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Spatial and temporal patterns of scleractinian coral, soft coral, and zoanthid disease on a remote, near-pristine coral reef (Palmyra Atoll, central Pacific)

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ABSTRACT: There is an urgent need for accurate baselines of coral disease prevalence across our oceans in order for sudden or unnatural changes to be recognized. Palmyra Atoll allows us to study disease dynamics under near-pristine, functionally intact conditions. We examined disease prevalence among all known species of scleractinian coral, soft coral and zoanthid (*Palythoa*) at a variety of coral reef habitats at Palmyra over a 2 yr period. In 2008, overall disease prevalence across the atoll was low (0.33%), but higher on the shallower backreef (0.88%) and reef terrace (0.80%) than on the deeper forereef (0.09%). Scleractinian coral disease prevalence was higher (0.30%) than were soft coral and zoanthid disease (0.03% combined). Growth anomalies (GAs) were the most commonly encountered lesions, with scleractinian species in the genera *Astreopora* (2.12%), *Acropora* (1.30%), and *Montipora* (0.98%) showing the highest prevalence atoll-wide. Discoloration necrosis (DN) was most prevalent in the zoanthid *Palythoa tuberculosa* (1.18%), although the soft coral *Sinularia* and *Montipora* also had a prevalence of 0.44 and 0.01%, respectively. Overall disease prevalence within permanently marked transects increased from 0.65% in 2008 to 0.79% in 2009. *Palythoa* DN contributed most to this increased prevalence, which coincided with rising temperatures during the 2009 El Niño. GAs on the majority of susceptible genera at Palmyra increased in number over time, and led to tissue death. Host distribution and environmental factors (e.g. temperature) appear to be important for determining spatiotemporal patterns of disease at Palmyra. More sophisticated analyses are required to tease apart the likely inter-correlated proximate drivers of disease occurrence on remote, near-pristine reefs.

KEY WORDS: Coral disease · Coral disease progression · Growth anomaly · Discoloration necrosis · Soft coral disease · Zoanthid disease · Central Pacific · Palmyra Atoll

INTRODUCTION

Remote reef systems, removed as much as possible from present-day direct anthropogenic impacts, can provide insights into the structure and functioning of near-pristine reef habitats (Knowlton & Jackson 2008). Fish biomass is greater and dominated by large-bodied predators at these remote locations where fishing is reduced or absent (Sandin et al. 2008, Williams et al. 2011). In parallel, unfished reefs tend to have higher coral cover and recruitment (Mumby et al. 2007, Sandin et al. 2008) and a lower cover of competitive fleshy algae (McClanahan 1997, Mumby et al. 2006). However, one important ques-

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tion about coral reef health that has yet to be fully answered is: What are the 'natural' levels and dynamics of disease in these more functionally intact systems?

Marine diseases, particularly coral diseases, represent a global threat to coral reef health and function (Harvell et al. 1999, 2002) in addition to direct anthropogenic stressors such as overfishing and pollution (Hughes et al. 2003, Bellwood et al. 2004). Coral diseases are capable of reducing live coral cover (Nugues 2002, Richardson & Voss 2005), and severe outbreaks can lead to entire community phases-shifts on reefs (Aronson & Precht 2001), thus impacting fish and invertebrate biodiversity (Jones et al. 2004a, Idjadi & Edmunds 2006). Coral diseases are widespread throughout the Indo-Pacific (Sutherland et al. 2004), and although research has increased rapidly in recent times, many baseline disease studies represent reefs already heavily altered and disturbed by anthropogenic activities. If we are to detect real change and aspire to maintain coral disease prevalence at more 'natural' levels on reefs, then we must have accurate baselines from more functionally intact, pristine, or near-pristine systems.

Overall disease prevalence often appears to be low on pristine or near-pristine reefs (Aeby 2006, Sandin et al. 2008, Williams et al. 2008a, Vargas-Angel 2009). Palmyra Atoll National Wildlife Refuge (NWR) in the Northern Line Islands is one of the most remote reef systems in the world. With the exception of a period during WWII when Palmyra served as a US military base, the atoll has lacked a long-term resident population, and present-day direct anthropogenic impacts are minimal. As a result, Palmyra Atoll represents a largely pristine oceanic reef environment, with some of the highest coral diversity (Maragos & Williams 2011) and greatest concentrations of large-bodied predators (Sandin et al. 2008, Williams et al. 2011) in the central Pacific. Palmyra Atoll therefore provides us with the opportunity to study disease dynamics under more functionally intact reef conditions. Several cnidarian lesions have been identified and characterized at the cellular level at Palmyra (Williams et al. 2011) and, although baseline disease levels are known for scleractinian corals at this site (Sandin et al. 2008, Williams et al. 2008a, Vargas-Angel 2009), these studies have lacked temporal assessments of disease dynamics, particularly with regard to disease severity and the fate of infected hosts. This knowledge is crucial if we are to understand the potential impacts of disease on populations of corals and other cnidarians (Lafferty et al. 2004, Willis et al. 2004, Sato et al. 2009). In addition, previous studies have not assessed disease levels for soft coral or zoanthid species present at Palmyra.

This study therefore aimed to (1) carry out the most comprehensive baseline survey of scleractinian coral, soft coral and zoanthid disease, across a broad range of reef habitats, to date for Palmyra; (2) assess for temporal shifts in disease prevalence; and (3) monitor changes in disease severity and fate of the diseased hosts. The present study represents one of the first to quantify spatiotemporal patterns of disease under near-pristine reef conditions, thus contributing to our limited understanding of disease dynamics in such systems. More broadly, this study aimed to provide critical baseline data to compliment ongoing research into disease etiology and environmental associations of disease prevalence at Palmyra, to better inform the long-term management of this important reef site.

MATERIALS AND METHODS

Study site. Palmyra Atoll is located in the central Pacific Ocean (05° 52′ N, 162° 06′ W), 1930 km south of the main Hawaiian Islands (Fig. 1). The coral reefs surrounding the atoll cover approximately 60 km^2. Occupation by the military during WWII led to several modifications to the atoll including extensive dredging, land reclamation, causeways linking the islets, building of airstrips, and the excavation of a 9 m deep channel to allow ship access to the lagoon (Dawson 1959). These modifications severely degraded the lagoon environment (Maragos et al. 2008), but the other regions remain largely pristine (Sandin et al. 2008). Palmyra was designated a US National Wildlife Refuge in 2001 and is now co-owned by the US Fish and Wildlife Service and The Nature Conservancy.

Disease spatial and temporal patterns. Surveys were conducted at 12 sites during 4 research trips: July–August 2008, October–November 2008, July 2009, and October–November 2009 (Fig. 1). Regions of the atoll were specifically targeted to encompass a range of habitat strata and benthic community patterns previously identified at Palmyra (Williams et al. 2008b). The exact site and transect locations were then randomly distributed within these pre-defined regions. Both random and permanently established belt transects (50 m in length) were used at each site (Table 1). For the backreef and terrace sites, a central point was randomly chosen and marked with a stainless steel pole. From there, random bearings were generated to run transect lines in a radiating pattern from the central marker. The distance from the central marker to the start of each transect line was also random. Permanent transects were marked every 5 m with plastic cattle tags, attached with cable ties to the surrounding dead substrate. Five permanent transects were established at each backreef and terrace site (with

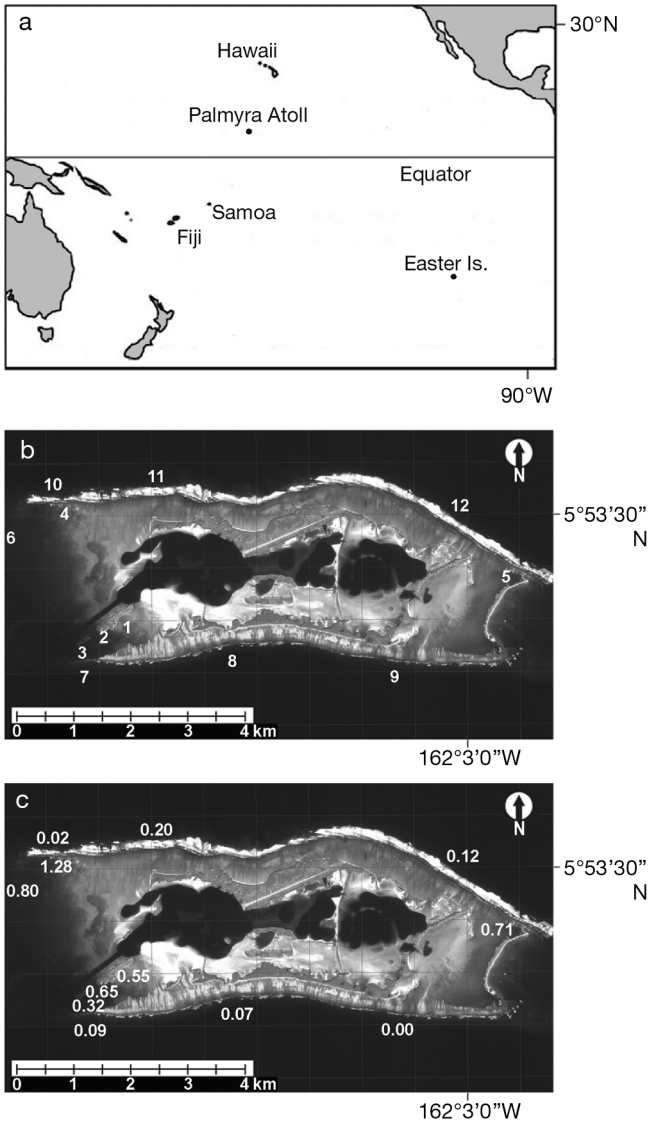

Fig. 1. (a) Location of Palmyra Atoll and (b) the 12 survey sites: 1, Penguin Spit inner buoy; 2, Penguin Spit middle buoy; 3, Penguin Spit outer buoy; 4, Tortugonas backreef; 5, North Coral Gardens; 6, Western Terrace; 7, Penguin Spit forereef; 8, Home & Paradise; 9, Holei & Bird; 10, Tortugonas forereef; 11, Strawn; 12, Quail & Whippoorwill. (c) Total scleractinian coral, soft coral and zoanthid disease prevalence (% individuals affected) at each site during 2008

the exception of North Coral Gardens, where n = 4) (Table 1). For the forereef sites, a central point was again randomly selected within a depth range of 9 to 10 m and marked with a stainless steel pole and subsurface buoy. Two permanent transects were established at each forereef site (Table 1). Transects were run either side of the central marker at random starting positions parallel to the depth contours of the site.

Lesions were classified according to gross morphology, and assigned the host genus and descriptive name (Work & Aeby 2006). All the diseases discussed in this study have been previously characterized at the gross and microscopic scale (Williams et al. 2011). In summary, growth anomalies generally manifest as hyperplasia of the basal body wall and of the coenenchyme in scleractinian and soft corals, respectively. An exception to this is the *Fungia* growth anomaly (GA), which is initially the result of the gall crab *Fungicola* sp. Cases of discoloration in the scleractinian coral *Montipora*, the soft coral *Sinularia*, and the zoanthid *Palythoa* all manifest as necrosis. The term discoloration necrosis (DN) is therefore used to describe this disease across all 3 genera. To quantify disease prevalence, all scleractinian corals, soft corals and zoanthids along the first half of each transect (25 × 2 m) were enumerated and identified (where possible) to species level, with the number displaying gross lesions recorded. Along the second half of each transect, colonies displaying lesions were also enumerated but over an extended area (25 × 6 m), giving a total of 200 m^2 surveyed for disease within each transect. Time constraints underwater prevented the enumeration of all colonies along the second wider part of each transect. Therefore, the total number of colonies surveyed for disease was estimated based upon the average number of colonies m^{-2} found within the first half of the transect. This method was justified as colony densities between transects within each monitoring site at Palmyra are known to be highly homogenous (Williams et al. 2008b), and relatively low variation in the number and density of colonies was observed during the present study (Table 1). In summary, the technique gave 2 measures of disease prevalence for each transect, the first an actual proportion of diseased colonies (within 50 m^2), and the second an estimated disease prevalence for the entire 200 m^2. A total of 59 transects representing 11800 m^2 of reef were surveyed during 2 field seasons in 2008 (Table 1). Changes in disease prevalence between 2008 and 2009 were calculated from the data obtained from the first half of each transect, representing 2000 m^2 of reef across 40 permanent transects (Table 1). This was to monitor actual change in disease prevalence rather than changes in estimated disease prevalence. To ensure consistency, all surveys were conducted by a single observer across all research seasons.

Scleractinian coral growth anomaly severity. A total of 53 diseased scleractinian colonies, all exhibiting signs of growth anomalies, within the 5 backreef sites and the Western Terrace were tagged in July–August 2008 (these diseased colonies represented a sub-set of the total diseased individuals encountered in 2008 and were selected at random): *Acropora* spp. (n = 9), *Astreopora myriophthalma* (n = 12), *Fungia concinna* (n = 7), *Montipora* spp. (n = 23), and massive *Porites* spp. (n = 2).

Table 1. Sampling effort across regions and sites for disease surveys at Palmyra Atoll. Numbers in parentheses refer to the number of random transects conducted in 2008 that subsequently became permanent transects for examining temporal change in disease prevalence in 2009. Variation (±SE) in colony density and number of colonies inspected for disease is shown between transects within each site

Atoll region	Site	No. of transects	Random area surveyed in 2008 (m^2)	Permanent area re-surveyed in 2009 (m^2)	Mean colony density (no. m^{-2}) in 2008	Mean no. of colonies inspected in 2008
SW backreef	Penguin Spit inner buoy	5 (5)	1000	250	3.48 ± 0.15	174.2 ± 7.4
	Penguin Spit middle buoy	5 (5)	1000	250	3.41 ± 0.17	170.4 ± 8.5
	Penguin Spit outer buoy	5 (5)	1000	250	4.93 ± 0.65	246.6 ± 32.4
NW backreef	Tortugonas backreef	5 (4)	1000	200	1.80 ± 0.11	89.8 ± 5.7
NE backreef	North Coral Gardens	4 (4)	800	200	4.38 ± 0.19	218.8 ± 9.4
Western reef terrace	Western Terrace	5 (5)	1000	250	4.75 ± 0.30	237.6 ± 15.2
SW forereef	Penguin Spit forereef	5 (2)	1000	100	5.44 ± 0.72	272.0 ± 36.1
South forereef	Home & Paradise	5 (2)	1000	100	4.51 ± 0.67	225.6 ± 33.3
SE forereef	Holei & Bird	5 (2)	1000	100	2.64 ± 0.49	131.8 ± 24.7
NW forereef	Tortugonas forereef	5 (2)	1000	100	8.64 ± 0.71	432.2 ± 35.2
North forereef	Strawn	5 (2)	1000	100	5.60 ± 0.19	279.8 ± 9.25
NE forereef	Quail & Whippoorwill	5 (2)	1000	100	6.46 ± 0.18	322.8 ± 9.0
Total	12	59 (40)	11800	2000		

Growth anomalies (tumor-like growths) are by far the most commonly encountered lesion on scleractinian corals at Palmyra (Williams et al. 2011). The rarity of other types of disease limited our initial tagging of diseased colonies in 2008 to growth anomaly lesions. Each colony was photographed and the number of growth anomalies noted *in situ* before being re-photographed during October–November 2008, July 2009, and October–November 2009.

Statistical analyses. We used a permutational analysis of variance (PERMANOVA) (Anderson 2001, McArdle & Anderson 2001) to test for spatial differences in disease prevalence in 2008. The technique does not require a normal distribution in the data set, and the statistical assessment of permuted p-values carries more weight than arbitrary thresholds determined from probability tables. Two factors were tested, reef type (backreef, terrace, and forereef) and site (5 backreef, 1 terrace, and 6 forereef). To examine for temporal change (2008 versus 2009) no statistical test was used, as our use of permanent transects (and therefore permanently monitored coral populations) meant that any change seen was not confounded by spatial artefacts. Additionally, a seasonal factor within each year could not be tested as we were unable to survey all 40 permanent transects during each of the 4 research seasons. Both spatial factors in 2008 were treated as fixed, with site nested within reef type. The effect of both factors, and their interaction, was tested using unrestricted random permutations of the raw data (maximum 9999), Type III (partial) sums of squares, and zero-adjusted Bray-Curtis similarity matrices (Clarke et al. 2006). Analyses were conducted using PERMANOVA+ (Anderson et al. 2008).

RESULTS

Spatial patterns of disease in 2008

A total of 55 156 colonies (scleractinian coral, soft coral, zoanthid) were inspected for disease across 11800 m^2 of reef in 2008. Across the whole atoll, scleractinian coral disease prevalence equaled 0.30%, and soft coral and zoanthid disease prevalence combined equalled 0.03% (Table 2). Overall, disease prevalence was higher on the backreef (0.88%) and terrace (0.80%) than on the forereef (0.09%) (pseudo-$F_{2,58}$ = 43.80, p = 0.0001), with differences also present among sites (pseudo-$F_{9,58}$ = 2.449, p = 0.0066). On the backreef and terrace, overall disease prevalence only differed between 2 sites, with total prevalence on the northwest backreef (1.28%) being significantly higher than on the outer southwest backreef (0.32%) (t = 2.729, p = 0.0171) (Fig. 1, Table 2). Among forereef sites, the southeast forereef had lower overall disease prevalence (0%) than both the north (0.20%) and northeast forereefs (0.12%) (t = 4.549, p = 0.0085 and t = 3.668, p = 0.0081, respectively). Along the north coast, the northwest forereef had lower overall disease prevalence (0.02%) than did both the north and northeast

The heading 'Scleractinian prevalence' was corrected after publication

Table 2. Scleractinian coral, soft coral and zoanthid (*Palythoa*) disease prevalence (% individuals affected) among 10 regions at Palmyra Atoll in 2008. Data generated from 59 transects covering 11 800 m^2 of reef. Disease prevalence is shown as a total for each disease at each site. Number of colonies inspected when calculating each prevalence value is shown in parentheses. Variation (±SE) in overall disease prevalence between transects at each site is shown. –: host did not occur

Atoll region	Site	Growth anomaly						Discoloration necrosis			Sclerac-tinian prevalence	Soft coral & zoanthid prevalence	Overall prevalence
		Acropora	*Astreo-pora*	*Fungia*	*Monti-pora*	*Porites*	*Sinu-laria*	*Monti-pora*	*Palythoa*	*Sinu-laria*			
SW backreef	Penguin Spit inner buoy	1.92 (104)	1.69 (652)	2.68 (112)	0.49 (700)	–	–	0 (700)	–	–	0.59 (3224)	0 (260)	0.55 ± 0.16 (3484)
	Penguin Spit middle buoy	0.60 (168)	1.56 (64)	0.89 (564)	1.22 (1228)	0 (4)	0 (24)	0 (1228)	–	0 (24)	0.66 (3312)	0 (96)	0.65 ± 0.24 (3408)
	Penguin Spit outer buoy	0.43 (232)	0 (8)	0.45 (2240)	0.83 (484)	0 (92)	0 (180)	0.21 (484)	–	0 (180)	0.34 (4708)	0 (224)	0.32 ± 0.15 (4932)
NW backreef	Tortugonas backreef	1.97 (608)	6.25 (16)	7.29 (96)	0.65 (308)	12.5 (8)	0 (8)	0 (308)	–	0 (8)	1.29 (1788)	0 (8)	1.28 ± 0.29 (1796)
NE backreef	North Coral Gardens	0 (52)	2.21 (272)	–	0.96 (1884)	6.25 (16)	–	0 (1884)	–	–	0.71 (3500)	–	0.71 ± 0.07 (3500)
Western reef terrace	Western Terrace	1.14 (88)	6.67 (60)	0 (320)	1.90 (1740)	0 (32)	0 (36)	0 (1740)	–	0 (36)	0.83 (4592)	0 (160)	0.80 ± 0.19 (4752)
SW forereef	Penguin Spit forereef	1.79 (56)	–	0 (1384)	0.41 (244)	0.45 (220)	0 (156)	0 (244)	6.25 (16)	0.64 (156)	0.10 (5204)	0.85 (236)	0.09 ± 0.03 (5440)
South forereef	Home & Paradise	0.40 (248)	0 (12)	0 (88)	0 (368)	0.38 (260)	0 (64)	0 (368)	2.78 (36)	1.56 (64)	0.05 (4240)	0.37 (272)	0.07 ± 0.04 (4512)
SE forereef	Holei & Bird	0 (60)	–	0 (304)	0 (136)	0 (212)	0 (8)	0 (136)	0 (36)	0 (8)	0 (2572)	0 (64)	0 (2636)
NW forereef	Tortugonas forereef	1.47 (68)	–	0.03 (3812)	0 (236)	0 (268)	0 (80)	0 (236)	3.12 (32)	0 (80)	0.02 (8172)	0 (472)	0.02 ± 0.01 (8644)
North forereef	Strawn	4.17 (48)	–	0 (40)	0 (292)	0.31 (320)	0 (92)	0 (292)	2.03 (396)	1.09 (92)	0.06 (4760)	0.96 (836)	0.2 ± 0.05 (5596)
NE forereef	Quail & Whippoorwill	2.27 (44)	–	0 (252)	0 (140)	0.45 (220)	0.39 (264)	0 (140)	0.72 (552)	0.39 (264)	0.04 (5372)	0.55 (1084)	0.12 ± 0.04 (6456)
Overall prevalence		1.30 (1776)	2.12 (1084)	0.28 (9212)	0.98 (7760)	0.36 (1652)	0.11 (912)	0.01 (7760)	1.18 (1068)	0.44 (912)	0.30 (51444)	0.03 (3712)	0.33 (55156)

forereefs ($t = 3.041$, p = 0.0079 and $t = 1.956$, p = 0.0226, respectively).

GAs (tumor-like growths) were the most commonly encountered lesions, affecting 6 scleractinian and soft coral genera, with *Astreopora* (2.12%), *Acropora* (1.30%), and *Montipora* (0.98%) showing the highest prevalence atoll-wide (Table 2). Of those genera displaying GAs, the soft coral *Sinularia* sp. had the lowest prevalence atoll-wide (0.11%), with this lesion only being observed on this species at the northeast forereef. At the site level, *Astreopora* and *Montipora* GAs were most prevalent on the Western Terrace (6.67 and 1.9%, respectively), while *Acropora* GAs were most prevalent (4.17%) on the deeper northeast forereef (Table 2).

Atoll-wide, DN was most prevalent in the zoanthid *Palythoa* (1.18%), although the soft coral *Sinularia* and the scleractinian coral *Montipora* also had a prevalence of 0.44 and 0.01%, respectively (Table 2). At the site level, *Palythoa* DN was most prevalent on the southwest forereef (6.25%), and *Sinularia* DN was most prevalent on the south forereef (1.56%). *Montipora* DN was only observed on the southwest backreef (0.21%). No other lesion types except those listed in Table 2 were seen within our transects. Rare cases of tissue loss diseases have previously been noted on the reefs at Palmyra (Williams et al. 2011), but no clear cases of black band disease or white syndromes (often commonly seen on reefs) were observed within (or even outside) our transects in either 2008 or 2009.

Change in disease prevalence between 2008 and 2009

Overall disease prevalence within our permanently marked transects increased from 0.65% in 2008 to 0.79% in 2009. All diseases increased in prevalence, with the exception of *Astreopora* and *Montipora* GAs, which decreased in prevalence (Table 3). The largest increase occurred for *Palythoa* DN, which increased in prevalence between 2008 and 2009 atoll-wide by 9.29%, with the largest increase in prevalence (50%) occurring on the northwest forereef (Table 3). The largest decrease occurred for *Astreopora* GAs, with prevalence decreasing atoll-wide by 1.81% between 2008 and 2009. The largest inter-annual decrease in *Astreopora* GA prevalence occurred on the northwest backreef and equalled 6.66%.

Scleractinian growth anomaly severity and fate

Montipora spp. corals with growth anomalies showed the most consistent signs of GAs increasing in number over time, with 17 of the 23 affected colonies (74%) showing an increase in GA abundance between July–August 2008 and October–November 2009. The mean (±1 SD) number of GAs per affected *Montipora* spp. colony increased from 17 ± 25 to 31 ± 39 GAs per colony. GA mortality (of any severity) was observed in 14 of the 23 colonies (61%). One colony showed signs

Table 3. Changes in the prevalence (% of population affected) of diseases at Palmyra Atoll between 2008 and 2009. Data generated from 40 permanent transects (each 50 m^2) representing 2000 m^2 of reef across 12 sites. The first value in each case equals the prevalence in 2008; the second value equals the prevalence in 2009. <: increase in prevalence; >: decrease in prevalence. –: host did not occur. Blank cell means that the disease was not observed within the first half of the permanent transects in 2008 or 2009

Site	Growth anomaly						Discoloration necrosis	
	Acropora	*Astreopora*	*Fungia*	*Montipora*	*Porites*	*Sinularia*	*Montipora*	*Palythoa*
Penguin Spit inner buoy		3.02 > 1.48	5.0 > 2.0	1.11 > 0.00	–			–
Penguin Spit middle buoy		0.00 < 1.54	2.76 < 3.65	0.96 > 0.55			0.00 < 0.36	–
Penguin Spit outer buoy			1.09 < 1.98	0.00 < 0.77				–
Tortugonas backreef	0.54 < 2.5	6.66 > 0.00	22.0 < 24.0					–
North Barren		3.79 > 1.09	–	1.52 < 1.78		–		–
Western Terrace	2.86 > 0.00	23.3 > 20.0	0.00 < 1.25	3.56 > 2.98	–		0.00 < 0.65	–
Penguin Spit forereef		–			3.85 < 4.54			
Home & Paradise	0.00 < 8.34							
Holei & Bird								
Tortugonas forereef		–	0.30 > 0.27		0.00 < 4.17			0.00 < 50.0
Strawn		–			0.00 < 3.13	0.00 < 2.78		
Quail & Whippoorwill		–						0.00 < 5.72
Overall prevalence	0.09 < 0.27	5.25 > 3.44	2.83 < 3.01	0.60 > 0.51	0.39 < 1.18	0.00 < 0.25	0.00 < 0.08	0.00 < 9.29
Overall change	Increase 0.18	Decrease 1.81	Increase 0.18	Decrease 0.09	Increase 0.79	Increase 0.25	Increase 0.08	Increase 9.29

of a decrease in GA abundance (from 2 to 1 GA), but this was due to the mortality (100% tissue loss) of the GA. Five of the 23 colonies (22%) showed no change in GA abundance over time; however, all displayed signs of initial GA tissue loss with subsequent colonization by filamentous turf algae. Two colonies showed signs of fish predation of the GA tissue during the observation period.

Of the 9 affected *Acropora* colonies monitored over time, 6 (67%) experienced an increase in GA abundance over time, increasing from 22 ± 48 GAs per colony to 35 ± 58. All 6 colonies showed signs of GA mortality over time. Two colonies showed signs of decreased GA abundance; however, in both cases this was due to the death of the GAs. Mortality generally occurred as a result of GA bleaching and tissue loss followed by colonization by filamentous turf algae and crustose coralline algae (Fig. 2).

Of the 12 affected *Astreopora* colonies monitored, 4 (33%) experienced an increase in GA abundance over time, increasing from 2 ± 2 GAs per colony to 5 ± 3. GA mortality only occurred for 1 of the colonies, with 1 of the 2 GAs experiencing 100% tissue loss. In 3 of the colonies, the GA tissue shifted morphology, with the polyps becoming more regularly arranged and the swollen coenosteum subsiding. The remaining 5 colonies showed no change over time, each consistently displaying a focal nodular GA with no signs of tissue loss.

Fungia growth anomalies showed no increase in abundance per colony during the monitoring, with the 7 affected colonies displaying either 1 or 2 lesions in an apical position adjacent to the polyp mouth at all times. Sediment accumulation and progressive algal growth occurred adjacent to the GA in 2 of the *Fungia* colonies, leading to partial death of the colony surface (particularly the mouth area) in both cases (Fig. 3).

Of the 2 affected massive *Porites* colonies monitored, 1 showed an increase in GA abundance (from 1 to 2 GAs), while the other displayed a single GA throughout the observation period. No GA death was observed on either of the colonies over time, and the GA tissue did not change in appearance.

DISCUSSION

Marine diseases, particularly coral diseases, represent a significant threat to reef health worldwide (Harvell et al. 1999, 2002, Sutherland et al. 2004), and detecting shifts in disease prevalence within a location relies on us first having accurate baselines (Ward & Lafferty 2004). More broadly, information regarding disease dynamics from remote, minimally impacted reef systems such as Palmyra Atoll helps to highlight any differences in disease levels or dynamics between such systems and more impacted reefs.

Spatial patterns of disease susceptibility

Overall disease levels of scleractinian corals, soft corals and zoanthids at Palmyra were low (0.33%), mirroring reports from other remote locations such as the neighboring Kingman Reef National Wildlife Refuge (0.04%) (Vargas-Angel 2009), the northwestern Hawaiian Islands (0.5%) (Aeby 2006), and the Wakatobi Marine Park in Indonesia (0.57%) (Haapkyla et al. 2007), and in marked contrast to heavily impacted reef systems such as the Florida Keys (19.2 to 54.6%) (Santavy et al. 2001). Vargas-Angel (2009)

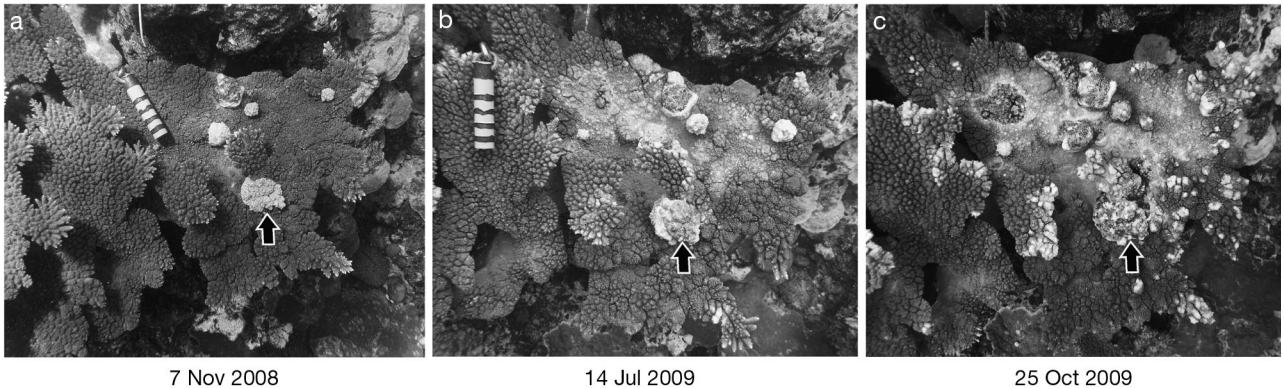

Fig. 2. Progression and death of growth anomalies (GAs) on a tabular *Acropora* sp. colony over time at Palmyra Atoll. Note the bleached appearance of the GA in (a) November 2008 (arrow) and the initial tissue loss and subsequent colonization by algae in (b) July 2009 (arrow). (c) By October 2009 this GA is completely dead (arrow) and crustose coralline algae have started to grow on parts of the dead coral skeleton. Reference bar in (a,b) = 12 cm in length. The growth anomaly indicated by the arrow increased in size over time: 31.4 cm^2 (Nov 2008), 35.3 cm^2 (Jul 2009), 36.4 cm^2 (Oct 2009)

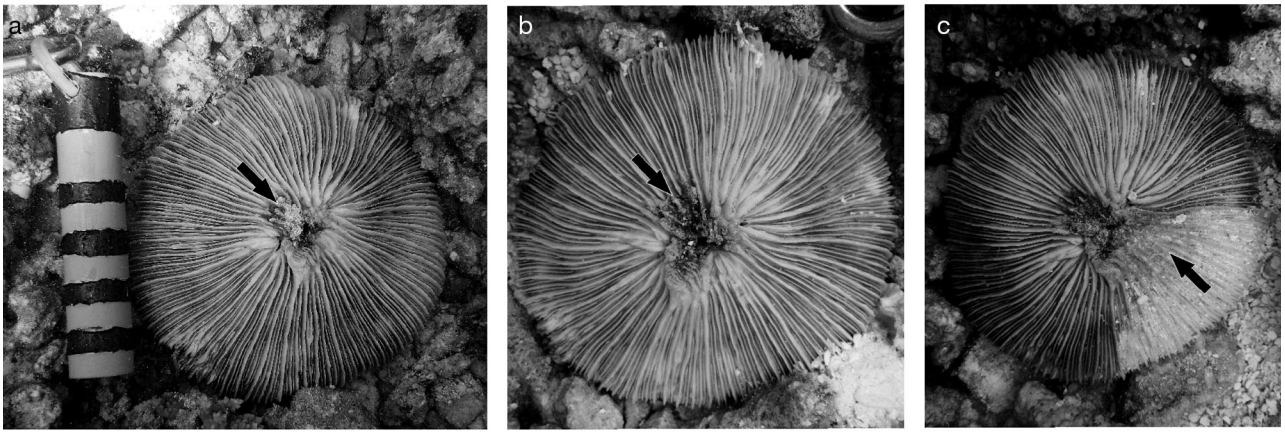

Fig. 3. *Fungia* growth anomaly (GA) development over time at Palmyra Atoll. The initial lesion is the result of the gall crab *Fungicola* sp. Note the accumulation of sediment adjacent to the GA in (a) 2008 (arrow) and the colonization of the area by filamentous turf algae in (b) July 2009 (arrow). Also note that the algae have extended downward from the polyp mouth by July 2009. In (c) October 2009 note the death of a segment of the colony and the subsequent spread of algae from the initial lesion across the dead coral substrate (arrow). Reference bar in (a) = 12 cm in length

reported lower disease prevalence at Palmyra (0.04%) than our value of 0.33%, but we additionally surveyed the shallow backreef areas (<4 m depth) where the majority of disease occurred during our surveys. Vargas-Angel (2009) surveyed the forereef and shallow western terrace (5 to 16 m depth), thus missing the most heavily diseased environments at Palmyra (e.g. northeast and northwest backreefs).

Differences in disease susceptibility among genera were apparent at Palmyra, and this is a common finding on reefs (Nugues 2002). The most susceptible genera were *Astreopora* and *Acropora*, consistent with other reports of high susceptibility of corals within the family Acroporidae (*Acropora*, *Anacropora*, *Astreopora*, *Montipora*) susceptibility in Australia (Willis et al. 2004, Dalton & Smith 2006, Page & Willis 2006), the Caribbean (Green & Bruckner 2000, Porter et al. 2001, Weil 2004), the Florida Keys (Miller et al. 2002, Patterson et al. 2002, Sutherland et al. 2004), Indonesia (Haapkyla et al. 2007), and the other Pacific Remote Island Areas (PRIAs) (Vargas-Angel 2009). Pocilloporidae, although one of the most dominant families, showed no signs of disease in our transects at Palmyra (although rare cases of *Pocillopora meandrina* tissue loss and growth anomalies were observed outside our transects). These findings are consistent with previous reports for Palmyra, neighboring Kingman Reef (Vargas-Angel 2009) and the NWHI (Aeby 2006), and in contrast to areas such as the Great Barrier Reef (Willis et al. 2004) and Guam (Myers & Raymundo 2009), where pocilloporids show high susceptibility to disease. Interestingly, our prevalence values for soft coral disease (family Alcyoniidae), although extremely low, are the first reported for Palmyra, and indeed the first reported for the entire PRIAs. Some soft corals can be more tolerant than scleractinian corals of stressful environmental factors, such as heavy sedimentation (McClanahan & Obura 1997). At Palmyra, *Sarcophyton* occurs at increased densities at backreef sites that experience high sedimentation, while *Lobophytum* sp. and *Sinularia* dominate the forereef regions experiencing the highest wave exposures (Williams et al. 2008b). Soft corals also show a high tolerance to increased seawater temperature, which causes scleractinian corals to bleach at Palmyra (Williams et al. 2010b). The ability of soft corals to tolerate stressful environmental conditions may make them more resistant to disease. However, not all soft corals show disease resistance. For example, in the Caribbean, sea fans (family Gorgoniidae) show high susceptibility to the fungal disease aspergillosis (Smith et al. 1996, Geiser et al. 1998, Kim & Harvell 2002).

At Palmyra, the shallow backreef and western reef terrace had higher disease prevalence than the deeper forereef. This pattern may reflect increased environmental stress, as the shallow sites experience higher temperatures and fine/silt sediment fallout, and wider fluctuations in temperature, turbidity and chlorophyll *a* than the deeper forereef (Williams et al. 2010b). Environmental stress, for example elevated seawater temperatures and reduced water quality, has been linked to increases in coral disease prevalence (Sutherland et al. 2004, Bruno et al. 2007, Harvell et al. 2009, Williams et al. 2010a). However, the patterns may also reflect the distribution of susceptible species. Coral community structure can dictate overall bleaching prevalence on a reef (McClanahan et al. 2007), and this is also likely to be true for overall disease patterns. The most

disease susceptible genera at Palmyra also dominate the shallow water coral communities (Williams et al. 2008b). On the forereef, these genera are found either at greatly reduced densities (e.g. *Acropora*) or are largely absent (e.g. *Astreopora*) (Williams et al. 2008b). The increase in disease prevalence on the north and northeastern forereefs, relative to all other forereef sites, can be explained by the distribution of the zoanthid *Palythoa*. DN in *Palythoa* represented the majority of disease cases found on the forereef and colony densities were highest at these sites.

Disease severity, fate and temporal patterns

It is important to understand how disease severity changes on an individual, the fate of infected individuals, and the temporal shifts in disease prevalence in order to understand the potential impacts of disease on coral populations (Lafferty et al. 2004, Willis et al. 2004, Sato et al. 2009). GAs on all susceptible genera at Palmyra, with the exception of *Fungia*, increased in abundance on a single infected colony over time. *Fungia* GAs at Palmyra are known to be initially caused by the gall crab *Fungicola* sp., while the etiologies of all the other scleractinian coral GAs remain unknown (Williams et al. 2011). Little is known about *Fungicola* sp. ecology, but no more than 2 lesions per *Fungia* colony have ever been observed at Palmyra (Williams et al. 2011). GAs on *Acropora* and *Montipora* colonies often died and on occasion were associated with whole colony mortality, whereas *Astreopora* and *Porites* GAs never showed signs of mortality, with *Astreopora* GAs sometimes reverting back to resemble normal-looking tissue. The fact that GAs increase in number on an infected individual over time, combined with their lack of normal polyp structure, reduced density of zooxanthellae and lack of digestive organs (Williams et al. 2011) means that they negatively impact affected corals at Palmyra, particularly *Acropora* and *Montipora* species. Coral GAs are now widely acknowledged as a deleterious condition, capable of leading to reduced colony growth, decreased density of coral skeleton, loss of mucus secretory cells and nematocysts, reduced density of zooxanthellae, reduced fecundity, tissue necrosis, and a loss, reduction or degeneration of normal polyp structure (Cheney 1975, Bak 1983, Peters et al. 1986, Coles & Seapy 1998, Yamashiro et al. 2000, Gateno et al. 2003, Domart-Coulon et al. 2006, Work et al. 2008, Williams et al. 2011).

Temporal as well as spatial changes in coral disease prevalence are common (Sutherland et al. 2004, Harvell et al. 2009) and often relate to seasonal shifts in environmental conditions, such as temperature (Edmunds 1991, Kuta & Richardson 1996, Bruckner & Bruckner 1997, Patterson et al. 2002, Jones et al. 2004b, Boyett et al. 2007, Bruno et al. 2007, Sato et al. 2009, Zvuloni et al. 2009). Environmental stress impairs coral host immunity and promotes pathogen virulence (Fitt et al. 2001, Harvell et al. 2002, Blanford et al. 2003, Lafferty & Holt 2003, Ward et al. 2007). As such, disease prevalence (predominantly tissue loss diseases) often increases during or proceeding coral bleaching events when temperatures are high and the coral hosts are compromised (Jones et al. 2004b, Miller et al. 2006, Whelan et al. 2007, Brandt & McManus 2009, Bruckner & Hill 2009, Cróquer & Weil 2009). At Palmyra, overall disease prevalence within our permanent transects was higher in 2009 than 2008 (by 0.14%). In some cases genus-specific growth anomaly prevalence between years declined; however, this was either due to the complete death of the GAs (so that technically the colony no longer possessed any GAs and could not be counted as diseased) or due to entire colony mortality; no GA experiencing tissue loss was ever seen to heal. The late 2009 El Niño meant that sea-surface temperatures increased across the eastern and central equatorial Pacific (NOAA 2010) and that mean temperatures at Palmyra were approximately 1.5°C higher in late 2009 than in late 2008, leading to a mild coral bleaching event (Williams et al. 2010b). DN in the zoanthid *Palythoa* contributed the most to the inter-annual increase in disease prevalence at Palmyra, suggesting that the etiology of this disease may be positively associated with temperature. We saw no clear inter-annual patterns in GA prevalence, with prevalence in some genera increasing and prevalence in others decreasing. Our findings, therefore, support growing evidence that the more acute tissue loss diseases seem to respond to increases in temperature but that more chronik diseases, such as growth anomalies, do not, or at least not in the same manner (Williams et al. 2010a, Aeby et al. 2011). However, many other environmental factors change with season, so assuming temperature alone, or even at all, was responsible for the increase in *Palythoa* DN prevalence without further evidence would be a mistake (Sokolow 2009). More sophisticated analyses are required (e.g. Williams et al. 2010b, Abey et al. 2011) to tease apart the likely inter-correlated biotic and abiotic factors that are associated with spatial and temporal prevalence patterns of disease at Palmyra.

In summary, overall disease levels across scleractinian corals, soft corals and zoanthids at Palmyra Atoll were low (0.33%), providing critical baseline levels of disease for more functionally intact reef conditions. However, differences in susceptibility across genera were apparent at Palmyra, meaning that, although

overall disease prevalence within a near-pristine reef environment can appear minimal, particular genera may still be at risk and disease levels therefore require careful monitoring and management. More sophisticated analyses are required to understand the proximate drivers of spatiotemporal patterns of disease prevalence on remote, pristine, or near-pristine coral reefs, and further information about disease etiology will be essential for the full interpretation of disease patterns at Palmyra.

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