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Three scleractinian coral diseases in Dominica, West Indies: distribution, infection patterns and contribution to coral tissue mortality

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Abstract: Coral diseases have been documented in many areas of the Caribbean, but studies in the eastern Caribbean region have been lacking. The prevalence, distribution patterns and contribution to the mortality of coral tissue by black band disease (BBD), white plague (WP) and dark spots disease (DSD) were examined at five reef sites along the west coast of Dominica. 185 of the 325 diseased colonies recorded between March and August 2000, in a survey area of 5884 m^2, were WP. This disease contributed to 89% of the total 4.08 m^2 of tissue mortality caused by diseases during the survey period. WP also affected the largest average tissue surface area (relative to colony size) per colony and exhibited the largest average tissue loss per infection when compared to BBD and DSD. The species most susceptible to WP and BBD in Dominica differed from most other described Caribbean locations with *Siderastrea siderea* being most susceptible. *S. siderea* was also the only species noted to be susceptible to DSD. Measurements of colony size revealed that each disease affected the larger colonies of some coral species. Comparisons between disease prevalence at each site and various physical parameters, including temperature, wave height, depth, and current patterns, did not exhibit significant correlations. The lack of a direct correlation between temperature and disease prevalence indicates that there are other seasonal factors contributing to the higher prevalence of diseases recorded during the summer months in Dominica. WP prevalence at each site was positively correlated to the relative species abundances of the species most susceptible to WP. This was the dominant factor in determining site-specific disease densities of this disease and may therefore be a valuable predictive and management tool. There were no correlations between BBD or DSD and the relative abundances of susceptible species. The spatial distribution patterns of WP, BBD and DSD were clustered, which is a distribution pattern that suggests an infectious disease.

Key words: Dominica, eastern Caribbean, coral diseases, black band disease, white plague, dark spots disease

Since the first report of a coral disease (Antonius 1973), attempts to identify causative factors and pathogens, and to quantify the loss of living coral, have been ongoing. It is likely that a synergism of various environmental and anthropogenic factors is contributing to the observed increases in coral diseases recorded worldwide (Harvell *et al.* 1999). The short-term effects of coral diseases can be quantified in terms of colony and tissue loss, but the long-term effects have only rarely been determined (Aronson and Precht 1997). Coral diseases have been reported in many Caribbean locations (Antonius 1973, Garret and Ducklow 1975, Rützler *et al.* 1983, Peters 1993, Edmunds 1991, Bruckner and Bruckner 1997a,b, Steiner and Borger 2000), the Indo-Pacific (Antonius 1985a, Littler and Littler 1996, Miller 1996) and the Red Sea (Antonius 1988). Though the majority of the reports of disease outbreaks focus on central and northern areas of the Caribbean, the expansion of

research into other areas is necessary in order to gain a regional picture. This study examined coral diseases in Dominica, which is located along the eastern edge of the Caribbean basin. Previous to this report, there has been only one published document available concerning coral diseases in the eastern Caribbean (Steiner and Borger 2000).

There have been few studies of the coral reef communities of Dominica (see Steiner and Borger 2000), a high relief, volcanic island located in the Lesser Antilles. The coral communities are virtually unprotected by law, aside from designated mooring buoys used by tour operators in the marine park of the southern Soufriere Bay. The study reported here involved a detailed examination of the prevalence, distribution, and infection patterns of BBD, WP and DSD at various locations along the west coast of Dominica. Coral degradation caused by diseases via tissue and colony loss was quantified.

Of these three diseases, black band disease (BBD), the first described coral disease (Antonius 1973), is currently the most understood. The disease is caused by a consortium of microorganisms that form a thin (~1 mm), dark band on the surface of a coral colony (Rützler and Santavy 1983, Carlton and Richardson 1995, Richardson 1996). The band then progresses across the colony at an average rate of 3.1 mm day^{-1} (Rützler *et al.* 1983), leaving behind denuded skeleton that is subsequently colonized by filamentous algae. There have been correlations between BBD incidence and increased water temperature (Rützler *et al.* 1983, Antonius 1985b) and pollution (Antonius 1988). The disease primarily infects massive, reef-building coral species (such as *Montastraea* spp. and *Diploria* spp.) and may thereby be contributing to the decline of various reef communities (Porter and Meier 1992, Kuta and Richardson 1997, pers. obs.).

White plague (WP) has been documented as having two etiologies, Type I (Dustan 1977) and Type II (Richardson *et al.* 1998). These etiologies differ in species susceptibility and the rate of tissue loss, with Type II having a wider species infection range and higher rates of tissue loss of up to 2 cm day^{-1}. WP manifests itself as a rapidly moving interface of bare white skeleton directly adjacent to either bleached tissue or apparently healthy tissue of normal coloration. The potential severity of this disease in terms of tissue and/or colony loss has been documented for *Dichocoenia stokesi* (Milne, Edwards and Haime, 1848) in the Florida Keys reef tract (Richardson *et al.* 1998) and *Diploria labyrinthiformis* (Linnaeus, 1767) in Puerto Rico (Bruckner and Bruckner 1997a), following WP outbreaks at each location.

Dark spots disease (DSD) is a disease/syndrome that has not been characterized in detail and infects colonies of *Siderastrea siderea* (Ellis and Solander, 1786) and *Siderastrea radians* (Pallas 1766) (Garzon-Ferreira and Gill 1998, Goreau *et al.* 1998, Cervino *et al.* 2001, Peters 2001). Aside from a visual description of the infection, little is reported about its distribution patterns (see Cervino *et al.* 2001), contribution to coral tissue mortality, potential pathogens and/or infectivity. Cervino *et al.* (2001) recorded a tissue necrosis rate of 4 cm $month^{-1}$ for *S. siderea* colonies infected with DSD. In Dominica, the disease is manifested as dark purple to black spots and/or blotches dispersed in various densities across colonies of *S. siderea*. Only blotches that are located along the perimeter of a tissue-algal interface display apparent tissue loss (i.e. bare, white skeleton). Other blotches scattered across a colony's surface may exhibit small clumps of filamentous algae growing in the center of the blotch, but in general tissue loss and/or necrosis is slow or not apparent. There are no obvious microorganisms or microbial mats, as is the case with BBD, colonizing the surface of the marks.

MATERIALS AND METHODS

Five reef sites along the west coast of Dominica were examined, covering 5 884 m^2 of study site area. Sites were accessed using both snorkel and SCUBA and included (followed by approximate maximum depth): 1- Coral Gardens (20 m), 2- Salisbury (6.5 m), 3- Tarou Point (7.5 m), 4- Floral Gardens (13.5 m) and 5- Cachacrou/Scott's Head East (20 m) (Fig. 1). Site selection was based upon location in protected zones (sites 1 and 5), proximity to po-

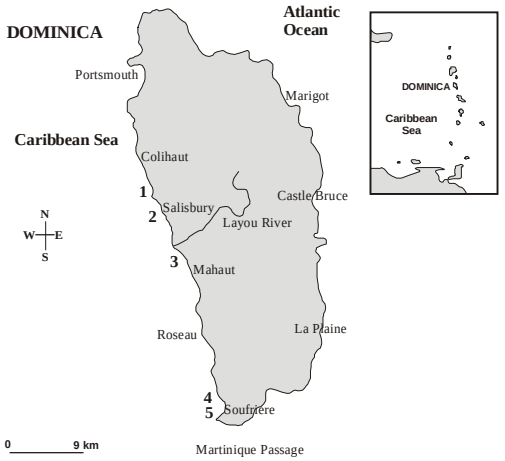

Fig. 1. Map of Dominica, West Indies with numbers indicating location of study sites along the west coast of the island: 1. Floral Gardens, 2. Salisbury, 3. Tarou Point, 4. Coral Gardens and 5. Cachacrou.

tential sources of pollution (e.g. large rivers or factories) (site 3), depth ("shallow" – snorkel sites (2 & 3) vs. "deep" – SCUBA sites (1, 4 & 5), and exposure to high-energy impact zones (site 2). The coral communities of Dominica are subjected to high levels of rainfall, sedimentation, run-off and river input (especially during storm events), and consist of coral colonies growing on volcanic rock, with little build-up of a carbonate base framework (Steiner and Borger 2000). Study sites, representative of other reef communities along the west coast, are dominated by the species *Porites astreoides* (Lamarck, 1816), *Siderastrea siderea* and *Meandrina meandrites* (Linnaeus, 1758).

Disease prevalence: The five sites were surveyed on multiple occasions during March, June and August 2000. The survey period encompassed 24 weeks of the year (March 15 – August 30, 2000) and included the summer season. This coincides with the reported seasonality of coral diseases since higher prevalences of BBD and WP have been reported in the warmer months of the year (Rützler *et al.* 1983, Antonius 1985b, Richardson *et al.* 1998). A grid-based search method was used to ensure complete coverage of delineated site areas. Corals with WP, BBD and/or DSD were tagged with roofing nails, plotted on a map, and monitored over time. The species and size of each diseased colony were recorded. Colony size measurements were taken as the maximum diameter multiplied by the maximum height of live tissue cover (cm^2). In addition, 25-30 colonies (chosen randomly, i.e. included both healthy and diseased colonies) of each susceptible species at each site were measured and are referred to as "community" corals in representative figures and text. Colony sizes were divided into 5 size classes: 1) 0-100 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) >5001 cm^2 (large). Comparisons between colony size classes of diseased and community corals were made using a Chi-Square contingency table analysis. Cases in which tagged colonies became infected with additional diseases, or in which the progression of a disease ceased, were also recorded.

WP in Dominica appeared to have similar (high) rates of progression typical of Type II but affected the species noted to be susceptible to both Type I and Type II (Dustan 1977 and Richardson *et al.* 1998). Therefore, within the framework of this study, it will be referred to as "white plague" (WP), without further distinction into etiologies.

Disease intensity and tissue & colony mortality: When diseased coral colonies were tagged, a measure of disease intensity was recorded. This measurement was defined by two parameters. The first was a visual approximation of the percent of the total colony that was affected by the disease, using a scale of: 1 = 0–20%, 2 = 21-40%, 3 = 41-60%, 4 = 61-80% and 5 = 81-100%. These approximations were made relative to the individual colony sizes. The second parameter was the number of active infections per colony. In the case of BBD and WP, infections considered active had stark white, exposed coral skeleton not yet colonized by filamentous algae, which was an indication that the disease band had very recently destroyed coral tissue. In the case of DSD, infections were counted as each spot or blotch present on a colony's surface.

The measurements of tissue mortality were made in late August in order to acquire values

representative of total tissue death after a complete disease season. The death of whole colonies was recorded throughout the survey period. Tissue mortality was recorded for each species and each disease. Measurements were made with a flexible measuring tape and consisted of the maximum width times the maximum length of lost tissue surface area (cm^2). All measurements were conservative in that only tissue death that was definitely caused by recent disease infections was measured.

Average tissue loss per infection was also estimated by using the total value of tissue mortality per disease (cm^2) divided by the total number of disease infections.

Physical and community measurements: Measurements of several environmental parameters relevant to disease prevalence (compared using Pearson Product Moment Correlation statistic when valid) included: water temperature (using a mercuric thermometer at 1 m below surface), depth (diver's depth gauge), wave height (using the scale of Van Duyl 1985), sustained low visibility (defined as being <3m for a time period of greater than 24 hours), predominant current direction, coral species diversity (Shannon-Weiner Index, H'), percent coral cover, and community composition (relative species cover, or densities). The last three parameters were calculated from data collected and compiled by S. Steiner (in prep.). Target species were defined and identified as those most susceptible to each disease. Relative abundances of such species at each site were then compared to disease densities using the Pearson Product Moment Correlation statistic.

During the identification of diseased coral colonies, associations with potential disease transmission vectors were also recorded. The presence of damselfish *Stegastes dorsopunicans* (Poey 1863), *Stegastes variabilis* (Castelnau 1855), *Stegastes partitus* (Poey 1868) and *Microspathodon chrysurus* (Cuvier 1830), echinoids *Diadema antillarum* (Philippi 1845) and/or fireworms *Hermodice carunculata* (Pallas 1766) on or within 50 cm of a diseased coral colony was noted. Due to the fact that these were snapshot measurements over time, numbers of associations are likely a conservative estimate.

Spatial distribution analysis: A spatial distribution analysis (using nearest neighbor distances) was conducted to determine whether the distribution of each disease was random or clumped. The distance between each diseased colony and every other diseased colony within 10 m was measured, and these measurements were then compared to a predicted spatial pattern of individuals with a Poisson distribution, as described by Schmale (1991). A Chi-Square goodness of fit test was implemented to determine significant differences. Due to the limitations associated with using small values in a Chi-Square analysis, only sites with high disease prevalence were utilized in the spatial distribution analyses.

RESULTS

Across all sites, approximately 70% of the mapped tagging nails were relocated. Missing nails were likely lost naturally via surge, wave action, and damselfish, or were removed by recreational divers.

Disease prevalence: A total of 325 diseased coral colonies were recorded between March and August 2000 within the study area of 5884 m^2 (Table 1). *Siderastrea siderea* exhibited the highest prevalence of each of the three diseases (Table 1). There were a total of 38 *S. siderea* colonies infected with WP (20.6% of all WP infections), 16 infected with BBD (84.1% of all infections) and 121 infected with DSD (100% of all infections). *Montastraea faveolata* (Ellis and Solander, 1786), *M. annularis* (Ellis and Solander, 1786), *Meandrina meandrites* and *Colpophyllia natans* (Houttuyn, 1772) were also notably susceptible to WP with 34, 26, 24 and 18 total colonies infected, respectively. WP had the highest overall prevalence (n = 185), followed by DSD (n=121) and BBD (n=19). Cachacrou, the southern-most site, had the highest total disease density per area (0.14 diseases m^{-2}) and also the highest number of WP infected colonies (n=69) (Table 2). Tarou Point had the highest BBD prevalence (n=12), and Salisbury had the highest number of colonies with DSD (n=56).

TABLE 1

Number of coral colonies infected with white plague (WP), black band disease (BBD)and dark spots disease (DSD) per species recorded at the five study sites between March and August 2000. Numbers in parentheses represent the percent of the total number for each individual disease

Species	WP	BBD	DSD
Siderastrea siderea	38 (20.6%)	16 (84.1%)	121 (100%)
Montastraea faveolata	34 (18.4%)	--	--
Montastraea annularis	26 (14.1%)	1 (5.3%)	--
Meandrina meandrites	24 (12.9%)	--	--
Colpophyllia natans	18 (9.7%)	--	--
Stephanocoenia intersepta	11 (5.9%)	--	--
Agaricia agaricites	11 (5.9%)	1 (5.3%)	--
Dichocoenia stokesi	10 (5.4%)	1 (5.3%)	--
Porites astreoides	5 (2.7%)	--	--
Diploria strigosa	4 (2.2%)	--	--
Montastraea cavernosa	2 (1.1%)	--	--
Mycetophyllia sp	2 (1.1%)	--	--
TOTAL	185	19	121

Total number of all diseased colonies = 325

TABLE 2

The number of coral colonies infected with white plague (WP), black band disease (BBD) and dark spots disease (DSD) at each site between March and August 2000.The area surveyed and the total number of diseased colonies per m^2 at each site are also shown

Site	WP	BBD (# of colonies)	DSD	Area surveyed (m^2)	# of diseased colonies m^{-2}
Floral Gardens	37	1	9	826	0.06
Salisbury	4	3	56	2134	0.03
Tarou Point	30	12	22	1506	0.04
Coral Gardens	45	2	30	881	0.09
Cachacrou	69	1	4	537	0.14

TABLE 3

Total disease prevalence (WP = white plague, BBD = black band disease and DSD = dark spots disease) at each site in March, June and August 2000

	March			June			August		
Site	WP	BBD	DSD	WP	BBD	DSD	WP	BBD	DSD
Floral Gardens	3	0	0	14	0	8	25	1	6
Salisbury	1	0	5	0	2	36	3	2	39
Tarou Point	3	1	3	17	3	12	21	11	12
Coral Gardens	2	0	1	30	2	22	25	1	18
Cachacrou	6	1	0	42	0	4	25	0	1
Total (all sites)	15	2	9	103	7	82	99	15	76
Total (all sites & all diseases)		26			192			190	

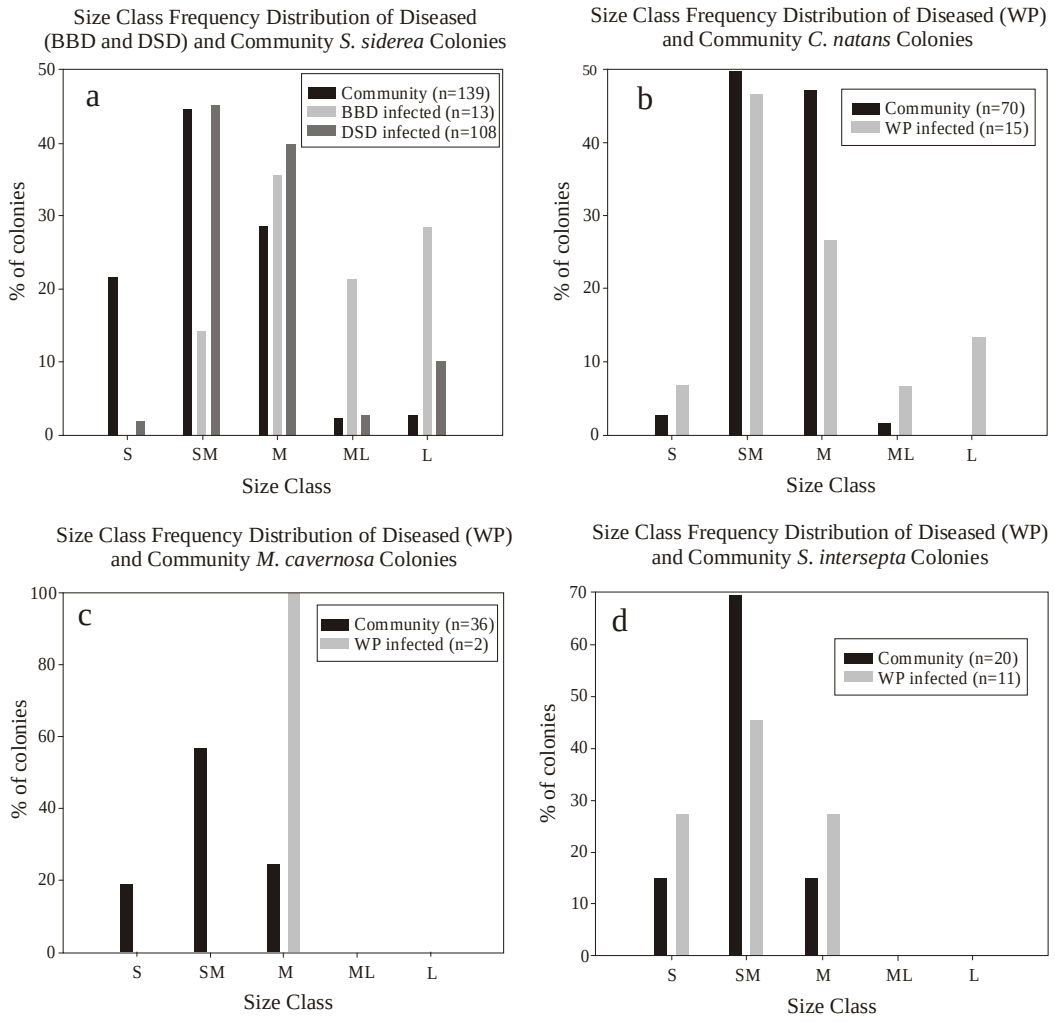

Fig. 2. The size class frequency distributions of coral colonies measured randomly at each study site ("community") and colonies infected with diseases: a. *S. siderea* community, BBD infected and DSD infected colonies, b. *C. natans* community and WP infected colonies, c. *M. cavernosa* community and WP infected colonies, d. *S. intersepta* community and WP infected colonies. Size classes are categorized as follows: S = small (0 – 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 = larger (> 5001 cm^2).

Total disease prevalence increased from March to June 2000 at all sites (Table 3). However, from June to August, disease numbers displayed a wide variation of increases and decreases between sites. For example, WP increased from 14 diseased colonies to 25 (+78.6%) from June to August at Floral Gardens, while at Cachacrou, WP numbers decreased from 42 to 25 (-40.5%) during the same time period. The overall prevalence of diseased colonies recorded in June and August was nearly identical (192 in June vs. 190 in August).

BBD and DSD affected the larger colonies of *S. siderea* across all sites (BBD: chi-square; p=<0.001, χ^2=37.79, d.f.=3 and DSD: chi-square; p=0.025, χ^2=10.47, d.f.=3) (Fig. 2a.), whereas WP affected the larger colonies of *C. natans* (chi-square; p=0.025, χ^2=10.25, d.f.=3) (Fig. 2b.). Though there were not enough values to execute the Chi-Square contingency table analysis, it appears that WP also affected the

larger colonies of *Montastraea cavernosa* (Linnaeus, 1767) (Fig. 2c.). In the case of all other susceptible species (see Table 1. for list), WP infections mirrored the size class frequency distributions and had the highest numbers of infections on the most abundant colony size class present as is evident for *S. intersepta* (Fig. 2d. - data for other susceptible species not shown).

Of 103 WP infections, 32 (31.1%) infections arrested from June to August. BBD persisted over time with stable infections that progressed slowly and consistently between March and August. Only 3 cases of DSD (3.66%) arrested from June to August.

Four colonies with active WP infections acquired additional diseases or infections during the survey period. Two of the colonies (both *S. siderea*) became infected with BBD and DSD, and two (both *M. faveolata*) acquired additional WP infections. The estimates of both infection cessation and cases of multiple/additional infections are likely underestimated due to the loss of reference tags.

Disease intensity and disease-induced tissue and colony mortality: WP destroyed the largest average tissue surface area per colony, relative to colony size (21-40%, n=165), and these values differed between species (Table 4). *M. meandrites* and *Mycetophyllia* sp. experienced the largest WP infections relative to total colony size (average of 41- 60% of the total colony). There were, on average, 1.6 ± 0.1 (n=165) WP infections per colony.

DSD (which only affected one coral species) had the highest average number of infections per colony (13.3 ± 1.6, n=100) but simultaneously affected the smallest amount of tissue per colony (1-20%, n=100) (Table 4.). BBD also only destroyed a small percentage of each colony (1-20%, n=18) and displayed an average of 1.6 ± 0.3 infections per colony. The species with the largest BBD infection (relative to colony size) was *D. stokesi*. It is important to note that the species *M. annularis* was omitted from these analyses due to the difficulty in identifying single colonies in larger, massive clusters (thereby making any measurements standardized to colony impossible).

The total area of coral tissue mortality due to the three diseases at the five study sites from March to August 2000 was 4.08 m^2 (Table 5). The estimated total living coral tissue area across all sites was 600 m^2 (this value was calculated by multiplying the overall site area (m^2) by site-specific % coral cover measurements taken by S. Steiner). Thus, tissue

TABLE 4

The mean percent of colony tissue affected by each disease (values based on estimations into % subgroups: 1 – 20%, 21 – 40%, 41 – 60%, 61 – 80%, 81 – 100%), and the mean number of infections per colony of each species with white plague (WP), black band disease (BBD) and dark spots disease (DSD).

	WP (n=155)			BBD (n=15)			DSD (n=100)		
Species	n	mean % affected	mean # of infections (per colony)	n	mean % affected	mean # of infections (per colony)	n	mean % affected	mean # of infections (per colony)
S. siderea	35	1 – 20	2.2 (±0.1)	13	1 – 20	1.7 (±0.1)	100	1 – 20	13.3 (±1.6)
M. faveolata	30	1 – 20	1.3 (±0.1)		--	--		--	--
M. meandrites	23	41 – 60	1.1 (±0.2)		--	--		--	--
C. natans	24	21 – 40	1.4 (±0.2)		--	--		--	--
S. intersepta	11	1 – 20	1 (±0.2)		--	--		--	--
A. agaricites	10	21 – 40	1.1 (±0.3)	1	1 – 20	1 (±0)		--	--
D. stokesi	10	1 – 20	1.4 (±0.1)	1	21 – 40	1 (±0)		--	--
P. astreoides	5	1 – 20	2 (±0)		--	--		--	--
D. strigosa	4	1 – 20	1.3 (±0)		--	--		--	--
M. cavernosa	2	21 – 40	2 (±0.5)		--	--		--	--
Mycetophyllia sp.	1	41 – 60	1 (±0)		--	--		--	--
Overall Mean		21 – 40	1.6 (±0.1)		1 - 20	1.6 (±0.3)		1 - 20	13.3 (±1.6)

TABLE 5

Total area (cm^2) of coral tissue mortality per coral species caused by white plague (WP), black band disease (BBD), and dark spots disease (DSD) between March and August 2000, and the percent of total coral tissue cover (area) contributedby each species within 200 m^2 (surveyed by S. Steiner, in press).**no data: lost tagging nail*

Species	WP cm^2	BBD cm^2	DSD cm^2	TOTAL cm^2	% coral tissue cover
Siderastrea siderea	969	1885	2659	5513	12.2
Montastrea faveolata	13646	--	--	13646	8.9
Montastrea annularis	4925	--	--	4925	4.9
Meandrina meandrites	3306	--	--	3306	11.2
Colpophyllia natans	5753	--	--	5753	2.2
Stephanocoenia intersepta	1102	--	--	1102	1.3
Agaricia agaricites	440	80	--	520	0.2
Dichocoenia stokesi	770	--**	--	770	0.4
Diploria strigosa	2556	--	--	2556	1.5
Montastrea cavernosa	2423	--	--	2423	1.3
Mycetophyllia sp.	289	--	--	289	0.02
TOTAL (m^2)	3.62 m^2	0.19	0.27	4.08	44.1†
tissue loss/infection (cm^2)	125.62	65.50	1.96		

* Based on calculations of coral tissue cover in 40 m^2 quadrats at each site (total = 200 m^2).
† The remaining 55.9% of coral tissue cover consists of other, non-susceptible coral species.

loss was equal to approximately 0.68% of all living tissue within the 24-week time frame. The majority of this loss (88.7%) was caused by WP infections, with *M. faveolata* exhibiting the highest amount of tissue loss. Five colonies were completely killed by WP during this time period. These included two *M. meandrites* colonies, two *C. natans* colonies and one *Mycetophyllia* sp. colony. According to data collected by Steiner (in prep.) on coral species community structure at all five study sites, *C. natans*, *M. cavernosa*, *M. faveolata*, *M. annularis* and *Mycetophyllia* spp. together accounted for <17.5% of total coral tissue cover across all sites (Table 5). These five species experienced 66.3% of total coral tissue death from disease. Comparisons of estimates of tissue loss per infection indicate that WP had the largest amount of tissue loss per infection (125.6 cm^2) and DSD had the smallest (1.96 cm^2) (Table 5).

Physical and community measurements: The water temperature in Dominica was 26ºC in March, 28ºC in June, and 28.5º C in August. Though the number of WP, BBD and DSD infections increased from March to June (coinciding with a 2°C increase in water temperature), there were both increases and decreases in disease prevalence between June and August. Therefore, there was an initial increase in disease densities that correlated with a small increase in water temperature, however, due to the dynamic fluctuations in diseased colony numbers between June and August, there was no direct correlation between water temperature and disease densities over the three survey periods.

Analysis of data thus far on wave height, turbulence (surge), depth, low visibility and currents compared to disease distribution did not render significant results (Table 6). There was a significant, negative relationship between DSD prevalence and diversity values (Shannon-Weiner Index, H') at each site (Pearson; p=<0.04, r=-0.91, d.f.=2). However, there were no other correlations between diversity values or percent coral cover and disease prevalence at each site.

There was a significant, positive correlation between the numbers of WP infected colonies and the relative abundances of target species

TABLE 6

The average wave height[*], average depth, occurrence of sustained low visibility (<2m), direction of predominant currents, presence of surge, diversity (Shannon-Weiner Index, H'), percent coral cover relative to total substrate area[**] and the total tissue area (cm^2) of target species at each site

Site	wave height (n=10)	depth (m)	low visibility	current direction	surge	diversity (H')	% coral cover	target species tissue area (cm^2)	
								WP	BBD/DSD
Floral Gardens	6	15.2	no	N and S	no	0.91	16.7	348.5	34.5
Salisbury	4.2	4.5	yes	N and NW	yes	0.57	21.5	134	112
Tarou Point	5.3	6.1	yes	Eddying	yes	0.87	12.1	178.5	127.5
Coral Gardens	6	12.1	no	S and N	no	0.80	23.3	372.5	157
Cachacrou	6	16.7	no	none detected	no	0.85	17.4	410.5	15

*Van Duyl Scale of wave heights (1985): 1=2.0–3.5 m, 2=1.5–2.0 m, 3=1.0–1.5 m, 4=0.5–1.0 m, 5=0.3–0.5 m, 6=0.0–0.3 m
** Based on calculations of coral tissue cover in 40 m^2 quadrats at each site (total = 200 m^2).
[***]Target species for WP = *S. siderea*, *M. meandrites*, *M. annularis*, *M. faveolata* and *C. natans*; target species for BBD & DSD = *S. siderea*.

TABLE 7

*Observations of potential disease transmission vectors (damselfish,*D. antillarum *and* H. carunculata*) associated with colonies infected with white plague (WP), black band disease (BBD) and dark spots disease (DSD)*

Potential Vector	WP	BBD	DSD	TOTAL
damselfish	13	1	27	41
Diadema antillarum	3	1	6	10
Hermodice carunculata	9	1	1	11
TOTAL	25	3	34	62

(Table 6). Target species for WP were *S. siderea*, *M. meandrites*, *M. annularis*, *M. faveolata* and *C. natans*. The amount of coral tissue area contributed by each of the species (measured randomly within 40 m^2 at each site) were combined for each site and compared to the density of WP at each site, and there was a positive, significant correlation (Pearson; $p<0.05$, $r=0.89$, d.f.=2). The target species for BBD and DSD was *S. siderea*. When comparing the densities of BBD and DSD infected colonies at each site to the relative abundance of *S. siderea*, there were no significant relationships.

There were a total of 62 associations of diseased colonies with potential disease transmission vectors (Table 7). Of the damselfish associations, 65.9% occurred on colonies with DSD, while 81.8% of the *Hermodice carunculata* associations were on colonies with WP. In the latter case, the fireworm was frequently seen located directly on top of the disease-healthy tissue interface.

Spatial distribution analysis: Diseased individuals were clustered within each site examined and for each disease measured. The frequency distributions further indicated that the diseased colonies were clustered across space (Fig. 3 a-g). Comparison of predicted (Poisson) and observed nearest neighbor distances for BBD at Tarou Point exhibited a significant difference (chi-square; $p<0.0001$, $\chi^2=79.45$, d.f.=2). WP infected colonies were also clustered at Tarou Point (chi-square; $p<0.0001$, $\chi^2=104.44$, d.f.=9), Floral Gardens (chi-square; $p<0.0001$, $\chi^2=75.49$, d.f.=9), Cachacrou (chi- square; $p<0.0001$, $\chi^2=548.66$, d.f.=9) and Coral Gardens (chi-square; p=<0.05,

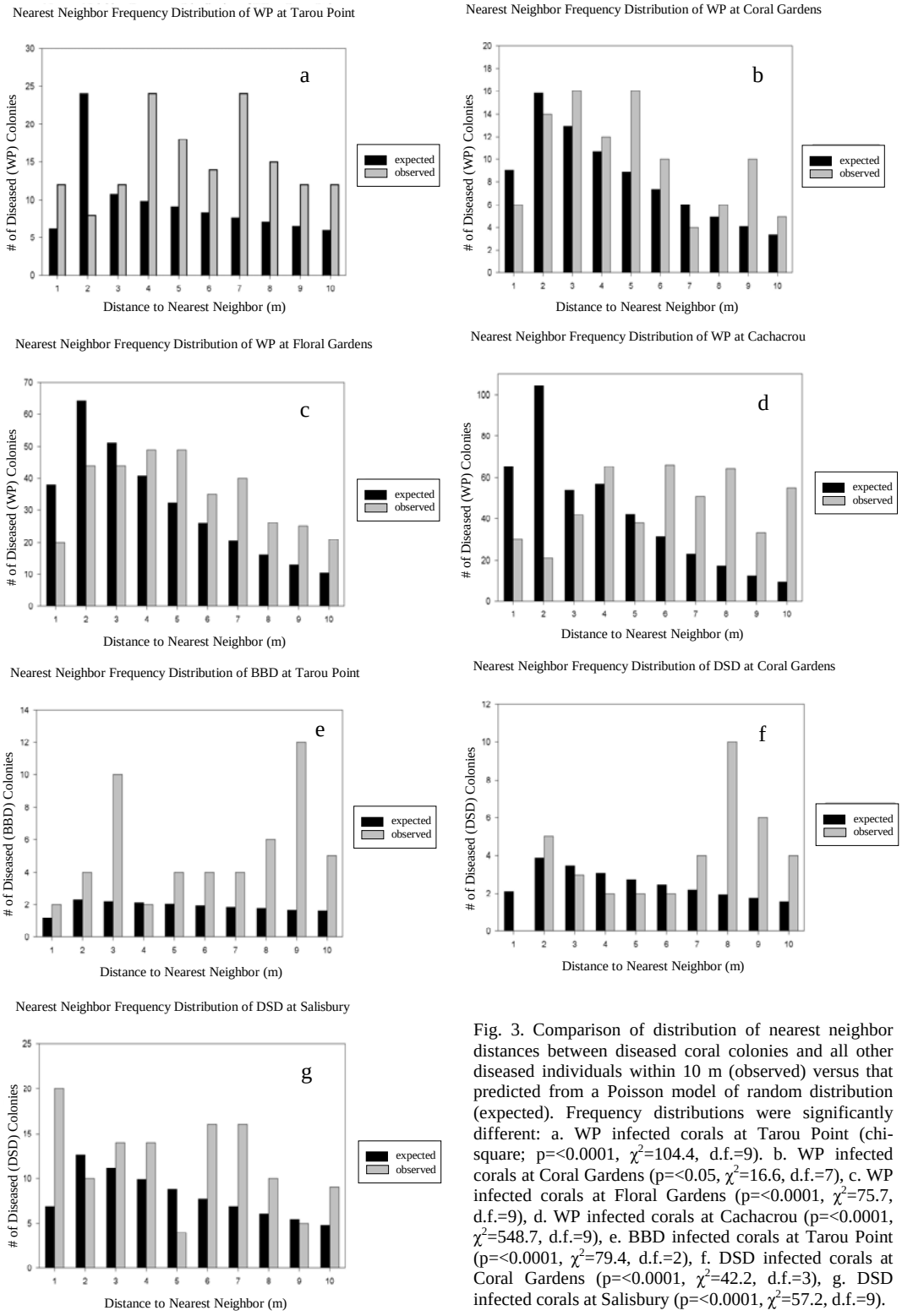

Fig. 3. Comparison of distribution of nearest neighbor distances between diseased coral colonies and all other diseased individuals within 10 m (observed) versus that predicted from a Poisson model of random distribution (expected). Frequency distributions were significantly different: a. WP infected corals at Tarou Point (chi-square; p=<0.0001, χ^2=104.4, d.f.=9). b. WP infected corals at Coral Gardens (p=<0.05, χ^2=16.6, d.f.=7), c. WP infected corals at Floral Gardens (p=<0.0001, χ^2=75.7, d.f.=9), d. WP infected corals at Cachacrou (p=<0.0001, χ^2=548.7, d.f.=9), e. BBD infected corals at Tarou Point (p=<0.0001, χ^2=79.4, d.f.=2), f. DSD infected corals at Coral Gardens (p=<0.0001, χ^2=42.2, d.f.=3), g. DSD infected corals at Salisbury (p=<0.0001, χ^2=57.2, d.f.=9).

χ^2=16.63, d.f.=7). DSD nearest neighbor distance frequencies at Salisbury (chi-square; p<0.0001, χ^2=57.22, d.f.=9) and Coral Gardens (chi-square; p< 0.0001, χ^2=42.16, d.f.=3) exhibited significant differences when compared to Poisson distributions.

DISCUSION

Coral diseases are contributing to the death of coral tissue in Dominica. In only 6 months, over 4 m^2 of coral tissue area was lost to diseases. Although this value contributed to less than 1% of the total coral tissue area for all the sites combined, the deleterious effects were species specific. This may be compounded if a disease exhibits multi-year, repeat infections on the same colonies (Kuta and Richardson 1997) or if high disease prevalence persists for many years. Based upon comparisons between coral species community structure (Steiner in prep.) and the amount of tissue loss per species, the species most threatened by diseases on these reefs are *C. natans*, *M. cavernosa*, *M. faveolata*, *M. annularis* and *Mycetophyllia* spp. (Table 5). The prevalence of WP at certain sites (e.g. Cachacrou, with almost 1 infection per 6 m^2) was comparable to episodes described as epizootics and/or outbreaks in other locations (Richardson *et al.* 1998, Bruckner and Bruckner 1997a). WP was responsible for 3.62 m^2 (88.7%) of coral tissue death, and is therefore the coral disease most strongly impacting Dominica. This is likely a function of the overall higher prevalence of WP, the faster progression rates, and the higher disease intensity when compared to BBD and DSD. BBD and DSD together contributed to only 11% of the total coral tissue loss. BBD persisted at low levels on only selected reefs, and DSD appeared in most cases to be an unchanging discoloration of tissue with little apparent tissue loss. Therefore, the impact of these two diseases on corals in Dominica was minimal during the survey period.

Coral species infected with WP and BBD in Dominica differed from those in most other described Caribbean locations. In the case of WP, Richardson *et al.* (1998) noted that *D. stokesi* was the coral species most susceptible to WP in the Florida Keys. Although there were colonies of *D. stokesi* with WP in Dominica, these contributed to only 5.6% of all WP infections recorded. In a WP outbreak in Puerto Rico however, Bruckner and Bruckner (1997a) noted *Siderastrea* as one of the susceptible genera. This appears to be a more similar situation to Dominica, where *S. siderea*, *M. faveolata* and *M. annularis* were the three most susceptible species.

BBD also exhibited a different species infection pattern. Most studies of BBD identify *Diploria labyrinthiformis*, *Diploria strigosa* (Dana, 1848), *M. annularis*, *M. cavernosa* and *C. natans* as the most susceptible species (Garrett and Ducklow 1975, Antonius 1981, Rützler *et al.* 1983, Edmunds 1991, Kuta and Richardson 1997, Grosholz and Ruiz 1997). There were no BBD infections recorded on any of these species in Dominica. However *S. siderea* accounted for over 84% of the BBD infections. Bruckner and Bruckner (1997b) did note that *S. siderea* was susceptible to BBD in Jamaica. *Stephanocoenia intersepta* (Edwards and Haime, 1848) (unpublished 2001 data), *D. stokesi* and *Agaricia agaricites* (Linnaeus, 1758) were also recorded with BBD infections, which has not been documented elsewhere. This differs from data collected thus far, which has indicated that non-framework, scleractinian species are not susceptible to BBD (Kuta and Richardson 1997).

Based on these observations, it may be difficult at present to assume generalizations about species-specific infections for each disease across the Caribbean region as a whole, because susceptible species apparently differ in various locations.

BBD, DSD and WP all affected the larger colonies of certain species across all sites surveyed, which have important ramifications. The ability of a coral colony to grow to a larger size increases its chance of long-term survival (Hughes and Jackson 1985), and larger colonies may also have both a larger reproductive output (Kojis and Quinn 1984, Szmant-Froelich 1985, Szmant 1986) and greater fitness (Hughes 1984). In addition, because many coral species' early life history involves a reproductive delay, larger colonies are likely contributing more to reproduction

than smaller conspecifics (Connell 1973). Therefore, by affecting the larger colonies of a community and by breaking large tissue areas into smaller fragments via partial mortality, diseases may be causing long-term changes (via reduction of reproductive output) and deleterious effects on the community structure and longevity of reefs.

The fluctuations in water temperature in Dominica are small, changing less than 3° C in a year's time. This is primarily due to the island's high relief topography, in which there is a short shelf extending into the deep, offshore waters. However, diseases in Dominica had higher prevalence in the summer. The temperature threshold for increases in coral diseases may be less than 2ºC, or there may be other confounding environmental and/or physical factors that may be contributing to this pattern. A multi-year investigation, which is underway, may clarify these discrepancies.

The lack of a relationship between depth and disease prevalence differed from what Richardson *et al.* (1998) reported in the Florida Keys where higher densities of WP occurred at a depth of 14 m. Bruckner and Bruckner (1997) discovered patterns between current movement and the spread of BBD across reefs, but there were no such relationships in Dominica. The latter may be due to the inconsistency of current patterns along the west coast of Dominica (predominant currents alternating with tidal currents in opposing directions) and/or the small scale used in this analysis (per site vs. across sites).

Higher prevalence of BBD has been observed in areas with relatively higher pollution levels (Antonius 1988). Although no water quality data were taken, Tarou Point, the site with the highest prevalence of BBD, is qualitatively the most polluted site in Dominica. This site is less than 1 mile from the largest factory (soap processing) and river (Layou River) in Dominica. The northern cove of the point collects a large amount of organic (e.g. deceased animals) and synthetic debris that was noted to reduce the visibility in the area by as much as 10 m in less than one hour. Therefore, there may be a qualitative relationship between the higher prevalence of BBD at Tarou Point and levels of pollution.

The spatial distribution patterns of WP, BBD and DSD all indicated a clumped or clustered pattern. This reveals a similar distribution pattern to that of an infectious, horizontally transmitted disease. These findings are in agreement with those described by Bruckner *et al.* (1997) and Kuta and Richardson (1997) for the distribution patterns of BBD. There are still many questions remaining as to how diseases are being transmitted between colonies. The data collected on potential transmission vectors (62 associations with diseased colonies) warrants further attention.

The distribution of WP, the disease with the greatest, negative impact on corals in Dominica, across sites was related to the abundance of susceptible species, and there was a significant, positive correlation between these two parameters. Thus, host species preference may be an important factor in predicting where specific diseases might occur. This could be valuable for consideration in management programs designed to preserve coral reefs.

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