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Scleractinian coral diseases in south Florida: incidence, species susceptibility, and mortality

Jill L. Borger

Rosenstiel School of Marine and Atmospheric Science, Division of Marine Biology and Fisheries, 4600 Rickenbacker Causeway, Miami, Florida 33149, USA

ABSTRACT: There are limited quantitative data available documenting the natural, or non-epizootic, occurrence of scleractinian coral diseases over multiple years. Individual coral colonies exhibiting black band disease (BBD), white plague (WP), dark spots syndrome (DSS), and white band disease (WBD) were monitored 3 times per year on 5 south Florida reefs over a 2 yr period. Surveys included measurements of coral population composition, coral diversity, disease type, coral species affected, colony size, percent of colony affected, and the number of lesions or active infections per colony. Data on re-infections of the same colonies, multiple infections per colony, disease duration, disease-associated tissue mortality, and coral recruitment are also presented. A total of 674 coral colonies exhibiting coral diseases were tagged and monitored. DSS was the most common syndrome (n = 620 infected colonies), but BBD and WP infections caused the largest amount of coral tissue death. The only disease that exhibited a linear increase in incidence with elevated temperature was BBD. DSS and BBD were the most persistent conditions, and WP infections were comparatively short-lived, with obvious signs of disease typically disappearing after 2 to 3 mo. The only disease that caused total colony death as oppposed to partial mortality during the survey period was WBD. WP and DSS incidence was significantly positively correlated with the relative frequency of the species most commonly affected by each disease at each study site. Of the 61 colonies examined in the recruitment survey, only 5 scleractinian coral recruits were identified. The most commonly recorded colonizer of exposed coral skeleton was filamentous/turf algae, thus indicating the potential for a shift towards algal-dominated reef communities.

KEY WORDS: Coral diseases · Coral mortality · Florida Keys

INTRODUCTION

The frequency of occurrence, and the geographic and host species range, of scleractinian coral diseases has increased in the past decade (Santavy & Peters 1997, Goreau et al. 1998, Hayes & Goreau 1998, Richardson 1998, Harvell et al. 1999, Williams & Bunkley-Williams 2000, Porter & Tougas 2001). There is a rising concern over the ability of diseases to cause species shifts, restructure coral populations, and cause a decrease in coral species diversity (Aronson & Precht 1997, Porter & Tougas 2001, Bruckner 2002). In order to understand the role of coral diseases in effecting changes in community structure, it is necessary to quantify their temporal and spatial dynamics over multiple year time frames. The data available are usually limited in temporal scope and involve an examination of specific disease outbreaks (Dustan 1977, Gladfelter 1982, Feingold 1988, Dustan 1993, Peters 1993, Kuta & Richardson 1996, Bruckner et al. 1997, Richardson et al. 1998a,b, Nugues 2002, Miller et al. 2003). There is a general paucity of information on the non-epizootic occurrence of coral diseases, as well as on the duration and severity of specific diseases in individual colonies (Bruckner 2002). Without this critical information, the management of coral reef ecosystems will be deficient. This study is a detailed survey of coral diseases over large, protected reef areas in Biscayne

*Email: jborger@earthlink.net

National Park and the Florida Keys National Marine Sanctuary in the upper Florida Keys reef tract (USA). Individual diseased colonies were tagged and monitored over time, allowing for the disease- and species-specific quantification of survival and mortality.

MATERIALS AND METHODS

Five reef sites in south Florida were surveyed for black band disease (BBD) (e.g. Antonius 1973, Rützler & Santavy 1983, Rützler et al. 1983, Carlton & Richardson 1995, Richardson 1996, Antonius 1988), white plague (WP) (e.g. Dustan 1977, Richardson et al. 1998a,b), dark spots syndrome (DSS) (e.g. Garzon-Ferreira & Gil-Agudelo 1998, Goreau et al. 1998, Cervino et al. 2001, Borger 2005), and white band disease (WBD) (e.g. Gladfelter 1982, Aronson & Precht 1997, 2001) in 2001 (April, July and September) and 2002 (April, July and September). Diseases/syndromes were identified utilizing published descriptions of each condition (Dustan 1977, Santavy & Peters 1997, Richardson et al. 1998a,b, Garzón-Ferreira & Gil-Agudelo 1998, Borger 2005) and U.S. government issued (National Oceanic and Atmospheric Administration, NOAA) disease identification cards (Bruckner & Bruckner 1998a,b,c,d). Due to the fact that the different types of WBD and WP are based partly upon disease progression rates, these distinctions were not made in the surveys. Sites were selected randomly from a list of 16 reef sites in Biscayne National Park (BNP) and the Florida Keys National Marine Sanctuary (FKNMS) that were visited between 1998 and 2000 on unrelated field excursions. A pilot study was implemented during the summer of 2000 and spring of 2001 in order to become more familiar with the sites, construct site underwater maps, and to practice the survey methodology. The first complete survey of each site was executed in April 2001, and thus incidence values were acquired for July 2001. All surveys were conducted by a single observer, thereby eliminating inter-observer bias. The sites were (from North to South): (1) Anniversary Reef (BNP), (2) Alina's Reef (BNP), (3) Marker 3 Reef (BNP), (4) Picture Reef (BNP), and (5) Algae Reef (FKNMS) (Fig. 1). Picture Reef was not surveyed in July 2001, and Algae Reef was not surveyed in September 2001 due to adverse field conditions, which made it impractical to survey all of the study sites during those months. All reefs ranged in depth from 3 to 7.5 m and were distributed across approximately 8500 m^2 of reef area.

Coral population parameters (species richness, diversity, and percent cover) were assessed at each site using a quadrat percent cover method. A 1 m^2 PVC quadrat was sub-divided into 100, 10 × 10 cm squares

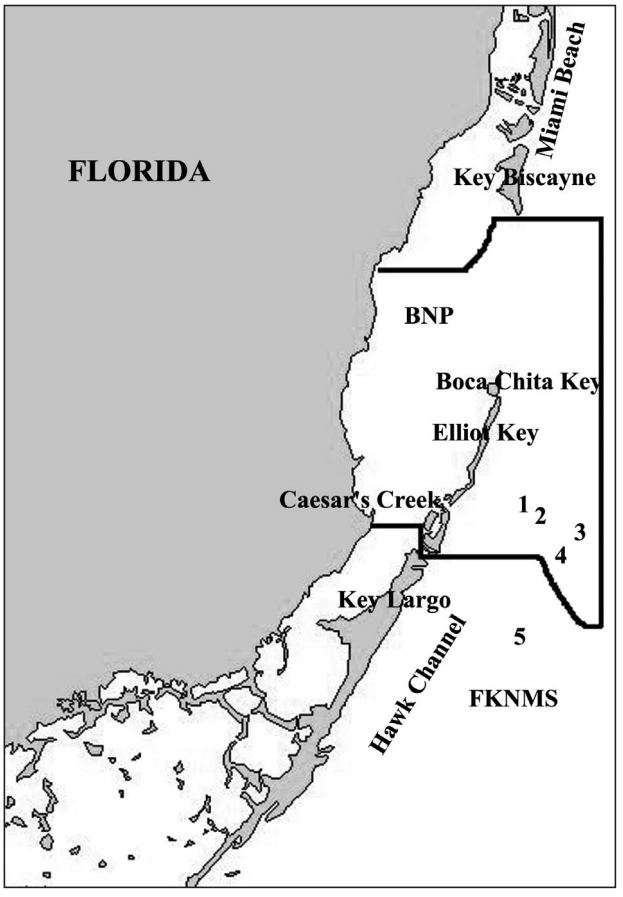

Fig. 1. Site locations in South Florida, USA. (1) Anniversary Reef (BNP), (2) Alina's Reef (BNP), (3) Marker 3 Reef (BNP), (4) Picture Reef (BNP), (5) Algae Reef (FKNMS). BNP = Biscayne National Park and FKNMS = Florida Keys National Marine Sanctuary

with twine. The quadrat was placed randomly upon the reefal substrate at each site 40 times (total area surveyed per site = 40 m^2), using a list of random numbers for swim kicks and compass headings. Coral cover was estimated (visually) by counting the number of 10 × 10 cm subdivisions filled by each species per each 1 m^2. When a species was present within the quadrat but did not fulfill the minimum requirement of 0.5 subdivisions, it was recorded as present ('P') and used in the calculation of diversity indices (Shannon-Wiener Diversity Index; $H'_{max} = \log k$; k = number of species present).

Sites were sampled using SCUBA and surface diving. Site boundaries were established with markers (nails), and a pivoting line search pattern was used to survey the entire area for the presence of diseased coral colonies (range of individual site areas = 991 m^2 to 2158 m^2, see Table 1). The monthly survey of each site took approximately 5 to 7 h. The use of underwater maps facilitated the location of diseased colonies in

subsequent surveys and, in conjunction with the repetition and overlapping inherent in the survey technique, ensured the complete coverage of each site. In this study, disease occurrence is expressed as incidence, which is defined as 'the number of new cases of a specific disease occurring during a certain time period' (Stedman 2000). This definition is suggested for use by the Coral Disease and Health Consortium (National Oceanic and Atmospheric Administration, NOAA) (2003).

In each survey, all colonies exhibiting BBD, WP, DSS or WBD were tagged with roofing nails on non-living areas of the colony, adjacent to active infections. The following information was recorded: (1) type of disease, (2) coral species, (3) colony size, (4) percent of colony affected, and (5) the number of lesions, or active infections, per colony. A *t*-test was utilized to determine whether disease incidence differed among years. The percent of each colony affected by a disease was estimated visually using the following scale: 1 = 1–20%, 2 = 21–40%, 3 = 41–60%, 4 = 61–80% and 5 = 81–100%. An accurate count of the number of lesions in excess of 30 per colony was difficult and time consuming, especially on days with adverse field conditions (e.g. poor visibility and high surge levels). Therefore, when a colony exhibited more than 30 lesions, a category of '>30' was recorded, and a value of 30 was used in calculations. Cases of re-infections (of previously tagged colonies), multiple infections, and infection persistence or resolution were monitored throughout the survey period. Target species, defined as those most commonly affected by each disease, were identified in each survey year.

Colony size measurements were recorded as the maximum diameter multiplied by the maximum height of contiguous live tissue cover (cm^2). For comparison, 25 to 30 colonies of susceptible coral species were randomly measured at each site, regardless of disease status, and are referred to as 'population' corals in figures and text. A Chi-square analysis was used to compare the colony size classes of diseased and population corals. Colony sizes were divided into 5 size classes: (1) 0–100 cm^2 (S, small), (2) 101–1000 cm^2 (SM, small to medium), (3) 1001–4000 cm^2 (M, medium), (4) 4001–5000 cm^2 (ML, medium to large), and (5) > 5001 cm^2 (L, large). The only species with a sufficient number of diseased colonies for comparison was *Siderastrea siderea* (DSS).

In September 2001 and 2002, total disease-related tissue mortality was recorded per infected colony at each site. Measurements were restricted to areas of bare skeleton adjacent to active lesions, with minimal or no algal growth, indicating recent tissue mortality. Disease incidence at each site was compared to relative target species densities, site coral diversity values (Shannon-Wiener Index, *H'*), and site percent coral cover, using the Pearson Product Moment Correlation statistic (r). Temperature data (acquired using a diver's depth gauge and BNP underwater datason database: Endico/YSI Meter, Model 6000, multi-parameter probe) from each survey month were also compared to disease incidence values to determine possible significant relationships using a linear regression statistic (R^2).

In September 2002, all tagged colonies (including some from the 2000 pilot survey) exhibiting disease induced tissue loss were surveyed for the presence of coral recruits on affected skeletal areas. A total of 61 colonies were examined, including colonies affected by each disease (DSS = 34, WP = 16, BBD = 9, WBD = 2). In addition to coral recruits, other general categories of colonizers were recorded and included: (1) filamentous algae, (2) macroalgae (predominantly *Dictyota* spp. and *Halimeda* spp.), (3) sponges, (4) boring sponges (predominantly *Cliona* sp.), and (5) tubicolous polychaetes. These data were collected to test Edmunds' (1991) hypothesis that coral diseases may create primary substrate for the recruitment and colonization of other scleractinian corals.

RESULTS

During the months surveyed from July 2001 to September 2002, there were a total of 674 diseased coral colonies, or 8 diseased coral colonies per 100 m^2 of reef area. The estimated percent coral cover at all sites combined was 7.8% (±2.9 SD, n = 5), and the most dominant species were *Porites astreoides*, *Siderastrea siderea*, *P. porites* and members of the *Montastraea* species complex (Table 1). The most common disease/syndrome was DSS (n = 620). *S. siderea* was the species most susceptible to DSS and, therefore, was also the species with the highest number of diseased colonies (n = 609) (Table 2). WP infected a total of 37 colonies, while BBD and WBD infected only 10 and 6 colonies, respectively. The species most commonly affected (i.e. target species) by WP in 2001 and 2002 was *Diploria clivosa*. However, the target species of BBD differed between years: *Diploria strigosa* in 2001 and *Montastraea cavernosa* in 2002. *Acropora cervicornis* was the only species noted with WBD during the survey period.

In 2001, BBD and DSS incidence decreased from July to September, while WP and WBD incidence increased (Fig. 2). In 2002, WP, BBD and DSS incidence increased from April to July. DSS decreased in September, WP increased, and BBD remained the same from July to September. DSS incidence was significantly higher in 2002 than in 2001 (*t*-test, $t = -2.379$,

Table 1. Coral population composition at each site, including % coral cover, diversity (*H'*, Shannon-Wiener Index), and the area of site surveyed. Each species is followed by its relative abundance (%) in 40 m^2 surveyed per site (P: present). Ac: *Acropora cervicornis*, Aa: *Agaricia agaricites*, Ag sp.: *Agaricia* sp., Cn: *Colpophyllia natans*, Dsto: *Dichocoenia stokesi*, Dc: *Diploria clivosa*, Dl: *Diploria labyrinthiformis*, Dstr: *Diploria strigosa*, Ef: *Eusmilia fastigiata*, Mmir: *Madracis mirabilis*, Mar: *Manicina areolata*, Mme: *Meandrina meandrites*, Mann: *Montastraea annularis*, Mc: *Montastraea cavernosa*, Mfav: *Montastraea faveolata*, Mfr: *Montastraea franksi*, Od: *Oculina diffusa*, Pa: *Porites astreoides*, Pp: *Porites porites*, Sr: *Siderastrea radians*, Ss: *Siderastrea siderea*, Sb: *Solenastrea bournoni*, Si: *Stephanocoenia intersepta*

	Anniversary Reef	Alina's Reef	Marker 3 Reef	Picture Reef	Algae Reef
Species (relative abundance, %)	Pa (46.3)	Mann (34.2)	Ss (39.2)	Mann (36.7)	Mfav (24.1)
	Pp (20.2)	Mfav (16.0)	Pp (21.7)	Pp (22.4)	Pa (20.8)
	Ss (18.4)	Ss (13.6)	Pa (7.7)	Dc (12.9)	Ss (17.5)
	Dstr (6.9)	Pa (12.4)	Mann (7.0)	Ss (10.5)	Pp (11.8)
	Dc (3.0)	Pp (8.3)	Si (4.5)	Mfav (3.9)	Cn (9.6)
	Mc (2.3)	Dstr (6.1)	Dl (4.2)	Mc (3.4)	Mc (3.1)
	Dsto (1.5)	Ac (3.6)	Mmir (3.7)	Cn (2.2)	Mann (2.5)
	Mfav (0.5)	Mfr (1.9)	Sb (2.7)	Dstr (2.0)	Dsto (2.3)
	Aa (0.5)	Cn (1.7)	Aa (2.2)	Pa (2.0)	Si (2.3)
	Si (0.4)	Dl (0.6)	Dsto (2.0)	Dsto (2.0)	Dstr (1.9)
	Ac (P)	Si (0.6)	Mfav (1.5)	Mme (1.4)	Aa (1.5)
	Sr (P)	Mc (0.5)	Mme (0.7)	Aa (0.4)	Dl (1.0)
	Dsto (0.3)	Sr (0.7)	Sr (0.2)	Sr (0.6)	
	Aa (0.2)	Mc (0.5)		Sb (0.6)	
	Sr (P)	Od (0.5)		Mme (0.4)	
		Ef (0.3)		Ef (P)	
		Ag sp. (0.3)			
		Cn (0.3)			
		Mar (0.3)			
Coral cover (%)	9.8	11.8	5	6.3	6
Diversity (*H'*)	0.65	0.85	0.86	0.81	0.91
Survey area (m^2)	1835	1821	991	2158	1730

Table 2. Total number of coral colonies of each species affected by each disease/syndrome between July 2001 and September 2002. These values represent newly infected colonies. Species abbreviations: see legend for Table 1

Species	DSS	BBD	WP	WBD
Ss	607	–	2	–
Dc	–	1	16	–
Si	13	–	1	–
Dstr	–	4	3	–
Ac	–	–	–	6
Mc	–	2	3	–
Dsto	–	–	4	–
Mme	–	2	1	–
Cn	–	1	1	–
Pa	–	–	2	–
Pp	–	–	1	–
Mann	–	–	1	–
Aa	–	–	1	–
Dl	–	–	1	–
Total	620	10	37	7

$p < 0.05$, df = 16). However, there were no other significant differences in coral disease incidence among survey years. There was a significant positive, linear relationship between BBD incidence and water temperature ($R^2 = 0.871$, $p < 0.05$, df = 4). WP and WBD incidence was highest during the warmest survey month (September), but the relationship between disease incidence and water temperature was not significant when using a linear regression statistic (R^2).

When compared to other diseases, WBD affected the largest mean percentage of each colony (21 to 40% = estimation mean value = 2 ± 1.9 SD, n = 5) (Table 3), and DSS had the highest mean number of lesions per colony (mean = 17.44 ± 10.72 SD, n = 642). When considering total disease-associated coral tissue mortality (i.e. across all sites and all survey periods), BBD and WP infections contributed to the largest amount of coral tissue death (Table 3). The disease that resulted in the least amount of tissue mortality was DSS. Only 17.9% of all colonies with DSS exhibited related tissue mortality. In most cases, the associated mortality was limited to a very small area of tissue at the center of the DSS lesions that was covered in small tufts of filamentous algae (indicating a slow progression of tissue death). Total tissue mortality increased from 3779.5 cm^2 in 2001 to 10158.5 cm^2 in 2002. The species that experienced the largest total loss of tissue were *Acropora cervicornis* in 2001, and *Diploria strigosa* in 2002 (Table 4).

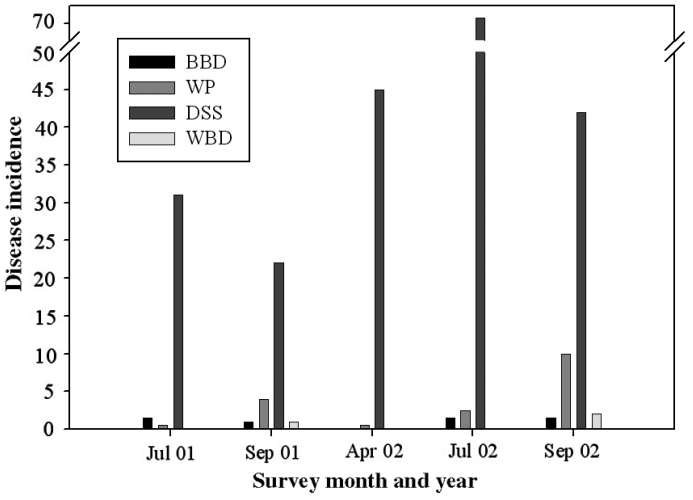

Fig. 2. BBD, WP, DSS and WBD incidence from July 2001 to September 2002. Mean temperature values were: July 2001 = 28.9°C, September 2001 = 28.4°C (range = 21.5 – 30.5°C), April 2002 = 26.4°C, July 2002 = 30.1°C, September 2002 = 30.4°C (range = 21.9 – 32.4°C)

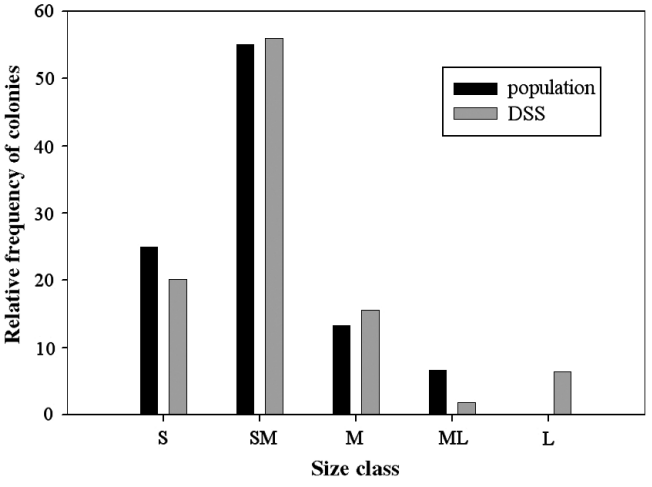

Fig. 3. *Siderastrea siderea*. Relative size class distribution of colonies exhibiting DSS and population colonies (includes measurements of both healthy and affected colonies). (S) 0 – 100 cm^2 (small), (SM) 101 – 1000 cm^2 (small to medium), (M) 1001 – 4000 cm^2 (medium), (ML) 4001 – 5000 cm^2 (medium to large), and (L) > 5001 cm^2 (large)

The size class frequency distributions of the *Siderastrea siderea* population and colonies with DSS were not significantly different ($\chi^2 = 1.72$, $p < 0.001$, df = 3) (Fig. 3). Therefore, DSS did not predominantly affect any size class of *S. siderea* colonies.

DSS infections were more persistent than the other coral diseases. In 2001, 41% of the colonies tagged in July still had DSS lesions in September. In 2002, 50% of those tagged in April were still active in July, and 38% were still active in September. Thirty colonies with DSS in 2001 were noted to still have DSS in 2002. However, since no surveys were conducted in any year between October and March, it was impossible to determine the persistence of DSS between years. Comparatively, WP was much less persistent. Only 1 colony with WP in July 2002 was still active in September. The remaining colonies with WP in 2001 and 2002 were free of active WP infections in each subsequent survey (time frame of approximately 2 to 3 mo). Two of the colonies with WP in 2002 were previously infected by BBD in 2000 (*Montastraea cavernosa*) and by WP in 2001 (*Diplora clivosa*).

BBD was relatively more persistent with most infections noted in July still active in September (2001: 89% and 2002: 67%). Only one *Meandrina meandrites* colony was infected in consecutive years, with disease cessation occurring between the years. WBD was only identified in September of both sur-

Table 3. Mean (± SD) percent of each colony affected by each disease/syndrome, mean ± SD number of lesions per colony, and disease-related tissue mortality for each disease (2001 and 2002 values combined). The mean % affected was estimated using the following scale: 1 = 1–20%, 2 = 21–40%, 3 = 41–60%, 4 = 61–80%, and 5 = 81–100%

Disease	Mean % affected colony^{-1}	Mean # of lesions colony^{-1}	Tissue mortality (cm^2)
BBD	1.4 ± 0.7 (n = 10)	1.6 ± 1.4 (n = 10)	6409
WP	1.8 ± 1.1 (n = 34)	1.2 ± 0.9 (n = 36)	5916
DSS	1.4 ± 0.8 (n = 620)	17.4 ± 10.7 (n = 642)	38
WBD	2.4 ± 1.9 (n = 5)	2 ± 1.7 (n = 5)	1519

Table 4. Total tissue mortality (cm^2) per species in 2001 and 2002. Species abbreviations: see legend for Table 1

Species	Tissue mortality 2001 (cm^2)	Tissue mortality 2002 (cm^2)	Total (cm^2)
Dstr	585	3275	3860
Mme	1350	2232	3582
Mc	–	2061	2061
Ac	1440	79	1519
Dc	182	1116	1298
Dl	–	756	756
Dsto	168	169	337
Cn	–	204	204
Pa	–	102	102
Ss	24	63	88
Aa	–		45
Si	30	45	30
Total	3779	10102	13882

vey years. In at least one case, the disease caused the death of the whole colony.

There was a significant, positive correlation between the relative frequency of identified target species (WP = *Diploria clivosa* and DSS = *Siderastrea siderea*) at each site and the incidence of WP (2002: $r = 0.948$, $p < 0.05$, $df = 3$) and DSS (2002: $r = 0.953$, $p < 0.05$, $df = 3$). There was also a significant, negative correlation between total disease incidence and percent coral cover at each site (2001: $r = -0.888$, $p < 0.05$, $df = 3$). There were no significant relationships between disease incidence and coral diversity.

A total of 5 coral recruits were identified on the 61 colonies examined in the recruitment survey, or 0.03 recruits colony^{-1} yr^{-1}. The recruits included 3 *Siderastrea siderea* and 1 *Eusmilia fastigiata* on a *Colpophyllia natans* colony infected with WP in 2000 and 1 *S. siderea* on a *C. natans* colony infected with WP in 2001. The most commonly recorded colonizer of exposed coral skeletal surface was filamentous algae (n = 59 colonies), followed by macroalgae (n = 21), boring sponges (n = 10), boring polychaetes (n = 2) and sponges (n = 1).

DISCUSSION

This study documents coral disease occurrence and dynamics across 2 consecutive summers. It differs from most other studies of coral disease distribution, in which surveys were conducted in response to specific disease outbreaks. These data indicate that coral diseases were variable in occurrence and species susceptibility between 2001 and 2002 at 5 sites in the upper Florida Keys (BNP and FKNMS). The number of colonies and species exhibiting disease and the total disease-related tissue mortality increased from 2001 to 2002.

Disease dynamics

Dark spots syndrome (DSS)

DSS affected the largest number of coral colonies in 2001 and 2002 when compared to other coral diseases. In contrast, Santavy et al. (2001) recorded DSS infections at most survey sites in the lower Florida Keys reef tract, but it was not the most common coral disease at any of the sites surveyed. The significant, 3-fold increase in DSS incidence between years did not coincide with any obvious, visual environmental changes. Although the number of colonies with DSS was very high, and colonies with DSS displayed the highest number of mean lesions per colony, this syndrome caused the least amount of coral tissue mortality. Thus, the potential ecological impact of DSS is relatively low. The lack of a significant relationship between water temperature and DSS incidence in BNP and FKNMS supports the findings of a study of DSS in Dominica (Borger 2005). However, these findings contradict those proposed by Gil-Agudelo & Garzón-Ferreira (2001), in which DSS incidence exhibited significant seasonal differences that were positively associated with increases in seawater temperature in Colombia. The only species identified with DSS in over 8000 m^2 of south Florida reef area were *Siderastrea siderea* and *Stephanocoenia intersepta*. This contradicts other surveys in south Florida (Santavy et al. 2001) and Colombia (Garzón-Ferreira et al. 2001) in which multiple other scleractinian species were identified with DSS. Therefore, coral disease species susceptibility can vary greatly both inter- and intra-regionally, thereby emphasizing the need for a large range and number of survey areas.

White plague (WP)

WP affected the largest number of species (n = 13 scleractinian species), and *Diploria clivosa* was most commonly affected by WP in both 2001 and 2002. An additional 6 species were identified with WP in 2002 that had not exhibited WP in 2001. By comparison, *D. clivosa* was not identified with WP in a 2-yr study of other south Florida reefs (Santavy et al. 2001). *Dichocoenia stokesi* was identified as the species most commonly affected by WP Type II in the Florida Keys between 1995 and 1997 (Richardson et al. 1998a), and *Siderastrea siderea* was most commonly affected by WP in Dominica (Borger 2003, Borger & Steiner 2005). This further supports the conclusion that coral susceptibility can vary greatly over small spatial and temporal scales.

WP contributed to 10% of the total disease-related coral tissue mortality in 2001 and 55% in 2002. Therefore, its potential ecological impact on reefs is high. However, WP is a transient coral disease. Most infections were inactive by the time of the subsequent survey (approximately 2 to 3 mo later). It is unclear why infections disappear, though Richardson et al. (1998b) noted that WP activity abruptly halted in mid-October during a 1995 outbreak, and suggested that this was due to a decrease in water temperature. Although all cases of WP in BNP and FKNMS caused only partial mortality, 2 colonies were re-infected in consecutive years (with disease cessation between years). In these cases, cumulative coral tissue mortality could likely lead to total colony death after multiple, annual infections.

Although there was no linear relationship between WP incidence and water temperature during the survey period, there were more colonies infected with WP in September of 2001 and 2002 than any other survey months. August and September are the 2 warmest sea temperature months in the south Florida region. This may indicate that coral resistance and response to stress plays a major role in the disease etiology. It is possible that only after the coral has reached a threshold of temperature stress, with exposure over an extended period of time, does it become susceptible to infection by WP pathogens.

Black band disease (BBD)

The number of colonies infected with BBD was comparatively very low across all sites in both years. However, BBD infections accounted for 51% of the total coral tissue mortality in 2001 and 44% in 2002. Therefore, even low levels of BBD can be damaging to coral reefs by contributing to a relatively large amount of coral tissue death. The species noted to be most susceptible to BBD differed between years, which highlights the need for multiple year surveys. This is also important in terms of developing monitoring and management plans for coral reefs, because the effects of coral diseases, in terms of tissue mortality and species susceptibility, are variable across years and within regions.

BBD infections persisted between survey months, and BBD incidence was significantly related to water temperature. *Phormidium corallyticum*, the predominant BBD pathogen, photosynthesizes optimally between 30 and 37°C (Richardson & Kuta 2003). Therefore, the direct relationship between pathogen virulence and temperature may explain the relationship between water temperature and BBD occurrence. There was only 1 colony that was infected with BBD in both 2001 and 2002 (with disease cessation between years). Therefore, the cumulative, yearly effects of BBD were less than those of WP.

White band disease (WBD)

The number of colonies infected with WBD was low across both years. However, the general paucity of *Acropora cervicornis* (the only species noted with WBD) at all sites may have contributed to the low number of infected colonies and a reduction in the critical threshold of host density for maintaining the disease. Despite the low number of colonies infected with WBD, the disease induced tissue mortality and disease severity (% of each colony affected) was very high. WBD was the only coral disease noted to cause total colony death during the survey period, and *A. cervicornis* had the largest amount of tissue death recorded in 2001. WBD has contributed to the regional decline of *A. cervicornis* and *A. palmata* (Aronson & Precht 1997, 2001).

Disease incidence and coral population characteristics

WP and DSS incidence was significantly related to the relative frequencies of target species (WP = *Diploria clivosa* and DSS = *Siderastrea siderea*) at each site, and there was a significant, negative correlation between percent coral cover and total disease incidence. Therefore, the occurrence of some coral diseases was more dependent upon the species composition at each site (in terms of the relative abundance of target species) than total host density. These findings agree with other studies in which the number of diseased colonies per study site was related to the abundance of specific susceptible species (Reigl 2002, Borger 2003, Borger & Steiner 2005).

Coral recruitment

The recruitment survey was implemented in order to test Edmunds' (1991) hypothesis that within a space-limited reef community, coral diseases create primary space for the colonization of other scleractinian corals. The recruitment surveys were limited to the exposed skeletal areas of affected colonies and, therefore, cannot be compared to more extensive surveys of recruitment, in which other substrates were examined for coral recruits. However, in similar studies, Edmunds (2001) found low rates of recruitment onto exposed coral skeleton (0.11 to 0.22 recruits colony^{-1} yr^{-1}) in the US Virgin Islands, and Kuta & Richardson (1997) also found low recruitment rates (0.18 recruits colony^{-1} yr^{-1}) in south Florida. However, the recruitment rate was 3 times lower in this study. These discrepancies could be a result of differences in sampling techniques or may reflect documented regional and local spatio-temporal variation in coral recruitment (e.g. Chiappone & Sullivan 1996, Smith 1997, Dunstan & Johnson 1998, Moulding 2005). The exposed skeletal surface was more frequently colonized by algae, and Patterson et al. (2002) suggested evaluating the role of coral diseases in creating substratum for algal colonization.

Coral disease related tissue mortality does not appear to be important in promoting recruitment and propagation of reef-building corals. The rate of tissue destruction via coral diseases ranges from an average of 3 mm d^{-1} for BBD (Rützler et al. 1983), to 4–5 mm d^{-1}

Table 5. Coral growth rates (mm yr^{-1}) in the Caribbean (Florida, Bahamas and Puerto Rico) as recorded by Ma (1959). LE = lateral expansion, H = expansion in height/length

Species	Growth rate (mm yr^{-1})
Acropora spp.	64 (H)
Agaricia agaricites	7 (LE)
Diploria clivosa	5.5 (H) and 8.5 (LE)
Diploria labyrinthiformis	7.4 (H)
Diploria strigosa	6.7 (H)
Dichocoenia stokesi	3.5 (H)
Montastraea annularis	5.5 (H) and 7–10 (LE)
Montastraea cavernosa	5.7 (H) and 7.5–9 (LE)
Meandrina meandrites	8.4–10.7 (H)
Siderastrea siderea	3.9 (H) and 5–10 (LE)
Stephanocoenia intersepta	1.5–4 (H)

for WBD (Davis et al. 1986), and a maximum of 2 cm d^{-1} for WPII (Richardson et al. 1998a). In comparison, coral growth rates are highly variable and are dependent upon various environmental factors, such as water chemistry, temperature, and turbidity (Hudson 1981, Lidz & Shinn 1991). However, both indirect and direct measurements of coral growth establish average coral growth rates of approximately 6 to 14 mm yr^{-1} of vertical accretion (see Table 5 for a summary of growth rates of specific species as measured by Ma 1959, Stearn & Scoffin 1977, Stearn et al. 1977, Hudson 1981, Davies 1983, Davies et al. 1985, Neumann & Macintyre 1985, and Buddemeier & Smith 1988), with branching species growing at faster rates of several tens of mm yr^{-1} (Ma 1959, Buddemeier & Smith 1988). Therefore, the rate of tissue destruction via coral diseases in the southern Florida reef tract is far greater than the rate of coral growth.

Conclusion

In conclusion, the coral diseases studied in this survey varied greatly over both spatial and temporal scales, especially in terms of species susceptibility. The re-infection of individual coral colonies between years will likely lead to a much greater loss of reef-building species than can be determined from a single-year survey. Thus, multi-year, comprehensive surveys are critical to the understanding of the dynamics of coral diseases and towards the development of sound management practices. In addition, if host resistance and selection remain consistent, there may be a shift in population and community structure towards reefs dominated by more disease-tolerant species. Management efforts should thus be focused on the species noted to be most susceptible to each disease and to the diseases that cause the most coral mortality. The rates of recruitment of scleractinian corals in south Florida do not seem capable of countering the rates of tissue destruction by coral diseases, and there is the potential for a shift in communities dominated by corals to those dominated by algae.

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