CORAL HEALTH AND DISEASE ASSESSMENT IN THE U.S. PACIFIC REMOTE ISLAND AREAS

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ABSTRACT

During 2006 and 2007, the first comprehensive, quantitative coral disease assessment was conducted at seven coral islands and atolls in the central Pacific: Johnston, Palmyra, Kingman, and Wake Atolls, and Howland, Baker, and Jarvis Islands. Collectively, they are referred to as Pacific Remote Island Areas (PRIA), spanning over 4000 km and nearly 20° latitude. Distribution and prevalence of disease were determined from 25-m belt transects at 80 sites, covering an area of over 22,000 m^2 of reef habitat. Six broad disease categories were recognized affecting 12 different scleractinian genera; prevalence was computed based on the estimated total number of coral colonies per survey site. The most geographically and taxonomically widespread disease was skeletal growth anomalies detected at nearly 40% of sites and on six different anthozoan genera. In contrast, the most prevalent disease was acute tissue loss (or white syndrome) which was hosted by four scleractinian genera, including *Acropora*, *Montipora*, *Goniastrea*, and *Platygyra*. Pigmentation response and other sublethal lesions, such as algal and cyanophyte infections, and tube-worm infestations, were infrequent and occurred in low prevalences. The overall abundance of coral diseases in the U.S. PRIA was low; however, patterns of prevalence varied among coral genera and indicated a few taxa were disproportionately affected by disease, namely the Acroporidae and Poritidae. Of potential concern is white syndrome, which results in severe and rapid tissue loss, particularly on the tabular *Acropora cytherea* (Dana, 1846) at Johnston Atoll.

Diseases, the result of an interactive process between a host, an agent, and the environment (disease triangle; Wobeser, 2006), are an influential force shaping the structure and dynamics of both individual species and living communities at a broad range of ecological, spatial, and temporal scales (Burdon et al., 2006). Pathogens reduce the fitness of individual organisms by killing them, reducing growth and fecundity, or impeding competitive ability. However, pathogens may also prevent competitive exclusion and help maintain species diversity (Gilbert, 2005). Today, the dramatic ecological impacts of disease on coral reef communities are clearly illustrated by the catastrophic, Caribbean-wide *Acropora* white band epizootic (Aronson and Precht, 2001; Bruckner, 2003). Although to a lesser extent, the acute tissue loss disease, or white syndrome, has also resulted in considerable losses of Pacific reef corals in the Great Barrier Reef, the Marshall Islands, and the Northwestern Hawaiian Islands (Aeby et al., 2007; Harvell et al., 2007). Some of the most notable examples of disease impacts on natural communities appear when pathogens and invasives are introduced to novel biological environments (Gilbert and Parker, 2006). Thus, with increasing human activity in the marine environment, we can expect to see escalating evidence of diseases affecting coral species, community dynamics, and ecosystem function.

To date, more than 25 different types of Pacific coral diseases and lesions have been documented in peer-reviewed articles and gray literature (Le-Champion-Alsumard et al., 1995; Miller, 1996; Korrubel and Riegl, 1998; Antonius, 1999; Baird, 2000; Din-

sdale, 2002; Ravindran and Raghukumar, 2002; Willis et al., 2004; Raymundo et al., 2005; Work and Aeby, 2006a,b; Harvell et al., 2007; Vargas-Ángel et al., University of Hawaii/NOAA, unpubl. data). For most coral diseases, paucity of ecological and epidemiological data hinders a clear understanding of disease causation, virulence, and transmissibility; therefore, there is mounting concern about the increasing number and impending ecological effects of these diseases. In addition, it is difficult to fully comprehend the underlying mechanisms of disease without an appreciation for the occurrence and prevalence dynamics in natural, undisturbed communities (Ward and Lafferty, 2004; Aeby, 2006).

The U.S. Pacific Remote Island Areas (PRIA) are a group of isolated and unincorporated coral islets and atolls located in the central Pacific Ocean that is not within the jurisdiction of any other U.S. territory or State (GAO, 1997; U.S. DOI, 2003). Seven of these islands are the primary focus of this discussion: Baker, Howland and Jarvis Islands, Johnston, Palmyra, and Wake Atolls, and Kingman Reef. Six of the seven PRIA in this study are National Wildlife Refuges under the jurisdiction of the U.S. Fish and Wildlife Service, Department of the Interior (DOI). Wake Atoll is also under the control of the DOI but is operated by the U.S. Air Force. Here I examine the spatial distribution and prevalence of coral diseases at these coral islands and atolls, based on Rapid Ecological Assessments (REA surveys) conducted in 2006 and 2007 by the NOAA National Marine Fisheries Service, Pacific Islands Fisheries Science Center (PIFSC) Coral Reef Ecosystem Division (CRED), including first-time quantitative evaluations of disease for Howland, Baker, and Jarvis Islands, Kingman Reef, and Wake Atoll. Because the PRIAs lie beyond the influence of urban centers and attendant human disturbances, this study is relevant in that it provides a measure of disease in natural coral populations and a basis to compare levels of disease prevalence in coral reef environments that are influenced by humans.

Materials and Methods

It is thought that the seven PRIA in this study were uninhabited at the time of their discovery by European and American whalers and explorers; archeological evidence in support of early Polynesian or Micronesian visits has only been established for Howland and Baker Islands (Emory, 1934; Shun, 1987; USFWS, 2007a,b,c). In the middle to late 19th century, active guano mining occurred at Howland, Baker, and Jarvis Island, and Johnston Atoll. No mining occurred at Palmyra Atoll because it was too wet for guano accumulation or at Kingman Reef since it is mostly submerged and therefore lacked guano (Maragos et al., 2008; USFWS, 2007a,b). Notwithstanding, all of the PRIA (except Wake Atoll) were claimed for the United States under the Island Guano Act of 1856. Wake Atoll was occupied and claimed for the United States during the Spanish American War (Lobel and Lobel, 2008). During the mid-to-late 1930s and early 1940s, and in preparation for World War II, the U.S. Government authorized the construction and operation of naval air bases at Wake, Johnston, and Palmyra, and airstrips at Howland and Baker Islands. Each of these played strategic roles as refueling and assault stations during the Pacific campaign. However, Baker, Howland, and Palmyra were quickly abandoned by the U.S. military after the successful outcome of the war; military activities and occupation at Johnston Atoll ceased in 2003. Currently, five of the seven PRIA are unoccupied, except for caretakers and a dozen or more researchers at Palmyra, and nearly 40 U.S. Army, U.S. Air Force, and contractor personnel at Wake Atoll.

Disease surveys were conducted at 80 REA sites around Howland, Baker, and Jarvis Islands, Palmyra and Johnston Atolls, and Kingman Reef during January–April 2006, (n = 8, 6, 9, 13, 18, 14; respectively), and at Wake Atoll (n = 12) during April–May 2007 (Fig. 1). At each site, at

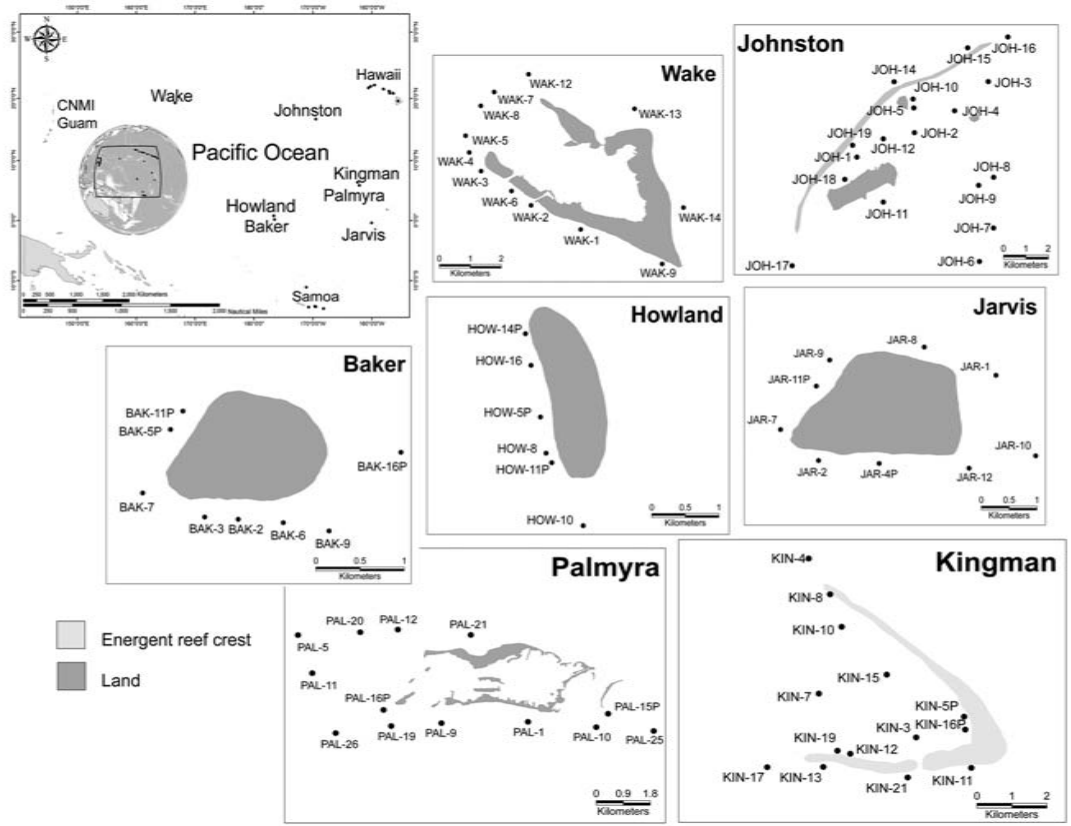

Figure 1. Location of the 2006–2007 coral disease Rapid Ecological Assessment (REA) survey sites for each island/atoll in the U.S. Pacific Remote Island Areas.

depths between 5 and 16 m, a survey methodology was implemented that had been previously developed and tested at Johnston Atoll and the Northwestern Hawaiian Islands (Brainard et al., 2005; Aeby, 2006): two haphazardly selected 25-m transects were positioned end to end, separated by approximately 5 m, and disease surveys were conducted in an area of 1–3 m (total area approx. 100–300 m^2) on each side of each transect line. The smaller survey area was implemented at Johnston Atoll as a result of a high incidence of disease; contrastingly, low incidence of disease at the other PRIA allowed surveys to be expanded to 3 m on each side of the transect line. Within belt transects, each diseased coral colony was carefully examined and identified to the lowest taxonomic level possible and assigned to one of five disease categories, including: skeletal growth anomalies (SGA), acute tissue loss (rapid progressive tissue loss, hereafter referred to as white syndrome; WSY), subacute tissue loss (distinguished from WSY by a slower progression of tissue loss; hereafter referred to as tissue loss; TLS), pigmentation response (bright pink/purple swollen lesions that may be complicated or uncomplicated; PRS), and "other" (OTH), including dark discolorations, algal and cyanophyte infections, tube-worm infestations, and other conditions resulting in impairment of normal function. In assessing coral health condition, colonies exhibiting full, partial, or spotty bleaching (BLE) were also tallied, and thus hereafter, bleaching is treated as a disease state (impairment of normal function; Wobeser, 2006); prevalence values for bleaching were also estimated and are presented alongside the diseases above. Other known Pacific coral diseases including black band, brown band, skeletal eroding band, and the ulcerative white spots, were searched for, but not encountered at any of the survey sites.

To allow for the estimation of disease prevalence, colony counts were conducted by a second diver within 25–50 cm on each side of each 25-m transect line. Within this survey area, all coral colonies were identified to genus level and enumerated. Their maximum diameter was estimated visually and recorded in one of seven size classes (Brainard et al., 2005). Because of a logistical impediment, coral colony counts could not be attained for sites JOH-19 and KIN-16 at Johnston Atoll and Kingman Reef, respectively. Finally, the line-intercept method at 50-cm intervals was used to estimate percent live coral cover.

For each survey site, overall prevalence of disease was computed as the percent of diseased colonies (counts) relative to the estimated total number of colonies at the site, as follows: P_O = [(total no. disease cases × 100) ÷ (colony density × total area surveyed for presence of disease)]. In addition, for each taxon and disease state, total prevalence was estimated as follows: P_T = [(total no. cases of a specific disease for the genus × 100) ÷ (colony density of that specific genus per site × total area surveyed for presence of disease)]. As a result of the lack of coral density data for sites JOH-19 and KIN-16, overall prevalence at these sites was estimated using the island-wide mean colony density for all scleractinians combined; in the same way, total prevalence was estimated using the island-wide mean colony density for each diseased coral genus. Overall prevalence was estimated to evaluate the impact of disease on the entire coral community. Comparatively, total prevalence provides a much closer evaluation of the effects of disease for each affected taxon. These two measurements were computed to offer a complementary approach.

Inter-island/atoll differences in mean overall prevalence were tested using Kruskal-Wallis analysis of variance (ANOVA) on ranks based on small sample size for Howland (n = 6 sites), and a posteriori Dunn's comparisons were performed to establish differences among island/atoll pairs. Mean total prevalence within scleractinian families was estimated based on the pooled putative genus/disease state total prevalence values. A nonparametric ANOVA was used to test for differences in total prevalence among scleractinian families. Additionally, the association between overall disease prevalence and percent live coral cover, and total prevalence per disease state and colony density by family, at each site, for each island, were examined using Spearman Rank Order Correlation analyses.

Results

Disease surveys, covering over 22,000 m^2 of reef habitat were conducted around the seven PRIA. Within this context, 186 individual cases were detected, with Johnston Atoll exhibiting 57% of these cases (Table 1). Coral diseases occurred at all islands but not at all sites; diseases were observed at 43 (54%) of the 80 survey sites. Records for all sites combined yielded a mean overall prevalence of 0.21% (SE 0.06) ranging from a minimum of 0.02% at site KIN-13 at Kingman Reef to a maximum of 3.13% at JOH-18 at Johnston Atoll. Among islands/atolls, mean overall prevalence was the greatest at Johnston, 0.77% (SE 0.23) followed by Wake Atoll, 0.16% (SE 0.05), where diseased corals occurred at 78% and 75% of the sites, respectively (Table 1). Mean overall prevalence for all of the other islands/atolls was an order of magnitude lower, and at these areas, disease conditions were detected at < 50% of the sites surveyed (Table 1). A non-parametric Kruskal-Wallis ANOVA indicated that mean overall prevalence at Johnston Atoll was significantly greater than at all of the other islands ($P < 0.05$; $\chi^2 = 29.0$, df = 6), and differences among the other locations were statistically nonsignificant ($P > 0.05$; Dunn's pairwise multiple comparisons).

Patterns of disease distribution and prevalence also varied among reef zones and within island ecosystems. Although 84% of the disease surveys took place in accessible, relatively protected forereef, backreef, lagoon, and terrace habitats, only five of 14 (36%) wave-exposed, forereef sites surveyed contained disease. Of the 22 lagoon and backreef sites surveyed, 16 (72%) sites contained diseased corals, with Johnston Atoll accounting for 86% of the cases. The two northwestern lagoonal sites, adjacent to Johnston Island (JOH-11 and -18), exhibited the greatest overall prevalence values estimated for any study site (2.8% and 3.13%, respectively). Additionally, 50% of the 44 leeward forereef and terrace sites surveyed contained coral diseases, with Johnston Atoll site JOH-06 on the southern terrace exhibiting an overall prevalence of 2.1%, the greatest for that type of habitat. Additionally, despite the disparity in the sampling effort and disease levels among reef zones, both within and among islands, it was noteworthy that of the seven islands/atolls surveyed, Kingman Reef, Palmyra and Johnston Atolls, and Baker Island indicated a statistically significant, positive association between overall disease prevalence and percent live coral cover (Spearman Rank Order correlations: $r = 0.54, 0.55, 0.65$, and 0.74, respectively; $P < 0.05$).

Of the 186 distinct cases of disease enumerated in this study, skeletal growth anomalies (SGA) comprised 60% of cases, of which nearly 70% were recorded at Johnston Atoll, where this condition attained an atoll-wide mean total prevalence of 3.4%. SGA were also common and prevalent at Wake Atoll, representing 60% of cases atoll-wide and attaining a mean total prevalence of 3.2% (Fig. 2). Mean total prevalence of SGA ranked an order of magnitude lower at the other islands/atolls, with no cases observed at Jarvis Island. Lesions involving white syndrome (WSY), tissue loss (TLS), pigmentation responses (PRS), and "other syndromes" (OTH), represented between 8% and 9% of all disease cases enumerated in this study. WYS and TLS were only observed at Johnston and Wake, and Howland and Kingman, respectively. Mean atoll-wide total prevalence of WSY was as high as 3.6% at Johnston Atoll and 0.36% for Wake Atoll; the former was the greatest mean total prevalence value estimated for any disease state in this study. Mean total prevalence of TLS did not exceed 0.03% for either Johnston or Wake Atolls. Cases of pigmentation response (PRS) were enumerated at all islands/atolls, except for Howland and Baker. At Johnston, PRS at-

Table 1. Summary statistics of coral disease parameters and percent live coral cover for each island/atoll, derived from the 2006 to 2007 Rapid Ecological Assessment disease surveys in the U.S. Pacific Remote Island Areas.

	Johnston	Wake	Baker	Howland	Jarvis	Palmyra	Kingman
Number sites surveyed	18	12	8	6	9	13	14
Total area surveyed (m^2)	1,800	3,125	2,400	600	2,700	3,900	4,200
Number sites with disease (percent of total)	14 (78%)	9 (75%)	3 (38%)	2 (33%)	4 (43%)	6 (46%)	5 (36%)
Total number cases (percent of regional total)	107 (57%)	24 (13%)	4 (2%)	2 (1%)	13 (7%)	18 (10%)	18 (10%)
Number disease states	4	6	2	2	3	4	3
Mean overall prevalence[1] (SE)	0.77 (0.23)	0.16 (0.05)	0.06 (0.01)	0.03 (0.02)	0.04 (0.02)	0.04 (0.01)	0.04 (0.01)
Mean total prevalence [2] (SE)	8.49 (2.39)	3.92 (1.97)	0.05 (0.01)	0.43 (0.39)	0.25 (0.13)	1.39 (1.01)	0.85 (0.63)
Mean percent live coral cover (SE)	31.0 (4.8)	29.6 (3.3)	30.8 (4.7)	51.4 (5.6)	35.2 (10.5)	26.8 (5.2)	32.9 (5.4)
Taxa affected by disease (total)*	a, j, l, m (4)	a, b, g, j, k, m (6)	a, d (2)	j, l (2)	i, j, l, m (3)	d, f, h, m (4)	a, j, m (3)

* a: *Acropora*; b: *Astreopora*; c: *Favia*; d: *Favites*; f: *Gardineroseris*; g: *Goniastrea*; h: *Hydnophora*; i: *Leptastrea*; j: *Monitpora*; k: *Platygyra*; l: *Pocillopora*; m: *Porites*.
[1]: Based on the estimated total number of colonies per site; [2]: based on the estimated total number of colonies per genus/disease state at each site (see methods for details).

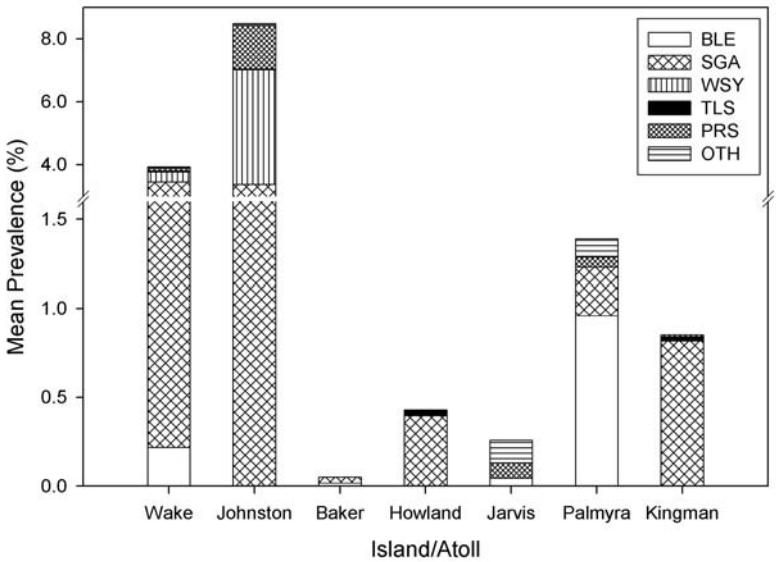

Figure 2. Mean total prevalence of coral disease for each of the seven U.S. Pacific Remote Island Areas, computed as the percentage of cases relative to the estimated number of colonies within each genus in the survey area (no. colonies m^{-2}; see methods for details). BLE: bleaching; SGA: skeletal growth anomalies; WSY: white syndrome; TLS: tissue loss; PRS: pigmentation responses; and OTH: other lesions and syndromes.

tained a mean total prevalence value of 1.4% and ranged between 0.01% and 0.09% for the other islands/atolls. OTH were also observed at all islands/atolls, except for Kingman, Howland, and Baker. Mean island/atoll-wide total prevalences ranged between 0.05% and 0.12%. Finally, cases of bleaching (BLE) were recorded at Jarvis and Baker Islands, and Palmyra and Wake Atolls. All cases were focal and mild, except for the one case at Palmyra which was moderate and widespread.

Disease conditions were observed in 12 scleractinian genera belonging to six families: Acroporidae, Agariciidae, Faviidae, Merulinidae, Pocilloporidae, and Poritidae (Tables 1 and 2). For all sites combined, mean total prevalence varied among scleractinian families (Kruskal-Wallis ANOVA: $\chi^2 = 53.9$, df = 5, $P < 0.001$; Dunn's pairwise comparisons; Acroporidae > Agariciidae, Faviidae, and Merulinidae), being the greatest on the Acroporidae, 1.7% (SE 0.49), followed by Poritidae, 0.61% (SE 0.33), and Pocilloporidae, 0.37% (SE 0.31), to mean values of less than 0.16% in each of the other three families (Table 3). Mean total prevalence was the greatest in the Acroporidae at all islands/atolls, except at Palmyra Atoll, where bleaching (BLE) of Agariciidae ranked first. Of the 186 cases of disease and bleaching recorded in this study, 62% occurred in the Acroporidae. This was the only taxon to exhibit a statistically significant, positive association between colony density and overall prevalence of disease (SGA and WSY; Spearman Rank Order correlations: $r = 0.36$ and 0.35, respectively, $P < 0.05$). In addition, more than 87% of disease cases in the Acroporidae were enumerated at Johnston Atoll where mean total prevalence of 5.5% (SE 1.5) was the greatest found in this study (Fig. 3). This was mainly a result of the high prevalence of WSY. Aside from WSY and SGA, the Acroporidae were also host to TLS, this latter on the genus *Montipora*.

Table 2. Number of cases and distribution of diseases among scleractinian genera in the U.S. Pacific Remote Island Areas. BLE: bleaching; SGA: skeletal growth anomalies; WSY: white syndrome; TLS: tissue loss; PRS: pigmentation responses; and OTH: other lesions and syndromes.

Family	Genus	BLE	SGA	WSY	TLS	PRS	OTH	Total
Acroporidae	*Acropora*	1	16	16				33
	Astreopora		2					2
	Montipora		71	9	2		4	86
Pocilloporidae	*Pocillopora*	2	3		1			6
Agariciidae	*Gardineroseris*	1						1
Faviidae	*Favia*		1					1
	Favites	1						1
	Goniastrea	2		1			2	5
	Leptastrea						2	2
	Platygyra			1				1
Merulinidae	*Hydnophora*						1	1
Poritidae	*Porites*	3	19		5	14	6	47
Totals		**10**	**112**	**27**	**8**	**14**	**15**	**186**

The family Poritidae hosted five (SGA, TLS, BLE, PRS, and OTH) of the six major disease categories, distributed throughout five of the seven islands/atolls (Fig. 3). For this family, mean total prevalence was relatively high at Wake Atoll, 1.17% (SE 0.56) where lesions on poritids involved, in order of numerical abundance, skeletal growth anomalies (SGA), bleaching (BLE), and pigmentation responses (PRS). PRS was the most common affliction of this family at Palmyra, Johnston, and Jarvis, but not at Kingman, where SGA and TLS were more prevalent. The family Pocilloporidae hosted three diseases, SGA, BLE, and TLS at Johnston and Palmyra Atolls and Jarvis and Howland Islands (Fig. 3). No other islands/atolls exhibited diseases in this scleractinian family. Interestingly, the relatively high total prevalence of SGA in *Pocillopora* at Johnston Atoll (1.6%) was based on low numerical abundance of this genus at the site where diseased specimens were encountered. All six major disease categories were observed in the Faviidae (Fig. 3), but affected a low proportion of corals (< 1%) except for WSY on *Platygyra* at Wake (3.9%) and SGA on *Favia* at Palmyra Atoll (3.1%). The families Agariciidae and Merulinidae were host to one disease category each; BLE on *Gardineroseris* and OTH (dark discolorations) on *Hydnophora*, both detected at Palmyra Atoll (Fig. 3).

Table 3. Mean total prevalence of disease within scleractinian families in the U.S. Pacific Remote Island Areas.

Taxon	Mean	SD	Max	Min
Acroporidae	1.66	0.49	22.5	0
Agariciidae	0.15	0.15	12.5	0
Faviidae	0.12	0.06	3.9	0
Merulinidae	0.02	0.02	1.3	0
Pocilloporidae	0.37	0.31	25	0
Poritidae	0.61	0.33	25	0

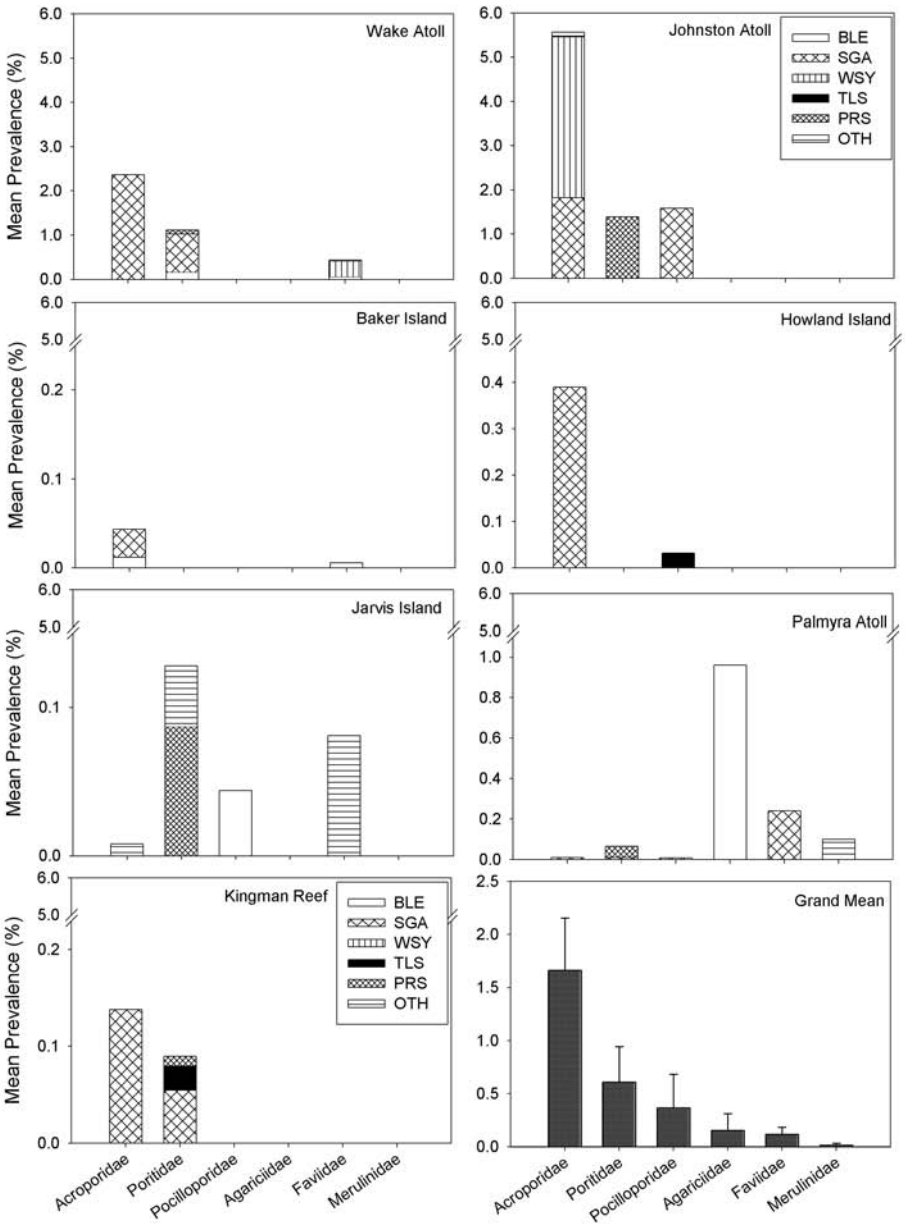

Figure 3. Mean total prevalence of coral disease in six scleractinian families at each of the seven U.S. Pacific Remote Island Areas in this study. Prevalence within scleractinian families was based on the pooled total prevalence estimates per genus/disease state in each survey area (see methods for details). BLE: bleaching; SGA: skeletal growth anomalies; WSY: white syndrome; TLS: tissue loss; PRS: pigmentation responses; and OTH: other lesions and syndromes.

Discussion

Spatial Patterns of Disease Distribution and Prevalence.—There is a growing concern about the threat of increased prevalence, geographic distribution, and host range of coral diseases on Indo-Pacific reefs, including the Great Barrier Reef (GBR), Marshall Islands, the Red Sea, Philippines, East Africa, and the Hawaiian Archipelago (Harvell et al., 2004; Loya, 2004; Willis et al., 2004; Golbuu et al., 2005; Aeby, 2006; Kaczmarsky, 2006; Harvell et al., 2007). While the evidence of increasing disease incidence for Caribbean reefs is strong (Weil et al., 2006), the lack of baseline data often hinders the elucidation of such spatial and temporal trends for many Pacific regions (Page and Willis, 2006; but see Antonius and Lipscomb, 2001; Willis et al., 2004). This study presents disease prevalence data for seven remote islands/atolls, six of which currently lie beyond the influence of attendant anthropogenic disturbances. Thus, it provides a basis against which to compare levels of prevalence in human impacted coral reef environments. In addition, this is the first time quantitative coral disease assessments have been conducted at the remote Wake Atoll, Kingman Reef, and Howland, Baker, and Jarvis Islands, thus, offering a baseline assessment of coral disease in isolated, relatively undisturbed coral reef communities. For Johnston Atoll, lesion types and prevalence values documented in this study conform to a prior semi-quantitative coral health assessment conducted in 2004 by G. S. Aeby (Brainard et al., 2005).

The detection of disease at 54% of the reef sites surveyed (n = 80), spanning a distance of over 4000 km and a nearly 20° range of latitude, supports the notion that background levels of diseases are a natural component of tropical coral reef-building communities. The estimated region-wide mean overall prevalence of 0.21% for the PRIA is in line with the findings of Aeby (2006) for the Northwestern Hawaiian Islands, another group of remote, relatively undisturbed coral reef communities. Six major disease categories affecting a total of 12 scleractinian genera (6 families) were detected in this study. Except for the absence of band diseases in the PRIA, the results of this study compare to the types of disease conditions reported for the Great Barrier Reef (Willis et al., 2004), the Philippines (Raymundo et al., 2005; Kaczmarsky, 2006), the Red Sea (Loya, 2004; Winkler et al., 2004), and American Samoa (Work and Rameyer, 2005; Brainard et al., 2008; Vargas-Ángel et al., University of Hawaii/NOAA, unpubl. data). In the Hawaiian Archipelago, Work and Aeby (2006a) categorized as many as 16 different disease states based on lesion type and coral host genus. In the present study, a pragmatic consideration was made; all coral genera exhibiting comparable gross morphological changes were grouped into broad disease categories. Although it is ideal to include the host species or genus into each disease name, it becomes cumbersome with more than 200 species and 40 genera of Indo-Pacific corals in the PRIA.

Patterns of disease distribution and abundance indicated that some disease states were abundant and widespread, while others were uncommon and rare. For example, skeletal growth anomalies represented more than 60% of all disease cases, and were present at all islands/atolls except for Jarvis Island. SGA were also noted in all reef zones and affected the greatest number of coral genera; six, namely: *Acropora*, *Astreopora*, *Favia*, *Montipora*, *Pocillopora*, and *Porites*. In contrast, bleaching (BLE) which also occurred on a wide range of coral genera (*Acropora*, *Favia*, *Gardineroseris*, *Goniastrea*, *Pocillopora*, and *Porites*), was rare; only 4% of cases involved this condition,

and most cases were mild and focal. The other diseases, white syndrome, subacute tissue loss, and pigmentation responses (WSY, TLS, and PRS) were less numerically abundant, each accounting for ~10% of all cases.

Occurrence and abundance of disease also varied among and within islands/atolls and habitat types. For example, Johnston Atoll exhibited 57% cases of disease, while only 1% of cases occurred at Howland Island; 88% of the cases occurred in the lagoon within Johnston. Despite the disproportionate in the sampling effort among reef zones (which was dictated by the site accessibility), the number of sites exhibiting disease was 50% greater in lagoonal settings compared to wave-exposed areas. Exposed sites exhibited only three disease types (SGA, BLE, and WSY), whereas protected backreef, forereef, and lagoonal sites contained five and six disease categories, respectively. Exposed sites also exhibited lower percent live coral cover and species diversity. As a result of unique environmental conditions affecting the coral community structure and dynamics in each reef zone, structural community differences may determine distribution and levels of coral disease (Aeby, 2006).

Spatial patterns of disease occurrence and prevalence have also been noted in other geographical regions. For example, Aeby (2006) reported that in the Northwestern Hawaiian Islands, *Montipora* tissue loss lesions were most common at backreef sites at Midway Atoll, whereas *Acropora* white syndrome and growth anomalies were most abundant in the lagoon at French Frigate Shoals. Aeby (2006) suggests that it is the taxon of corals found on a reef, and not the island on which it occurs, that primarily affects the types and levels of disease that will occur. In this study, diseases were classified in broad categories, and thus, no host-specific syndromes were identified. Instead, patterns of disease occurrence appeared to be island/atoll-specific, rather than host-specific. For example, montiporid corals were dominant at Johnston and Wake Atolls and Jarvis Island, but only at Johnston were *Montipora* diseases common; at Wake and Jarvis, diseases were more numerous on *Porites* and *Pocillopora*, respectively. Factors including, but not limited to pathogen distribution and life history, environmental conditions and disturbances, host susceptibility, and disease transmissibility and virulence, may also determine the occurrence and prevalence of disease (Wobeser, 2006).

Patterns of Disease Prevalence Among Scleractinian Families.—Patterns of disease prevalence among families indicated that the Acroporidae was the most susceptible. Willis et al. (2004) postulated that fast growing corals such as the Acroporidae and Pocilloporidae may have developed less resistance to disease as a consequence of life history strategies that channel resources into growth for space monopolization rather than into maintenance activities. Their study also suggested that long-lived, slow-growing massive corals seem to be more specialized in confrontational interactions and may have evolved greater resistance to disease. In the present study, the Acroporidae hosted the greatest number of disease cases and was the only taxon for which numerical colony density exhibited a moderate, statistically significant positive association with overall prevalence of disease. This was the result of a high concentration of Acroporidae SGA and WSY cases at Johnston Atoll, and an almost complete absence elsewhere. However, within Johnston Atoll, high total disease prevalence values resulted from a site-specific low colony numerical abundance of *Acropora* and an elevated number of disease cases in this host genus. Similar low numerical abundances of *Acropora* at Kingman and Wake resulted in elevated disease prevalence for the Acroporidae at these two atolls.

Comparable trends have also been noted for the Northwestern Hawaiian Islands, where despite the dominance of the Poritidae (~ 64%), the greatest disease prevalence values occur in the Acroporidae (> 2.5%; Friedlander et al., 2005; Aeby, 2006; Vargas-Ángel, University of Hawaii/NOAA, unpubl. data). In both the PRIA and the Northwestern Hawaiian Islands, skeletal growth anomalies and white syndrome appeared to be the most frequent afflictions to the Acroporidae. In the Northwestern Hawaiian Islands, the genus *Acropora* was the main host for these two diseases; at Johnston Atoll, WSY predominantly affected *Acropora*, while SGA were more numerous on *Montipora*. Disease assessments at other Pacific reefs, including the Marshall Islands and the GBR, also indicate that within the Acroporidae, the genus *Acropora* may be disproportionately affected by white syndrome, particularly tabular species, such as *Acropora cytherea* (Dana, 1846) and *Acropora clathrata* (Brook, 1891). Digitate, caespitose, and branching species of *Acropora* seem to be less affected by this disease (Willis et al., 2004).

Results also indicated that two major families, Poritidae and Faviidae, hosted the greatest number of diseases (five and six diseases, respectively). Although faviids represented an important component of the scleractinian fauna at all islands/atolls, diseases were not common on this family. Only at Wake Atoll did 25% of all cases occur on *Platygyra* and *Goniastrea*. Comparatively, at this same atoll, the Poritidae hosted > 45% of cases, particularly skeletal growth anomalies. This latter family also hosted the greatest number of disease cases at Kingman and Palmyra Atolls and Jarvis Island, but prevalence values were low because of high numerical abundance of poritids at the sites where disease specimens of this family occurred. Recent studies indicate an escalating number of diseases and levels of prevalence affecting the Pacific Poritidae and Faviidae including white syndrome-/tissue loss-type lesions, necrotizing disease, skeletal growth anomalies, band diseases (black band, yellow band, and skeletal eroding band), bleaching, and other infections/infestations including protozoa, cyanobacteria, algae, fungi, sponges, and parasitic interactions with other invertebrates (Sutherland et al., 2004; Willis et al., 2004; Raymundo et al., 2005; Aeby, 2006; Kaczmarsky, 2006). In the central Philippines, *Porites* skeletal growth anomalies and ulcerative white spot syndrome (PUWS) occur in prevalences as high as 39.1 and 53.7%, respectively (Kaczmarsky, 2006). While in lower prevalences, in the main Hawaiian Islands the genus *Porites* also commonly hosts SGA, PRS, and a parasitic diagenic trematode (i.e., trematodiasis; see Aeby, 2006). Although these latter conditions alone generally do not result in acute and rapid tissue deterioration or colony death, they both affect the growth and reproductive potential of the affected colonies (Friedlander et al., 2005). Increased disease occurrence and prevalence in the Poritidae may be a source of potential concern given that the Poritidae are important contributors to reef building and structural dynamics in many Pacific coral reefs.

The Pocilloporidae, Agariciidae, and Meruliniidae were host to one to three diseases each (BLE, SGA, and OTH), with bleaching affecting corals in the Pocilloporidae and Agariciidae and other diseases (mainly discolorations other than bleaching) affecting the Meruliniidae. Like Faviids, Pocilloporids were also common in many locales, particularly at Palmyra (~ 20%) but total disease prevalence was low (0.35%). Aeby (2006) also noted low levels of disease for the Hawaiian *Pocillopora* (< 0.1%). Contrastingly, in the GBR, Willis et al. (2004) reported high disease prevalence on the Pocilloporidae (~10%) with infections in some areas (e.g., Lizard Island) exceeding that of the Acroporidae. In the GBR, the main afflictions of the *Pocillopora* were

skeletal eroding band and black band disease. To date, neither disease state has been noted at any of the PRIA.

Johnston Atoll.—This regional survey revealed low levels of disease at all islands/atolls except for Johnston Atoll, where the Acroporidae was disproportionately affected by disease, predominantly white syndrome but also growth anomalies. A mean total prevalence of disease for *Acropora* at Johnston of 4.97% (SE 1.39) (range 0%–20%) is comparable to the mean total prevalence values recently reported for Caribbean sites (Weil, 2004) and reefs in the northern region of the GBR (Willis et al., 2004; Harvell et al., 2007). This is worrisome given the fact that the Caribbean surveys included some historically disease-affected areas in Jamaica and Mexico, and the GBR surveys targeted regions identified as having the highest known abundance of white syndrome. Of all the PRIA in this study, Johnston Atoll has been subject to the greatest levels of anthropogenic disturbance, including extensive dredging and construction to build up for naval air military activity, gunfire training, explosive detonation, and heavy metal, radioactive, and organic contamination (Lobel and Kerr, 2002). From the late 1930s throughout the mid 1940s, Johnston Atoll served as a strategic naval, military base for refueling airplanes and submarines in defense of the Hawaiian Islands. After WWII, military operations at Johnston Atoll diminished until the late 1950s when the atoll was used for atmospheric nuclear testing. Later in the 1970s, the atoll became a storage site for obsolete chemical warfare agents including nerve gas, mustard gas, and herbicides (Agent Orange) that originated from U.S. usage in the Vietnam War. Military activities and occupation at Johnston Atoll ceased in 2003 after the Chemical Agent Disposal Program had concluded; today, Johnston Atoll is abandoned (Lobel and Lobel, 2008).

Interestingly, our results revealed that the highest levels of disease prevalence for all Johnston sites combined were concentrated at JOH-05 and JOH-10 (12.5% and 20.0%, respectively) east of Akau (North) Island (PCB contamination; see Lobel and Lobel, 2008), as well as JOH-18 and JOH-19 (9.0% and 10.7%, respectively) north-northwest of Johnston Island (former Agent Orange storage site, open burn pit and trash dump, and fire training and explosive detonation area; Lobel and Lobel, 2008). In addition, ecological monitoring at 12 permanent stations indicates that stands of *Acropora cytherea* at Johnston Atoll have undergone > 50% population reduction between 2004 and 2006 (Maragos and PIFSC-CRED, unpubl. data). Although the association between prevalence of disease and environmental stress still remains largely unknown, research supports a connection between environmental deterioration and diminished coral immune capacity (Vargas-Ángel et al., 2007). In this study, except for one case reported at Wake Atoll, all lesions involving WSY occurred at Johnston Atoll. It is plausible that severe levels of anthropogenic impacts, including high levels of sedimentation in concert with heavy metal, PCB, and other organic contamination, have played a role determining the current levels of disease, coral-pathogen interaction dynamics, and thus, community structure at Johnston Atoll (Rogers, 1990; Guzmán and Jiménez, 1992; Kerr Lobel, 2005).

Alternatively, the occurrence of outbreaks of white syndrome in remote, less human-impacted sites, such as the outer-shelf reefs in the GBR (Willis et al., 2004), makes a case for the lack of association between direct human impacts and the abundance of this disease. Evidence from the GBR suggests that prevalence of white syndrome may be correlated with elevated temperatures and host density (Selig et al., 2006; Bruno et al., 2007). The propensity of some tabular *Acropora* species to host

white syndrome is only paralleled by the Caribbean *Acropora* white band epizootic. Further research pertaining to the etiology and environmental correlates for this disease are indicated.

In summary, this study represents the first comprehensive assessment of coral health and disease in the U.S. PRIA. Except for Johnston Atoll, occurrence and abundance of disease in these areas are low. Elevated levels of disease at Johnston Atoll suggest a possible association between prevalence of disease and environmental disturbances. Because coral diseases may act synergistically with other stressors, there is reason to believe that management practices may be able to influence the impact of disease. Thus, periodic monitoring of these remote areas coupled with continued research can allow a better understanding of natural coral disease dynamics, and therefore help managers formulate informed decisions regarding the potential risks to specific coral populations.

Acknowledgments

This work was supported by the NOAA Coral Reef Conservation Program. Institutional, logistic, and financial support were provided by the NOAA Pacific Islands Fisheries Science Center's Coral Reef Ecosystem Division. Institutional and logistical assistance were also provided by the U.S. Fish and Wildlife Service (Howland, Baker, Jarvis, Palmyra, and Kingman National Wildlife Refuges) and the U.S. Air Force (Wake Atoll). The officers and crew of the NOAA Ship Hi`ialakai are gratefully acknowledged for support with dive and small boat operations. J. Maragos and J. Kenyon conducted field colony counts, B. DeJoseph assisted with the computation of coral generic densities, and J. Helyer with the preparation of site maps. The author also wants to thank R. Schroeder, A. Hall, P. Vroom, R. Brainard, and two anonymous reviewers, whose comments substantially improved this manuscript.

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Date Submitted: 10 September, 2008.
Date Accepted: 8 January, 2009.
Available Online: 11 February, 2009.

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