *Acanthaster planci* has also been implicated as a potential transmitter of disease (Williams & Miller 2005, Dalton & Godwin 2006, Nugues & Bak 2009, Raymundo et al. 2009). Moreover, injury caused by feeding may compromise host resistance (e.g. by interrupting the antibacterial mucus on the coral's surface; Ritchie 2006), making corals more susceptible to disease. However, corallivores such as *Drupella* spp. may actually be attracted to diseased corals (Page & Willis 2008), just as they are to injured corals (Forde 1992). Hence it is unclear if the relationship between *Drupella* spp. and disease relates to transmission of pathogens, coral injuries caused by *Drupella* spp. feeding or opportunistic feeding of *Drupella* spp. on diseased corals.

Within Bill's Bay, prevalence of disease was weakly correlated with cumulative disturbance to sites by anoxic events. This was mainly due to a positive relationship between BBD and the severity of anoxic events. Anoxic events have repeatedly caused extensive mortality of corals, fish and other reef animals at inner reef sites within Bill's Bay (Long 2007). The results of our study support suggestions that an increased frequency or intensity of disturbance may cause a loss of resilience, making corals more susceptible to disease. Physiological changes associated with chronic or pulse environmental stress may act to compromise coral health, decreasing disease resistance through loss of antimicrobial activity in the surface mucous layer, and through reduced energy reserves and loss of regenerative ability (Fitt et al. 2000, Fine et al. 2002, Ritchie 2006, Brandt & McManus 2009). For example, studies in the Caribbean, Kenya, and on the Great Barrier Reef, have documented increases in dis-

ease following warm-water bleaching events (Jones et al. 2004, Bruno et al. 2007, Brandt & McManus 2009, McClanahan et al. 2009, Miller et al. 2009).

We found no strong relationship between human use and prevalence of diseased corals at Ningaloo Reef. Nonetheless, previous studies have suggested that human-mediated stress to corals reduces coral resistance to disease (Dinsdale et al. 2008). Poor water quality caused by elevated nutrients and other pollutants has been identified as a key cause of poor health in corals and is strongly linked to coral disease (Kuta & Richardson 2002, Bruno et al. 2003, Voss & Richardson 2006). Fishing pressure has also been identified as a potential driver of coral disease (Page et al. 2009, Raymundo et al. 2009), and recreational divers and snorkelers may pose a threat as vectors of disease or as a source of disturbance (Williams & Miller 2005, Takabayashi et al. 2008, Page et al. 2009). The lack of any strong relationship between disease and human activities at Ningaloo Reef may relate to the relatively low level of human pressure here, since disease elsewhere has been found to be more prevalent on reefs adjacent to areas of high coastal development (Kaczmarsky 2006, Harding et al. 2008).

In summary, disease prevalence at Ningaloo Reef is relatively low, although the occurrence of 7 diseases previously undocumented in this reef region — some of which have had significant effects on reefs in other regions (Bruckner & Bruckner 1997) — highlights the need for further disease studies and ongoing monitoring along the West Australian coast. With climate warming expected to increase disease outbreaks (Harvell et al. 2002, Rosenberg & Ben-Haim 2002), and with increasing pressure on reefs worldwide (Hughes et al. 2003), including those along the West Australian coast (Page & Stoddart 2010), we need to better understand the effects of cumulative stressors on corals and minimize the stresses on reefs. Results of this study provide a baseline level for the study of disease at Ningaloo Reef, which may enable the use of bio-indicators to determine reef stress in the future.

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