THE SPATIAL AND TEMPORAL DYNAMICS OF CORAL DISEASES IN DOMINICA, WEST INDIES

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ABSTRACT

This study involved a detailed documentation of individually tagged, diseased coral colonies at five reef sites in Dominica, West Indies from 2000 to 2002. At each reef site, survey areas were selected, and a pivoting line search pattern was used to identify and tag all scleractinian coral colonies exhibiting white plague (WP), black band disease (BBD), and dark spots syndrome (DSS) in March, June, and August of 3 yrs. There was an overall trend towards increasing disease incidence, but DSS was the only coral syndrome/disease that exhibited a significant increase among years. Conversely, the amount of disease-related tissue mortality (measured in August of each survey year) decreased each year. Coral diseases resulted in over 8 m^2 of coral tissue death during the 3 yr survey period, and 80% of this mortality was attributed to WP infections. The coral species affected by diseases varied in each year, thereby highlighting the need for multi-annual surveys to assess the long-term effects and management of coral diseases. WP and DSS incidence was significantly correlated to the relative frequency of the species most commonly affected by each disease/syndrome, and coral diseases predominantly affected the larger colonies of four susceptible species: *Siderastrea siderea* (Ellis and Solander, 1786), *Montastraea faveolata* (Ellis and Solander, 1786), *Dichocoenia stokesi* (Milne, Edwards and Haime, 1848), and *Colpophyllia natans* (Houttuyn, 1772). DSS progression rates on individual colonies were low (< 0.4 mm d^{-1}), and both BBD and WP progression rates were lower than those documented in other published studies. However, the progression of WP on affected colonies increased with warmer water temperatures. DSS and BBD infections were relatively persistent on individual colonies throughout the yearly surveys, while WP was the most short-lived of the three diseases/syndromes. The re-infection rate of affected colonies between survey years was approximately 25%. Scleractinian coral recruitment rates onto coral skeleton exposed by diseases were low, and the comparatively high occurrence of bioeroders and algae may have contributed to the overall degradation of reef structure or caused a shift toward reef communities dominated by algae.

There are many factors, most likely synergistic, contributing to the decline in coral reefs worldwide. These include, but are not limited to: coastal development, sedimentation, pollution, eutrophication, changes in global temperature, direct anthropogenic and natural degradation (e.g., dredging, boat groundings, and storm impacts), poor land use practices, and coral diseases. Coral diseases have become a persistent source of mortality on many reefs and thus have the potential to be more damaging to reefs than all other threats combined (Hayes and Goreau, 1998). In the 1980s, Bak and Luckhurst (1980) suggested that catastrophic physical disturbances, such as storms and hurricanes, were the predominant variables driving coral mortality. However, by the early 1990s Bythell et al. (1993) proposed that biologically driven disturbances, such as those induced by coral diseases, were the leading cause of coral mortality. They determined that biological agents destroyed more coral tissue than a major hurricane (Hurricane Hugo) on the reefs of the U.S. Virgin Islands.

There is mounting evidence that the emergence of new conditions/diseases is increasing, as well as the local and geographic distribution of diseases and host species

range (Santavy and Peters, 1997; Goreau et al., 1998; Hayes and Goreau, 1998; Richardson, 1998; Harvell et al., 1999; Williams and Bunkley-Williams, 2000; Porter and Tougas, 2001; Porter et al., 2001; Rosenberg and Ben-Haim, 2002). Further, diseases are recognized as a potential driving force in the alteration of community structure, contributing to a decrease in reef coral diversity, and effecting a general decline in the abundance of corals worldwide (Dustan, 1977; Gladfelter, 1982; Hughes, 1994; Holden, 1996; Aronson and Precht, 1997, 2001; Santavy and Peters, 1997; Aronson et al., 1998; Goreau et al., 1998; Greenstein et al., 1998; McClanahan and Muthiga, 1998; Richardson, 1998, Richardson et al., 1998a,b; Harvell et al., 1999; Porter and Tougas, 2001; Porter et al., 2001). One of the gaps in coral disease research is the paucity of information on the multi-annual spatial and temporal variability of coral diseases. Many attempts to quantify diseases are based on infrequent, limited sampling of small areas, and the duration and role of coral disease infections in directly causing coral mortality is not well defined (Bruckner, 2002). Most studies have focused on specific outbreaks and disease epizootics (Gladfelter et al., 1977; Gladfelter, 1982; Feingold, 1988; Edmunds, 1991; Kuta and Richardson, 1996; Bruckner et al., 1997; Richardson et al., 1998a,b) and thus, are not representative of the baseline dynamics of coral diseases. Additionally, there are very limited data available on coral diseases in the eastern Caribbean region (see Steiner and Borger, 2000; Borger, 2003; Steiner, 2003). This study involved a detailed examination of individually tagged, diseased coral colonies over 3 yrs in Dominica, West Indies. The goal was to examine individual colony survival, complete or partial mortality, species susceptibility, multiple infections, re-infection of colonies in successive years, disease persistence over time, and the fate of exposed coral skeleton as a potential substrate for coral recruitment.

Material and Methods

Coral Diseases Surveyed.—Reefs of Dominica, West Indies, were surveyed for three scleractinian coral diseases: black band disease (BBD), white plague (WP), and dark spots syndrome (DSS, otherwise referred to as "dark spots disease"). These diseases were selected because they were the only three diseases recorded in Dominica during the first survey year (along with bleaching, which was not considered in this survey). In subsequent survey years, yellow band syndrome was noted but not included in the surveys. There are different types of WP (Type I and II: Dustan, 1977 and Richardson et al., 1998a,b, respectively), and the distinction is based partly upon disease progression rates and the coral species affected. Progression rates were not measured in every instance of disease, and WP in Dominica appeared to have similar (high) rates of progression typical of Type II but affected the species known to be susceptible to both Type I and Type II (see Dustan, 1977; Richardson et al., 1998). Thus, the distinction between WP types was not made in the surveys. Diseases/syndromes were identified utilizing published descriptions of each condition (Dustan, 1977; Santavy and Peters, 1997; Richardson et al., 1998a,b; Garzón-Ferreira and Gil, 1998; Borger, 2003) and U.S. government issued (National Oceanic and Atmospheric Administration; NOAA) disease identification cards (Bruckner and Bruckner, 1998a–d). In addition, some bacterial samples were sent to S. Viehman at Florida International University for verification of pathogens and molecular profiling (see Viehman, 2002).

Survey Sites.—Dominica, located in the Lesser Antilles or eastern Caribbean region, is a lush, steep island surrounded by a relatively narrow island shelf. In general, the reefs and reef communities of Dominica are subjected to frequent storm impacts (lack of a barrier reef system) and excessive levels of runoff and river input. Five reef sites along the west coast of Dominica were sampled three times (March, June, and August) in 2000–2002, thus constitut-

Figure 1. Dominica, West Indies. Numbers indicate location of study sites along the west coast: 1. Floral Gardens; 2. Salisbury; 3. Tarou Point; 4. Coral Gardens; and 5. Cachacrou.

ing a 3-yr field survey of coral diseases. The five sites, from north to south, included (followed by approximate maximum depth): (1) Floral Gardens (20 m); (2) Salisbury (6.5 m); (3) Tarou Point (7.5 m); (4) Coral Gardens (13.5 m); and (5) Cachacrou (20 m) (Fig. 1). All survey sites combined included over 5800 m^2 of reef area and are representative of other reef communities along the west coast of Dominica. The dominant coral species at these sites were *Porites astreoides* (Lamarck, 1816), *Siderastrea siderea* (Ellis and Solander, 1786), *Meandrina meandrites* (Linnaeus, 1758), and members of the *Montastraea* species complex, and the percent coral cover ranged from 11.9% to 23.3%, depending on the site (see Steiner, 2003). Tarou Point could not be surveyed in August 2001 because of a combination of road construction just above the site (loosened fine sediments) and consistent heavy rains that resulted in a reduction in water visibility to < 1 m throughout the 30-d field excursion.

General Survey Methodology.—At each site, a survey area was delineated and marked with nails, and an underwater site map was constructed. Survey boundaries were either established by the natural topography (e.g., sand flats devoid of coral colonies were not surveyed and thus constituted a survey site edge), or were based upon the haphazard selection of an area that was considered practical in terms of time management. A single individual collected all of the field data, thus excluding problems associated with inter-observer bias. A pivoting line search pattern was implemented to sample the survey area, which involved swimming back and forth across the site (using snorkeling or SCUBA, depending on the depth of the site). The underwater maps were utilized to ensure that each site had been thoroughly surveyed for the presence of BBD, WP, and DSS and to help relocate tagged colonies in subsequent surveys.

Each time a diseased coral colony was encountered, it was tagged with a roofing nail (with a 2 cm diameter, flat head) on areas of the colony devoid of live coral tissue, and the following information was recorded: type of disease, coral species affected, number of infections or lesions per colony, percent of colony affected (estimated as 1 = 1%–20%, 2 = 21%–40%, 3 = 41%–60%, 4 = 61%–80%, and 5 = 81%–100%), colony size, and disease-related tissue mortality (cm^2). Approximately 70% of the nails used to tag colonies were relocated in the subsequent survey years. Missing nails were likely lost to surge, damselfishes, and recreational divers. Due to time constraints, when there were more than 30 lesions on a single colony a measurement of >30 was recorded, and a value of 30 was used in all calculations. A Kruskal-

Wallis one-way ANOVA (and an a posteriori Dunn's Method) was used to assess differences in the number of infections per colony and the percent of each colony affected by WP, BBD, and DSS. Additional data were collected on the re-infection of susceptible colonies in subsequent years, multiple infections on individual colonies, and disease resolution (no apparent signs of infection).

Target species were identified for each disease/syndrome during each survey year and were defined as those species that were selectively affected by each disease. Thus, the species that compromised 75% or more of all respective infections were grouped together to represent the target species for each disease. The relative number of infected colonies per species is referred to as "species susceptibility" in the text, but it is important to note that there are likely other factors that characterize susceptibility. 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 used by the Coral Disease and Health Consortium (NOAA) (2003). Either a one-way ANOVA or a Kruskal-Wallis one-way ANOVA was utilized to compare disease incidence among sites and survey years.

Colony Size Measurements.—The size of a colony was measured as maximum diameter (cm) multiplied by maximum height (cm). The only species not measured for size was *Montastraea annularis* (Ellis and Solander, 1786), due to the difficulty in discerning distinct colony boundaries. Colony sizes were then divided into five size classes: 1) 1–100 cm^2 (small); 2) 101–1000 cm^2 (small–medium); 3) 1001–4000 cm^2 (medium), 4) 4001–5000 cm^2 (medium–large), and 5) > 5000 cm^2 (large). The colony size of an additional 16–140 colonies, selected haphazardly, of susceptible corals (i.e., included both healthy and diseased colonies) was measured at each site. Variation in the number of colonies sampled reflected their occurrence in the representative study areas. These measurements were used to portray the size distribution of relevant species in Dominica, within the survey sites, and are referred to as "population" corals in representative figures and text. A Chi-Square analysis was used to compare the sizes of diseased and population corals. Due to the limitations of using values of < 5 in a Chi-Square analysis, the only species with a large enough number of diseased individuals to be used in this application were: *S. siderea*, *Montastraea faveolata* (Ellis and Solander, 1786), *Dichocoenia stokesii* (Milne, Edwards and Haime, 1848), *Colpophyllia natans* (Houttuyn, 1772), *Agaricia agaricites* (Linnaeus, 1758), and *M. meandrites*.

Coral Mortality.—Disease-related tissue mortality was measured during the last survey month of each year, thus quantifying annual coral tissue mortality at each site. A flexible tape measure was used to measure the area (maximum length and width) of bare skeleton associated with each disease infection. These measurements were limited to recently exposed coral skeleton, characterized by no or minimal growth of filamentous algae on the calcium carbonate surface, in an attempt to unequivocally establish the relationship of tissue death to the coral disease infections. A one-way ANOVA was used to determine significant differences in tissue mortality among diseases and coral species.

Disease Progression Rates.—In 2002, measurements were made in March, June, and August of the progression of individual diseases on randomly selected, tagged colonies. The front of the advancing disease band, or the edge of the lesion in the case of DSS, was marked with 2 cm long, concrete nails. The linear progression of tissue mortality, if present, was measured (mm) 7 d later. Not all cases of DSS exhibited apparent tissue loss (e.g., visible bare white skeleton, indicating recent tissue death), so when tissue loss was not present, the nails were used to measure the radial expansion of the dark purple DSS lesions. No measurements were acquired of DSS lesions exhibiting tissue mortality in June. A Kruskal-Wallis one-way ANOVA was applied to assess differences in the progression rates of each disease and differences in progression rates among survey months (a t-test was used to compare DSS in March vs August).

Environmental and Populations Factors.—Various physical and population parameters were measured at each site and compared to disease incidence at each site in 2000, 2001, and 2002. The following parameters were utilized in the comparison: depth (diver's depth

gauge), water temperature (taken once during each survey month at 1 m below the surface by a mercury thermometer), target species density, scleractinian coral diversity (H'- Shannon Weiner diversity index), and percent coral cover. The last three parameter values were taken from data collected by S. Steiner (2003) during the same sampling period in Dominica. All comparisons between physical and population variables and disease incidence at each site were made using either a Pearson's Product Moment correlation analysis (r), or a least squares linear regression analysis (R^2).

Coral Recruitment.—In both 2001 and 2002, tagged colonies (n_{2001} = 120, n_{2002} = 114) were examined for scleractinian coral recruits on areas of the coral skeleton that had been exposed by coral disease(s). Other general categories of colonizers that were identified during the survey included boring sponges (primarily *Cliona* sp.), filamentous algae, macroalgae (primarily *Lobophora variegata* (Lamouroux) Womersley and *Dictyota* spp.), *Millepora* spp., sponges, and polychaete worms (*Spirobranchus giganteus* (Pallas, 1766) and *Pomatostegus stellatus* (Abildgaard, 1879)).

Results

Disease Dynamics.—In total, 325, 375, and 560 colonies were identified with BBD, WP, and/or DSS infections in 2000, 2001, and 2002, respectively. While there was a trend towards increasing numbers of diseased colonies per year, these differences were not significant (Fig. 2). The high variability of the data resulted in only one significant difference: DSS incidence was significantly higher in 2002 than in 2000 (Kruskal-Wallis one-way ANOVA: H = 13.05, df = 2, P < 0.01; Dunn's Method: Q = 3.6, P < 0.05 for 2000 vs 2002). In 2000 and 2001, the site with the highest disease incidence was Cachacrou, but this difference was not significant (Table 1). In 2002, Coral Gardens had the highest significant incidence of coral diseases (one-way ANOVA of ln transformed data: F = 7.48, df = 14, P < 0.01). The disease incidence at Coral Gardens was significantly higher than at Tarou Point (Tukey Test: Q = 7.25, P < 0.01), Salisbury (Q = 5.40, P < 0.05) and Floral Gardens (Q = 5.28, P < 0.05). Cachacrou had the highest incidence of colonies with WP in all three survey years, but this distinction was only significant in 2002 *(*Kruskal-Wallis one-way ANOVA: H = 12.30, df = 4, P < 0.05; Table 1). Colonies with BBD exhibited the highest inci-

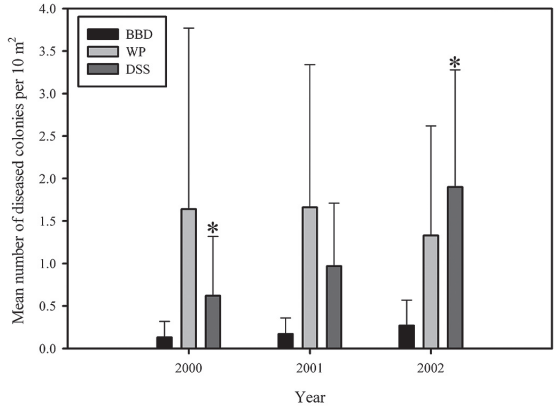

Figure 2. The mean number of diseased colonies per 10 m^2 at each site from March to August of each survey year (2000, 2001 and 2002). Error bars are ± SD. * indicates significant difference (Kruskal-Wallis one-way ANOVA, P < 0.01)

Table 1. Disease incidence at each site from March to August 2000, 2001, and 2002. Values are given as number of diseased colonies 10 m^{-2} of survey reef area. BBD = black band disease, WP = white plague, and DSS = dark spots syndrome.

Site	Disease	2000	2001	2002
Floral Gardens				
	BBD	0.12	0	0
	WP	4.48	4	3.51
	DSS	1.09	2.54	4.84
	Total	5.69	6.54	8.35
Salisbury				
	BBD	0.14	0.47	1.55*
	WP	0.19	1.5	1.55
	DSS	2.62	2.3	5.62
	Total	2.95	4.27	8.72
Tarou Point				
	BBD	0.8	1.06	1.53
	WP	1.99	1.53	0.93
	DSS	1.46	1.33	3.85
	Total	4.25	3.92	6.31
Coral Gardens				
	BBD	0.23	0.34	1.02
	WP	5.12	4.88	3.97
	DSS	3.4	6.13*	10.33
	Total	8.75	11.35	15.32
Cachacrou				
	BBD	0.19	0.56	0
	WP	12.85	11.36	10.06
	DSS	0.74	1.3	3.91
	Total	13.78	13.22	13.97

*Significant difference in disease incidence among sites (each year): Cachacrou (WP) and Salisbury (BBD): Kruskal-Wallis one-way ANOVA, $P < 0.05$; Coral Gardens (DSS): one-way ANOVA, $P < 0.05$; Coral Gardens (Total): one-way ANOVA, $P < 0.01$.

dences at Salisbury in 2002 (Kruskal-Wallis One Way ANOVA: $H = 11.05$, $df = 4$, $P < 0.05$). Coral Gardens had the highest incidence of colonies with DSS in all three survey years, but this distinction was only significant in 2001 (one-way ANOVA: $F = 4.46$, $df = 13$, $P < 0.05$).

WP, BBD, and DSS affected a total of 16, 6, and 1 species, respectively, during the 3-yr survey (Table 2). However, species susceptibility was inconsistent among survey years. For example, WP infected *Montastraea cavernosa* (Linnaeus, 1767) only in 2000, *Eusmilia fastigiata* (Pallas, 1766) and *Mussa angulosa* (Pallas, 1766) only in 2001, and *Diploria labyrinthiformis* (Linnaeus, 1767) only in 2002. BBD infected *D. stokesi* and *M. annularis* in 2000 but not in any of the following years. Similarly, *Stephanocoenia intersepta* (Milne, Edwards and Haime, 1848) and *M. meandrites* only exhibited BBD infections during the 2001 survey year. The only disease/syndrome with a consistent species susceptibility range was DSS, and *S. siderea* was the only species noted with DSS in all three survey years. The target species for DSS and BBD was *S. siderea*, and this remained consistent throughout the survey (Table 2). WP target species were similar in each year, but *C. natans* was considered a target species in 2000 and 2001 but not in 2002.

Table 2. Scleractinian coral species* identified with WP, BBD, and DSS at five reef sites in Dominica in 2000, 2001, and 2002. The relative contribution (%) of each target species to the total number of diseased colonies is provided. As per the definition of the target species, these relative values add up to at least 75%.

Disease/syndrome	2000	2001	2002
WP	Ssid (20.5%)	Ssid (32.5%)	Ssid (35.8%)
	Mfav (18.4%)	Mann (20.3%)	Mfav (18.8%)
	Mann (14.1%)	Mfav (12.7%)	Mann (12.7%)
	Mmean (12.9%)	Mmean (7.6%)	Mmean (8.5%)
	Cnat (9.7%)	Cnat (6.6%)	Cnat
	Sint	Sint	Sint
	Aagar	Aagar	Aagar
	Dstok	Dstok	Dstok
	Past	Past	Past
	Dstrig	Dstrig	Dstrig
	Mycet sp.	Mycet sp.	Mycet sp.
	Mcav	Efast	Dlaby
		Mang	Agar sp.
		Agar sp.	
BBD	Ssid (84.2%)	Ssid (86.5%)	Ssid (100%)
	Aagar	Aagar	
	Dstok	Sint	
	Mann	Mmean	
DSS	Ssid (100%)	Ssid (100%)	Ssid (100%)

*Coral species abbreviations: Aagar = *Agaricia agaricites*, *Agar* sp. = *Agaricia* sp., Cnat = *Colpophyllia natans*, Dlaby = *Diploria labyrinthiformis*, Dstok = *Dichocoenia stokesi*, Dstrig = *Diploria strigosa*, Efast = *Eusmilia fastigiata*, Mann = *Montastraea annularis*, Mang = *Mussa angulosa*, Mcav = *Montastraea cavernosa*, Mfav = *Montastraea faveolata*, Mmean = *Meandrina meandrites*, *Mycet* sp. = *Mycetophyllia* sp., Past = *Porites astreoides*, Sint = *Stephanocoenia intersepta*, and Ssid = *Siderastrea siderea*.

In total, 1183 colonies were considered in quantifying the number of lesions or infections per colony, and 1256 colonies were measured for the percent of each colony (surface area) affected by the disease. The results were consistent among years, and WP affected the largest percent of each colony (estimation value mean of 1.5 (1 = 1%–20% and 2 = 21%–40%), ± 0.9 SD, n = 502) (Kruskal-Wallis one-way ANOVA: $H = 58.82$, $df = 2$, $P < 0.001$; significantly greater than DSS, Dunn's Method: $Q = 5.34$, $P < 0.05$). Corals with DSS infections had the highest number of lesions per colony (mean = 11.5, ± 10.2, n = 619; Table 3) when compared to both WP and BBD (Kruskal-Wallis one-way ANOVA: $H = 652.47$, $df = 2$, $P < 0.001$; Dunn's Method: $Q = 22.95$, $P < 0.05$ for DSS vs WP and $Q = 14.64$, $P < 0.05$ for DSS vs BBD).

Infection patterns (re-infection, multiple infections, infection persistence, and cessation) of all diseases/syndromes were highly dynamic over time. In 2000, 31% of colonies tagged with WP in March and June were devoid of disease signs by August 2000. This increased to 38% in 2001 and 57% in 2002. In 2001, 21 colonies (~ 11% of total WP in 2001) with WP had been previously infected in 2000. In 2002, 35 colonies (~21% of total WP in 2002) with WP had been previously infected in either 2000 or 2001. During the 2001 survey, three colonies with WP also developed DSS, two developed BBD infections, and one acquired an additional WP infection on a previously unaffected area of the colony. In 2002, six colonies with WP also exhibited DSS, one exhibited BBD, and five developed new WP infections.

Table 3. Mean percent (± SD) of each colony surface area affected by each disease and the mean number of infections or lesions per colony for each disease in 2000, 2001, and 2002 and all years combined (total). Percent of each colony affected is based on the following estimation scale: 1 = 1%–20%; 2 = 21%–40%; 3 = 41%–60%; 4 = 61%–80%; and 5 = 81%–100%.

Disease/ syndrome	2000		2001		2002		Total	
	% affected	# lesions	% affected	# lesions	% affected	# lesions	% affected	# lesions
WP	1.6	1.6	1.5	2.0	1.5	1.6	1.5	1.7
	(± 0.9)	(± 1.4)	(± 0.9)	(± 2.4)	(± 1.0)	(± 1.3)	(± 0.9)	(± 1.8)
	n = 175	n = 166	n = 173	n = 199	n = 154	n = 188	n = 502*	n = 553
BBD	1.3	1.6	1.2	1.4	1.2	1.3	1.2	1.4
	(± 0.4)	(± 1.5)	(± 0.4)	(± 0.8)	(± 0.5)	(± 0.7)	(± 0.5)	(± 0.9)
	n = 16	n = 18	n = 31	n = 34	n = 44	n = 63	n = 91	n = 115
DSS	1.1	10.8	1.3	14.2	1.1	10.2	1.2	11.5
	(± 0.3)	(± 10.2)	(± 0.6)	(± 10.8)	(± 0.4)	(± 9.7)	(± 0.5)	(± 10.2)
	n = 112	n = 100	n = 153	n = 180	n = 325	n = 339	n = 590	n = 619*

*Significant results: Kruskal-Wallis one-way ANOVA, $P < 0.001$.

In 2000 and 2001, all BBD infections persisted from March to August. In 2002, 15% of the colonies with BBD had infections that arrested between March and August. In most cases, BBD infections persisted throughout the yearly survey period. In 2001, seven colonies (~22% of total BBD in 2001) with BBD had been previously infected in 2000, and in 2002 nine colonies (~14% of total BBD in 2002) had been previously infected in either 2000 or 2001. Only two of the colonies monitored during the 3-yr survey developed additional infections, all of which were new BBD.

In 2000, three colonies with DDS tagged in March and June were devoid of visible DSS lesions by August. In 2001, 19% of colonies with DSS in March no longer had visible DSS lesions by August, and this value was 9% in 2002. In 2001, 47 (~31% of total DSS in 2001) colonies with DSS had been previously affected in 2000. In 2002, 120 (~36% of total DSS in 2002) colonies with DSS had been affected in 2000 and/or 2001. A total of 107 colonies with DSS acquired new infections during the yearly surveys (56 new WP infections and 51 new BBD infections).

Overall, 23% of diseased colonies tagged in 2000 experienced re-infection or a continued infection in 2001, and 25% of all diseased colonies in 2000 and 2001 combined also exhibited diseases in 2002.

Colony Size.—Comparisons of colony size distributions of population and diseased corals revealed that diseases predominantly affected the larger size classes of most susceptible species. DSS, BBD, and WP all affected the larger size classes of *S. siderea* ($\chi^2_{DSS} = 148.39$, $P < 0.0001$, $df = 3$; $\chi^2_{BBD} = 425.98$, $P < 0.0001$, $df = 2$; $\chi^2_{WP} = 89.33$, $P < 0.0001$, $df = 3$; Fig. 3A). WP also affected the larger colonies of *M. faveolata* ($\chi^2 = 13.14$, $P < 0.01$, $df = 2$; Fig. 3B), *D. stokesii* ($\chi^2 = 11.44$, $P < 0.01$, $df = 2$; Fig. 3C), and *C. natans* ($\chi^2 = 156.36$, $P < 0.0001$, $df = 2$; Fig. 3D). WP did not preferentially affect any size class of *A. agaricites* ($\chi^2 = 0.98$, $P > 0.05$, $df = 1$) or *M. meandrites* ($\chi^2 = 3.69$, $P > 0.05$, $df = 2$). In these populations, the frequency distribution of diseased colony sizes mirrored the population colony size distribution (graphs not shown).

Coral Mortality.—During the 3-yr survey, a total of 8.74 m^2 of coral tissue surface area was killed by coral diseases (Table 4). Though most of this tissue destruction involved the partial mortality of affected colonies, there were ten individuals that experienced total colony death (three *M. meandrites*, three *C. natans*, one *Mycetophyllia* sp., one *M. faveolata*, one *M. annularis*, and one *P. astreoides*). The highest

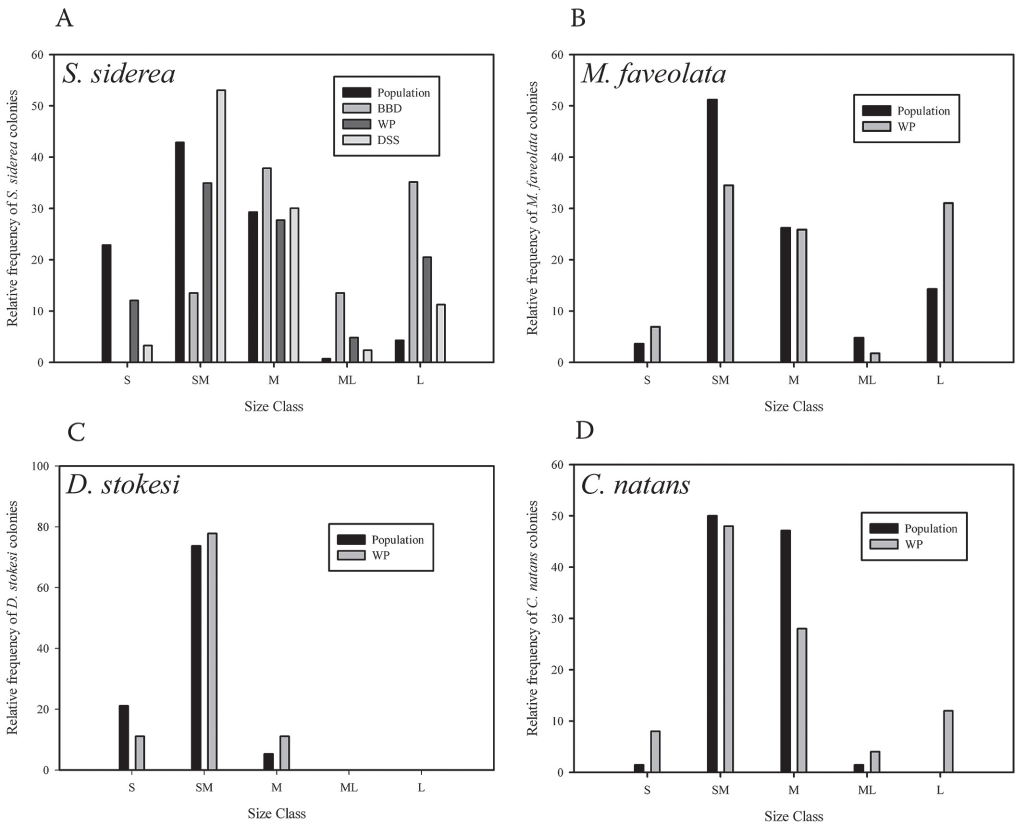

Figure 3. The relative frequency of the size class distributions of population and diseased coral colonies. (a) *Siderastrea siderea*: population, BBD, WP and DSS, (b) *Monastraea faveolata*: population and WP, (c) *Dichocoenia stokesi*: population and WP, (d) *Colpophyllia natans*: population and WP. Size classes: S (small) = 1–100 cm^2; SM (small-medium) = 101–1000 cm^2; M (medium) = 1001–4000 cm^2; ML (medium-large) = 4001–5000 cm^2; and L (large) = > 5000 cm^2.

Table 4. Total disease-related tissue mortality (cm^2) incurred by each species during each year and in all years combined (Total). Species are listed in order of magnitude of tissue mortality recorded each year (see Table 2 for species abbreviations). Values were significantly different among species when comparing all survey years. Significant differences are denoted by * and † (see footnote for further description).

2000		2001		2002		Total	
Species	Tissue mortality (cm^2)	Species	Tissue mortality (cm^2)	Species	Tissue mortality (cm^2)	Species	Tissue mortality (cm^2)
Mfav	13,646	Mfav	7,203	Mfav	7,194	Mfav*	28,043
Cnat	5,753	Ssid	6,751	Ssid	5,445	Ssid†	17,709
Ssid	5,513	Mann	5,810	Mmean	1,469	Mann*	11,717
Mann	4,925	Mmean	3,258	Dstrig	1,244	Mmean*	8,033
Mmean	3,306	Sint	2,671	Mann	982	Cnat*	7,472
Dstrig	2,556	Cnat	1,490	Sint	254	Dstrig*	4,188
Mcav	2,423	Mang	818	Cnat	229	Sint*	4,027
Sint	1,102	Dstrig	388	Dstok	177	Mcav	2,423
Dstok	770	Past	336	Agar sp.	104	Dstok*†	1,030
Aagar	520	Mycet sp.	264	Aagar	69	Mang	818
Mycet sp.	289	Agar sp.	152	Dlaby	49	Aagar*†	661
		Dstok	83	Past	43	Mycet sp.*†	583
		Aagar	72	Mycet sp.	30	Past	379
						Agar sp.	256
						Dlaby	49
Total	4.08 m^2		2.93 m^2		1.73 m^2		8.74 m^2

* Species mortality values that were significantly different from those of *M. faveolata* (Mfav) (Tukey Test: (1) Mfav vs Mycet sp., Q = 8.68, P < 0.001, (2) Mfav vs Aagar, Q = 8.65, P < 0.001, (3) Mfav vs Dstok, Q = 8.53, P < 0.001, (4) Mfav vs Sint, Q = 7.59, P < 0.01, (5) Mfav vs Dstrig, Q = 7.54, P < 0.01, (6) Mfav vs Cnat, Q = 6.50, P < 0.01, (7) Mfav vs Mmean, Q = 6.32, P < 0.01, and (8) Mfav vs Mann, Q = 5.16, P < 0.05).
† Species mortality values that were significantly different from those of *S. siderea* (Ssid): (Tukey Test: (1) Ssid vs Mycet sp., Q = 5.41, P < 0.05, (2) Ssid vs Aagar, Q = 5.39, P < 0.05, (3) Ssid vs Dstok, Q = 5.27, P < 0.05).

coral tissue mortality was recorded in August 2000 and there was a trend towards decreasing mortality over time. The species with the most tissue mortality in each year and all years combined was *M. faveolata* (one-way ANOVA: F = 7.73, df = 29, P < 0.001; see Table 4 for a posteriori Tukey Test results). WP infections caused the largest amount (approximately 84%) of recorded tissue mortality (one-way ANOVA: F = 31.35, df = 8, P < 0.001; Table 5). This difference was significantly greater than both BBD (Tukey Test: Q = 8.79, P < 0.05) and DSS (Q = 10.40, P < 0.05), thereby making it the most destructive disease in terms of tissue loss.

Disease Progression Rates.—The progression, or rate of advancement, among diseases was significantly different (Kruskal-Wallis one-way ANOVA: H = 21.15, df = 2, P < 0.001; Table 6). WP infections progressed most rapidly [mean = 1.73 (± 2.4 SD) mm d^{-1}, n = 29], which was significantly different when compared to DSS (Dunn's Method: Q = 4.05, P < 0.05) but not to BBD. Tissue death (e.g., presence of bare white skeleton, indicating the recent death of coral tissue) was not always present on colonies exhibiting DSS (~15%), but when present, rates of tissue mortality were the lowest measured of the three diseases/syndromes [mean = 0.13 (± 0.20 SD) mm d^{-1}, n = 23]. BBD also progressed at consistently slow rates [mean = 0.81 (± 0.64 SD) mm d^{-1}, n = 20], but these rates were significantly higher than those of DSS (Dunn's Method: Q = 3.99, P < 0.05). The rate of disease progression changed among survey months, but these differences were only significant for DSS (exhibiting bare skeleton)

Table 5. Total tissue mortality (cm^2) associated with each disease/syndrome in each year and in all years combined.

	Tissue mortality (cm^2)			
Disease/syndrome	2000	2001	2002	Total
WP	36,179	24,311	13,270	73,760*
BBD	2,659	3,043	2,970	7,978
DSS	1,965	1,942	1,049	5,650

* significant results: One-Way ANOVA, P<0.001.

(t-test of arcsin square root transformed data: t = 3.602, df = 21, P < 0.01). WP advanced across a colony surface over three times faster in August than in March and was significantly related to an increase in water temperature (least squares linear regression analysis: $R^2 = 1.00$, P < 0.01, df = 2). BBD progression rates doubled from March to June and then decreased in August, and DSS rates increased six-fold from March to August.

Environmental and Populations Factors.—There were no significant relationships between disease incidence at each site and temperature or percent coral cover. There was a significant, negative relationship between BBD and coral species diversity in 2001 (r = −0.988, P < 0.05, df = 3) and a significant, negative relationship with depth in 2002 (r = −0.985, P < 0.01, df = 4). DSS was significantly correlated to the relative frequency of target species at each site in 2001 (r = 0.985, P < 0.05, df = 3) and WP was significantly correlated to the relative frequency of its target species in 2002 (r = 0.880, P < 0.05, df = 4).

Coral Recruitment.—In 2001, ten scleractinian recruits were identified on tagged coral colonies on areas of the coral that had suffered from disease-induced tissue mortality. The recruits included: five *P. astreoides*, four *A. agaricites*, and one *M. meandrites*. Other colonizers identified on the respective skeletal areas were: filamentous algae (n = 100), boring sponges (n = 45), macroalgae (n = 30), and *Millepora* sp. (n = 1). In 2002, nine scleractinian recruits (six *P. astreoides* and three *A. agaricites*) were identified on the 114 colonies examined. Additional colonizers included: filamentous algae (n = 108), macroalgae (n = 59), boring sponges (n = 58), sponges (n = 6), *Millepora* sp. (n = 1), and polychaetes (n = 1). Thus, the most commonly identified colonizers of recently exposed coral skeleton were filamentous algae (89% of colonies examined), boring sponges (44% of colonies examined), and macroalgae (38% of colonies examined).

Table 6. Disease progression rates (mm d^{-1}) for each disease, measured in March, June, and August 2002 (n = colonies). All colony measurements per disease were combined to include a yearly mean value for each disease. DSS (bare skeleton) refers to cases of DSS in which tissue mortality was evident as exposed, bare coral skeleton. DSS (lesion) refers to the radial growth of lesions in which there was no evidence of bare coral skeleton. Rates are listed as mean (± SD).

Disease/syndrome	Progression rates (mm d^{-1})			
	March	June	August	Yearly mean
WP	0.83 (± 1.13)	1.96 (± 2.27)	3.10 (± 3.7)	1.73 (± 2.40)*
	n = 13	n = 9	n = 7	n = 29
BBD	0.68 (± 0.42)	1.16 (± 1.15)	0.70 (± 0.30)	0.81 (± 0.64) *
	n = 8	n = 5	n = 7	n = 20
DSS (bare skeleton)	0.06 (± 0.07) †		0.40 (± 0.29) †	0.13 (± 0.20)
	n = 18	-----	n = 5	n = 23
DSS (lesion)	0.16 (± 0.18)	0.15 (± 0.18)	0.12 (± 0.18)	0.14 (± 0.17)*
	n = 9	n = 12	n = 6	n = 27

* significant results: Kruskal-Wallis one-way ANOVA, P < 0.001
† significant results: t-test, P < 0.01

Discussion

There is a growing consensus that coral diseases are increasing in incidence, prevalence, and geographic and host range (e.g., Garzón-Ferreira et al., 2001; Porter et al., 2001; Borger, in press). The only published account of a multiple year survey in which disease prevalence did not increase between years was one of BBD in the northern Florida Keys (Kuta and Richardson, 1996). Although the variability of the data was high in the present study, there was a trend towards increasing disease incidence throughout the survey period in Dominica. However, this was significant only for DSS between 2000 and 2002.

The three coral diseases measured in this study affected a total of 16 scleractinian species. However, species susceptibility was inconsistent among years, which was also noted in a similar study in south Florida (Borger, in press). This highlights the critical need for surveys involving more than 1 yr of sampling in order to acquire an accurate representation of a highly dynamic phenomenon and to devise appropriate management policies.

The three coral diseases measured in this study affected different species than those described as most susceptible in other Caribbean regions. DSS only affected *S. siderea* in Dominica. This differs from other accounts of DSS as affecting multiple scleractinian species (Weil et al., 2000; Garzón-Ferreira et al., 2001; Gil-Agudelo and Garzón-Ferreira, 2001; Santavy et al., 2001; Borger, in press), also commonly found in Dominica. BBD affected six species of scleractinian corals in Dominica, though the majority (>90%) of colonies with this disease were *S. siderea*, which is similar to a study of BBD in Jamaica (Bruckner et al., 1997). In contrast, *Diploria strigosa* (Dana, 1848), *D. labyrithiformis*, *C. natans*, *Diploria clivosa* (Ellis and Solander, 1786), and *M. cavernosa* are thought to be highly susceptible to BBD in other regions (Edmunds, 1991; Kuta and Richardson, 1996; Bruckner and Bruckner, 1997a; Borger, in press) and were not identified with BBD at any of the five sites in Dominica over 3 yrs. WP affected more coral species than both BBD and DSS. Weil et al. (2000) also reported WP as having the largest coral species host range at 19 reef sites in the wider Caribbean region. The species with the most WP infections in Dominica between 2000 and 2002 were *S. siderea*, *M. faveolata*, and *M. annularis*, which differs from other reports on species susceptibility to WP (Bruckner and Bruckner, 1997b, Richardson et al., 1998a,b; Borger, in press). It is possible that the eastern Caribbean region differs in species susceptibility characteristics for DSS. These differences may reflect variations in exposure to stressors such as temperature, diving pressure, sedimentation, and pollution, or a pathogen that is highly locally adapted to its host(s). It is also possible, as suggested by Weil et al. (2000), that coral species in different Caribbean locations have developed different resistance capacities.

The progression rates of BBD, WP, and DSS were considerably lower than those described by Rützler et al. (1983), Richardson et al. (1998b), and Cervino et al. (2001), respectively. This may be due to the relatively low water temperatures off Dominica. In 3 yrs, measured water temperatures did not exceed 30 °C. The optimal temperature for the proliferation of the dominant BBD pathogen (*Phormidium corallyticum* Rützler and Santavy, 1983) is between 30 and 37 °C (Richardson and Kuta, 2003) and is greater than 30 °C for the WP pathogen (Remily and Richardson, Florida International University, unpubl. data). Therefore, the seawater temperatures in Dominica may not be warm enough to enable the maximum progression rates and virulence of

the BBD and/or WP pathogens. Though relative rates were low, all disease progression rates initially increased with warmer water temperatures (though BBD rates decreased from June to August), which may indicate that temperatures between 26 and 30 °C may be a critical threshold in the virulence of coral disease pathogens. Therefore, diseases may have varying degrees of impact on coral tissue death in different temperature regimes of the Caribbean and worldwide and may have an increased negative impact on corals during global warming.

During the 3 yrs in Dominica, over 8 m^2 of coral tissue was killed by coral diseases, which was estimated to be < 2% of the total scleractinian coral tissue area at all sites combined. Over 80% of this tissue death was caused by WP infections, indicating that this disease has the most significant, negative impact on the reefs in Dominica. However, the total tissue mortality consistently decreased in each successive survey year. Therefore, even though there was a trend towards increasing disease incidence with time, their cumulative impact in terms of causing coral tissue mortality decreased. Hence, from a management perspective, data on disease induced tissue mortality may be more important in assessing disease impacts than prevalence or incidence values.

From an ecological perspective, the three species that suffered the greatest amount of tissue loss were *M. faveolata*, *S. siderea*, and *M. annularis*. Coral diseases in Dominica also selectively affected the larger colonies of *S. siderea*, *M. faveolata*, and *C. natans*. These massive coral species are important to reef framework formation and stability in the Caribbean (Goreau, 1959; Glynn, 1973; Endean and Cameron, 1990; Ginsburg et al., 1996) and are the dominant reef-builders on Dominican reefs. Additionally, the effects of colony size of corals are thought to be more influential on population dynamics than age effects (Hughes and Connell, 1987; Babcock, 1991) because larger colonies have been demonstrated to have greater fecundity than smaller ones, the capacity for reproduction is size-dependent in some corals regardless of age, and smaller colonies are subjected to higher rates of mortality (Connell, 1973; Hughes, 1984; Szmant-Froelich, 1985; Szmant, 1986). Therefore, the death and selective infection of larger colonies of these species may have serious implications for the longevity and stability of the reefs in Dominica, which are exposed to frequent storm and hurricane impacts.

Approximately 25% of all affected colonies were either re-infected, or exhibited continued infections in successive years. It is possible that some of these colonies had infections that persisted through the cooler, winter season into successive survey years (which cannot be determined because no surveys were conducted between October and February of any year), but this value is still likely an underestimation due to the inability to relocate all tagging nails. Bruckner and Bruckner (1997a,b), Bruckner et al. (1997), and Kuta and Richardson (1997) also noted the re-infection of diseased coral colonies in successive survey years. Therefore, the cumulative effects of coral diseases on affected colonies can be far greater than a short (< 1 yr) survey can detect. Though most of the tissue death caused by disease leads to the partial mortality of the colony, multiple infections can eventually lead to a greater loss of entire colonies. In this case, the threat of localized extinction of highly susceptible species is far greater than has been previously acknowledged. Bruckner (2002) hypothesized that corals that survive disease outbreaks are likely to become resistant to future outbreaks, thereby increasing their chance of survival. This may be valid

for certain reefal areas but does not seem to be the case in Dominica, where many colonies are experiencing multiple, annual infections.

The lack of a significant, linear relationship between temperature and disease incidence could reflect the relatively low temperature fluctuations of Dominica's coastal waters. Though coral diseases were found in higher densities during the warmer months of the year, Santavy et al. (2001) also failed to establish a clear pattern of seasonality in coral diseases. Borger (in press) discovered a significant relationship between BBD and temperature in south Florida, but this relationship did not exist for either WP or DSS. According to Bruckner (1999), the direct relationship between disease prevalence and water temperature might be confounded by other important variables in disease perpetuation, such as water clarity, sediment load, and wave energy (both associated with distinct times of the year and/or seasons) in specific regions. While the lack of a linear relationship with temperature may be a function of not sampling during the winter, it also suggests that other physical parameters may be important to disease virulence (e.g., rainfall/hyposalinity —Dominica's rainy season commences during the summer months). The significant negative relationship between BBD and coral diversity and water depth agree with the findings of Rützler et al. (1983), Taylor (1983), Bruckner and Bruckner (1997), Bruckner et al. (1997), and Bruckner (1999).

Both DSS and WP were significantly correlated with the relative frequency of target species densities at each site, a relationship that has been previously noted (Bruckner et al., 1997; Richardson et al., 1998a,b; Reigl, 2002; Borger, 2003, in press). These data suggest that the transmission of diseases among conspecifics may be facilitated when the relative species tissue cover is high. It may also be useful to management applications in that disease prevalence could potentially be predicted by the composition of coral species at each reef site.

The recruitment survey was conducted to further test the hypothesis proposed by Edmunds (1991) that coral diseases have a beneficial role in opening up bare substrate for the recruitment and colonization of new coral species. However, the recovery of reefs in response to the loss of coral tissue is low (Dustan, 1977; Hayes and Goreau, 1998), and tissue regeneration was not observed in this study, in south Florida (Borger, in press), or in a 4-yr study of the impacts of BBD in Jamaica (Bruckner and Bruckner, 1997a). The rate of coral recruitment in Dominica was similar to that found in south Florida (Kuta and Richardson, 1997; Edmunds, 2001), and this was determined to be insufficient and limiting to the propagation of scleractinian corals (Borger, in press). In addition, all of the coral recruits observed in Dominica were non-massive species, thus their contribution to reef-building may be limited. This was further exacerbated by the relatively high occurrence of bioeroders and algae observed on the exposed skeletal surfaces. These observations are consistent with the hypothesis that coral diseases can drive a shift in community structure of reefs dominated by corals to those dominated by algae (Porter and Meier, 1992; Porter et al., 2001).

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