Caribbean Journal of Science, Vol. 41, No. 4, 815-823, 2005

Impact of a White Plague-II Outbreak on a Coral Reef in the Archipelago Los Roques National Park, Venezuela

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ABSTRACT.—During the last quarter of 2000, an outbreak of white plague disease (WPD-II) caused significant coral tissue mortality in several reef building species (*Montastraea* species complex, *Colpophyllia natans* and *Stephanocoenia intersepta*) in a fringing reef along Madrizquí Key, Los Roques National Park, Venezuela. Coral cover and abundance by species, and cover of other substrates (i.e., algae, sand, etc.) were measured using eleven 1 m^2 quadrats (divided in 100 areas of 100 cm^2 each) separated by two meter intervals along each of the eight, 20 m long transects haphazardly placed between 5-12 m depth. Measurements were taken at the onset of the epizootic event and a year later. The average coral cover and cover of other substrates were compared using a Friedman ANOVA, while changes in relative cover by species were tested using a t-test. *C. natans* and the *Montastraea* species complex showed the highest significant (t-student, $p < 0.05$) loss in live cover, from 12.28% to 8.11% and 38.34% to 34.84%, respectively. Overall, the average live coral cover decreased significantly (Friedman = 581.5, $p < 0.05$) from 35% in 2000 to 29% in 2001, with a corresponding significant increase in algal and bare substrate cover from 33% to 44%. Similar to other Caribbean areas, the results of this study indicated that outbreaks of WPD-II may cause fast and extended coral tissue mortality, with significant reductions in live coral cover with a corresponding increase in algal cover in a relatively short period of time.

KEYWORDS.—Coral diseases, Caribbean, white plague, reef community, *Montastraea*, *Stephanocoenia intersepta*, *Colpophyllia natans*

INTRODUCTION

Over the past decades, coral reefs have experienced significant changes in structure and function (Harvell et al. 1999; Jackson et al. 2001; Hughes et al. 2003; Gardner et al. 2003). Although many factors have contributed to this world-wide trend in coral reefs, in the wider Caribbean, coral diseases have played a central role in massive mortalities of key reef species (Richardson 1998; Weil 2004). The mass mortality of acroporids (Gladfelter 1982) and the die-off of the black sea urchin, *Diadema antillarum* (Lessios et al. 1984; Carpenter 1990a, b), were the most relevant events. The significant reduction in the population numbers of these key species produced significant impacts on the dynamics, function and structure of coral reefs throughout the region at local and geographic scales (Hughes 1994; Harvell et al. 1999; Aronson and Precht 2001; Bruckner et al. 2002). The only documented wide-spread and persistent epizootic event or condition currently occurring in the wider Caribbean is aspergillosis on sea fans (Kim et al. 2000; Weil et al. 2002; Weil 2004; Smith and Weil 2004); however, increasing frequencies of more localized outbreaks of other virulent diseases and syndromes such as yellow blotch and white plague type II, have been causing problems in many coral reef localities in the last decade (Weil 2004).

White plague disease was first described in the 1970s (Dustan 1977). It was later named WP type I to differentiate it from a more virulent event that produced mass mortalities of *Dichocoenia stockesii* in the

ms. received May 17, 2004; resubmitted February 25, 2005; accepted August 15, 2005.

Florida Keys and affected 15 other species (Richardson 1998). The potential putative pathogen was primarily isolated by Richardson et al. (1998b) and named WP type II (WP-II). It kills tissue at rates of 2-3 cm/day, which significantly exceeds the linear growth of any scleractinian coral (Richardson et al. 1998a, b). Recently, the pathogen was identified as *Aurantimonas coralicida*, a new bacterial genus (Denner et al. 2003). This particular disease is considered among the most damaging to coral populations because of its frequent outbreaks and fast killing of coral tissue of important species. This disease also has the widest host range affecting up to 41 coral species (Weil et al. 2002; Weil 2004). In the field, colonies infected by WP-II are easily distinguished from those with bleaching and/or predation signs because of a uniform, recently exposed, white band of clean skeleton and the fast mortality of living tissues.

During the last quarter of 2000, an epizootic event of WP-II occurred in coral reefs around Madrizquí Key, in Los Roques National Park, an oceanic reef system considered among the best conserved and relatively protected reef areas in the southern Caribbean (Zubillaga 2001; Weil 2003; Cróquer et al. 2003). This event caused significant mortalities in most of the common reef builder species (e.g., *Montastraea annularis*, *M. faveolata*, *Colpophyllia natans*). The availability of previous data on the structure and conditions of the coral community for this site (Zubillaga 2001), and the timely identification of the onset of the epizootic event, provided an excellent opportunity to study the changes/impact produced by the disease at the species and community level. The aims of this study were then to determine the effects of the WP-II epizootic event on the composition/structure of the coral community of Madrizquí key by assessing the changes in live coral cover of the major species and cover of other substrates from 2000 and 2001.

Materials and Methods

Study site

The Archipelago of Los Roques, located 160 km north off the Venezuelan coast (11°44′26′′-11°58′36′′ N, 66°32′42′-66°57′26′′ W; Fig. 1), is an oval-shaped, semi-atoll coral reef ecosystem. It is formed by two long fringing reef systems, including the eastern (20 km long) and southern (30 km long) reef barriers. The system is comprised by more than 40 coralline keys with fringing reefs, hundreds of patch reefs, over 200 sand banks, and extensive mangrove forests and seagrass beds (Weil, 2003). In general, coral reefs are healthy with high live coral cover (30-70%), abundant herbivorous and top predatory fish, low algal cover, and no direct impact of anthropogenic factors such as land development and/or sewage (Hung 1985; Zubillaga 2001; García et al. 2002; Weil 2003). The reef site surveyed is located to the east of Madrizquí Key and it is oriented SE-NW and close to El Gran Roque Island (Fig. 1). The reef forms a windward barrier separated by a sandy lagoon (50 to 100 m wide and 0.5-1.0 m depth). A shallow terrace of dead *Acropora palmata* followed by scattered colonies on a sandy substrate fringes this sloping reef to a depth of 10 to 15 m. The lower slope, which descends to 6-12 m, is the most extensive reef zone with the highest coral abundance and diversity compared to the reef platform (Cróquer et al. 2003).

Survey methods

Areas sampled in 2000 (at the onset of the WP-II outbreak) were re-surveyed in 2001 to assess changes in the cover of live coral tissue at the species level and changes in other important biological components (e.g., algae and bare substrate) as an indicator of WP-II impact. Eight 20 m long permanent transects randomly set in 2000 were re-used in 2001. Transects ran parallel to the long axis of the reef, between 5-12 m deep, where the highest coral cover and biodiversity were observed. Eleven 1 m^2 quadrats, 2 m apart were surveyed along each transect. Colonies of all species under the quadrats were checked for presence of the disease and predation. Live cover was measured for each coral species, other organisms, and substrates (e.g, algae and bare substrate).

Tissue mortality rate.—Fifteen colonies in-

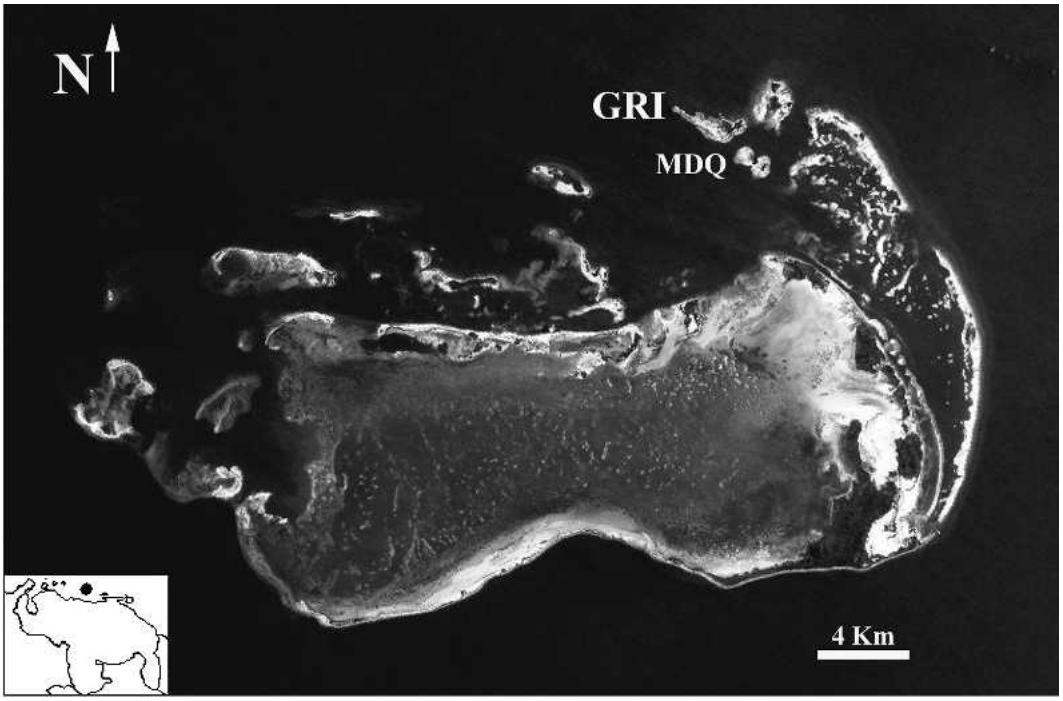

FIG. 1. Los Roques National Park, northern Venezuela. GRI: Gran Roque Island, MDQ: Madrizquí coral reef (white dot).

fected with WP-II (i.e., 6 *M. faveolata*, 1 *M. franksi*, 1 *M. cavernosa*, 1 *Siderastrea siderea*, 1 *S. intersepta*, 3 *Diploria strigosa* and 2 *C. natans*) were tagged to roughly estimate coral tissue lost during 2000-2001, and to calculate the rate of tissue mortality. Tagged colonies were classified into qualitative intervals of mortality (0-25, 25-50, 50-75 and 75-100%). Three nails were hammered at the edge of bare skeleton and infected tissues, and the following year we measured the distance from each nail to the necrotic area. The rate of tissue mortality was grossly estimated as the total amount of tissue lost divided by the period of observation (12 months).

Statistical analysis

Since cover data of the different substrate categories, from 2000 to 2001, did not have a normal distribution they were compared using Friedman ANOVA. The relative cover for each coral species was compared using a t-test for dependent samples. Normality was tested with Kolmogorov-Smirnov and Shapiro-Wilki test, while a Cochran test (Zar 1998) was used to test homogeneity of variances.

RESULTS

The total cover of live coral in the sampled area significantly decreased (Friedman ANOVA $p < 0.05$) from 34.5% to 28.5% in one year. These open areas were rapidly colonized by turf algae, which increased the algal cover from 32.5% to 40%. Coral rubble, formed mostly by recently dead *Madracis mirabilis* fragments, covered the sandy patches on the shallow habitats, which significantly increased rubble cover and decreased sand cover (Friedman ANOVA $p < 0.05$). No significant differences were found in the cover of hydrocorals, octocorals and sponges (Table 1) as none of these organisms were affected by WP-II in 2000.

In 2001, the general appearance of the reef was different compared to the previous year. In 2000, at least 24% of all coral

TABLE 1. Mean cover and standard deviation for different substrata at Madrizquí Key coral reef in 2000 and 2001. *Significant (Friedman ANOVA, $p < 0.05$). Live coral cover (LCC), dead coral covered by turf algae (DCOR + TURF), rubble (RUBB), hydrocorals (HIDR), sponges (ESP), octocorals (OCT), fleshy algae (FALG) and sand (SAND) from 2000 to 2001 after the WPD-II mortality event at Madrizquí Key, Los Roques National Park, Venezuela.

Friedman ANOVA and Kendall concordance coefficient (n = 88, df = 15) = 581.85, p = 0.001 Kcoenf = 0.477								
	2000				2001			
Substrate	Mean	Sd	Rank	Sum of Ranks	Mean	Sd	Rank	Sum of Ranks
LOC	34.62	21.14	13.82	1202.00	28.56	21.80	13.21	1149.00
DCOR	32.16	26.20	12.20	1061.50	43.61	31.88	12.57	1093.50
RUBB	7.89	20.02	6.67	580.50	9.31	22.60	6.70	583.00
HDR	0.05	0.21	4.98	433.50	0.96	2.14	6.11	532.00
ESP	3.63	9.17	7.41	644.50	3.87	9.45	7.40	643.50
OCT	3.83	7.89	7.40	644.00	5.27	8.48	8.20	713.50
FALG	0.42	1.12	5.61	488.50	0.62	1.66	5.78	503.00
SAND	25.84	38.49	9.93	864.00	7.81	17.01	8.00	696.00

colonies were infected with WPD-II, including *M. faveolata* (50%), *M annularis* (40.9%), *M. franksi* (25.56%), *C. natans* (27.09%) and *S. intersepta* (26.19%) (Table 2, Figs. 3A-3F). Other species, such as *D. strigosa* and *S. siderea*, exhibited few colonies infected (Table 2). In 2001, relatively few colonies exhibited signs of WP-II and large colonies of *M. faveolata* and *M. annularis* were almost completely cleared of live tissue, having only small patches of remnant live tissue in some areas. Furthermore, colonies of *C. natans* and other species were partially bioeroded by parrotfish grazing (see increase of percentage of predation on Table 2).

The relative cover of *C. natans* decreased significantly (t-student, $p < 0.05$) from 12.28% to 8.11% (Table 3). A similar trend was observed for the *Montastraea* species complex, whose pooled live percent cover significantly decreased from 38.34% to 34.84%, while *M. mirabilis* decreased from 12.51% to 9.24% (t-student $p < 0.05$, Table 3). No significant differences were found for *M. cavernosa*, *D. strigosa*, *Undaria danae* and hydrocorals (Table 3).

By the onset of the outbreak in 2000, 66% of all affected coral colonies had between 0-25% tissue mortality with very few with more than 75% tissue mortality, indicating that this event was starting (Table 4). The total linear tissue removed by WP-II during the survey period was 398.7 cm (n = 15 colonies) with an average of 26.58 ± 8.87 cm (Table 4). The rate of tissue mortality ranged from 0.92 cm/month (*Siderastrea siderea*) up to 3.34 cm/month (*D. strigosa*) with an average of 2.22 ± 0.74 cm/month (Table 4).

DISCUSSION

Increasing frequency of local coral disease outbreaks might cause, in a medium- to long-term, significant changes in the community structure and population dynamics of coral reefs (Aronson et al. 1998; Weil 2004). Therefore, the role that these outbreaks play in modifying the reef community structure and dynamics needs careful attention. White band disease caused the most geographically extensive impact on the Caribbean reefs in the early 1980's. Population declines of elkhorn (*Acropora palmata*) and staghorn (*A. cervicornis*) corals throughout the wider Caribbean significantly reduced the coral cover at local and geographic scales (Gladfelter 1982; Aronson and Precht 2001). This epizootic event changed the structural profile of shallow reef areas and brought about a series of important ecological cascading events that included a significant reduction in biomass and biodiversity when the tri-dimensional structure collapsed and the availability of habitat and refuges disappeared for many species (Harvell et al. 1999; Weil 2004). In the past decades, many different important

TABLE 2. Percentage of colonies (only major reef builders) with WP-II infection and other conditions at Madrizquí Key during the 2000 and 2001 period. (n = 926 in 2000 and 778 in 2001). BBD = black band disease, WP-II = white plague type II, DSD = dark spot syndrome, WBD = white band disease, YBD = yellow blotch syndrome.

Ag-Sep 2000	Healthy	BBD	WP	DSD	WBD	YBD	Predation	Pomacentrids	Sedimentation	N colonies
Montastraea faveolata	42.93	0.54	50.00			0.54	1.09	3.26	1.63	173
Montastraea annularis	53.03		40.91				1.21	1.82	3.03	310
Montastraea franksi	63.91		25.56				0.00	9.02	1.50	119
Diploria strigosa	27.78		13.89				16.67	8.33	33.33	15
Colpophyllia natans	56.98	1.16	27.91				6.98	2.33	4.65	74
Siderastrea siderea	83.02		5.55	5.88				5.55		19
Stephanochoenia intersepta	66.67		26.19	2.38				4.76		53
Porites astreoides	91.75						6.19	0.00	2.06	89
Acropora cervicornis	75.00				17.50		7.50			74
Ag-Sep 2001										
Montastraea faveolata	72.67		3.33			0.67	10.67	4.67	8.00	150
Montastraea annularis	88.19		1.11			0.37	4.80	4.06	1.48	271
Montastraea franksi	95.00		1.25				0.00	3.75	0.00	80
Diploria strigosa	45.28		1.89				41.51	11.32	0.00	53
Colpophyllia natans	58.11		2.70				8.11	12.16	18.92	74
Siderastrea siderea	100.00									11
Stephanochoenia intersepta	78.57						21.43			28
Porites astreoides	86.87						13.13			99
Acropora cervicornis	41.67				8.33		50.00			12

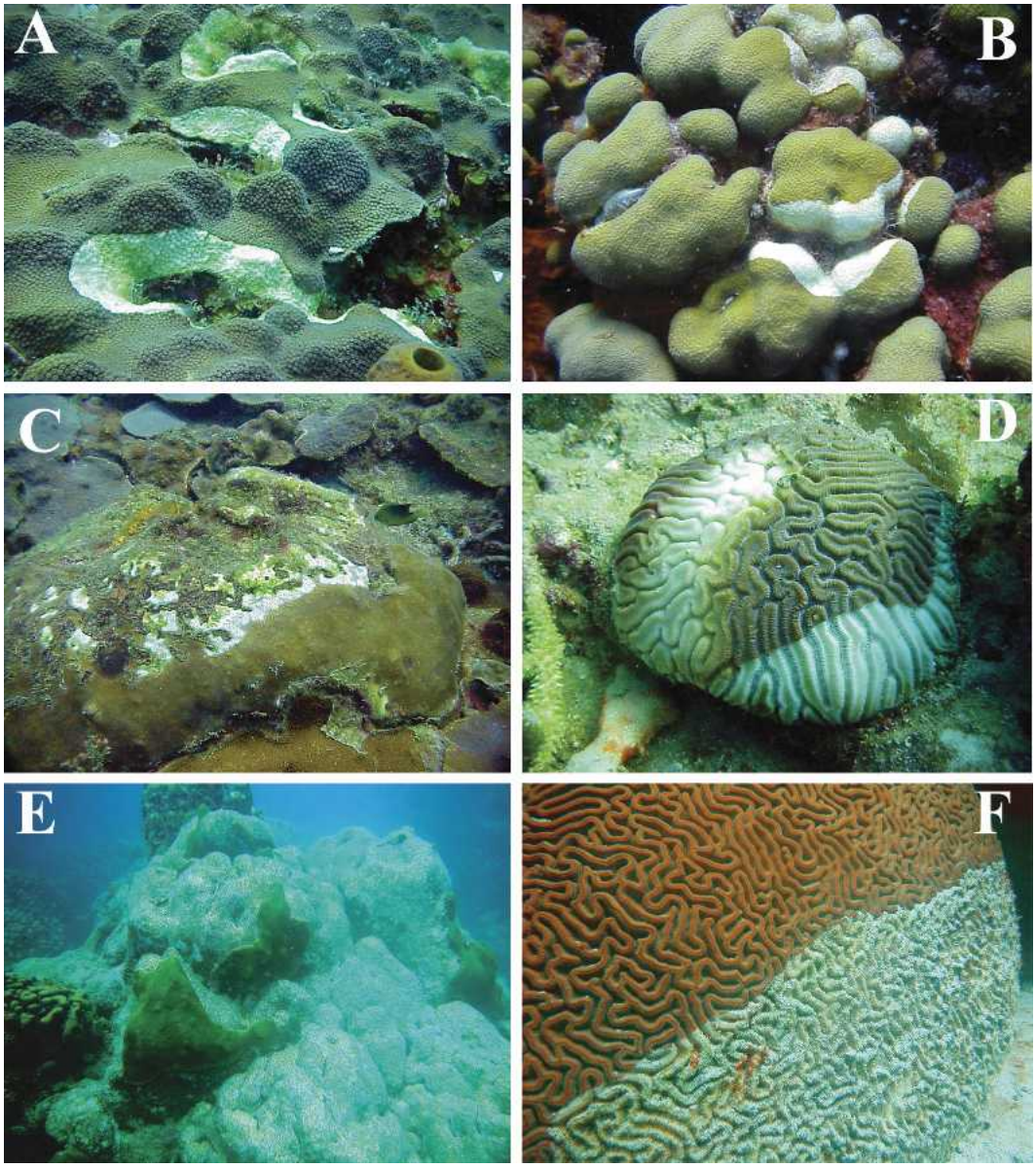

FIG. 2. Reef coral species affected by the WPD-II outbreak in Madrizquí Key. Large colonies of *Montastraea faveolata* (A) and *M. annularis* (B) at the base of the reef. Infected colonies of *Stephanocoenia intersepta* (C) and *Diploria labyrinthiformis* (D). A large *Montastraea faveolata* with 90% of partial mortality (E), and *Colpophyllia natans* with 50% mortality (F).

reef organisms in Caribbean reefs have been affected by disease problems (Weil et al. 2002; Weil 2004).

Currently, WP-II is one of the most widespread diseases in the wider Caribbean affecting the highest number of coral species. Even though no geographically widespread epizootic event has ever been reported, this disease shows the highest frequency of local outbreaks throughout the region (Florida Keys, Puerto Rico, Colombia, Virgin Islands, Dominique, Bermuda,

Fig. 2 live 4/C

TABLE 3. Average live tissue cover change of different reef-building species from 2000 to 2001 in Madrizquí Key reef after the WP-II mortality event with results of t-test. ++ *Montastraea* species complex *significant ($p < 0.05$).

Species (hard corals)	Mean 2000	Mean 2001	t-value	df	p-level
Montastraea spp. complex	38.34*	34.84*	2.79	14	0.04*
Montastraea cavernosa	0.570	1.594	–0.202	14	0.842
Colpophyllia natans	12.28*	8.11*	2.96	14	0.001*
Diploria strigosa	0.700	1.798	–0.392	14	0.700
Diploria labyrinthiformis	2.260	3.930	–0.204	14	0.896
Madracis mirabilis	12.51*	9.24*	2.98	14	0.03*
Madracis decactis	0.130	0.569	–0.199	14	0.888
Agaricia agaricites	0.880	0.650	–0.261	14	0.898
Agaricia agaricites danae	4.790	6.335	0.989	14	0.555
Agaricia agaricites purpurea	0.350	0.904	–0.302	14	0.766
Porites astreoides	1.740	3.315	2.03	14	0.09
Porites porites	1.290	1.903	–0.238	14	0.812
Esusmilia fastigiata	0.250	0.510	0.04	14	0.962
Acropora cervicornis	4.26*	0.98*	3.01	14	0.001*
Meandrina meandrites	0.890	0.498	–0.231	14	0.822
Mycetophillia aliciae	0.130	0.400	–0.123	14	0.981
Millepora alcicornis	11.610	15.380	3.22	14	0.756

TABLE 4. Percentage of colonies with different mortality categories, total tissue removed (cm) in one year period (2000-2001), and rate of living tissue loss (cm/month) at Madrizquí Key.

	Category of infection (%)					
Species	0-25	25-50	50-75	75-100	Total tissue removed (cm)	Rate of tissue mortality (cm/month)
M. faveolata	x				35.38	2.95
M. faveolata	x				33.12	2.76
M. faveolata				x	40.01	3.33
M. faveolata	x				20.10	1.68
M. faveolata		x			21.78	1.82
M. faveolata		x			23.01	1.92
M. franksi	x				15.80	1.32
M. cavernosa	x				18.90	1.58
S. intersepta			x		22.02	1.84
D. strigosa	x				30.02	2.50
D. strigosa	x				36.06	3.01
D. strigosa	x				40.07	3.34
S. siderea	x				11.01	0.92
C. natans			x		25.66	2.14
C. natans	x				25.77	2.15
Total (n = 15)	66.68	13.33	13.33	6.66	Mean 26.58 sd 8.87	Mean 2.22 sd 0.74

Grenada, Venezuela, etc.) (Richardson 1998; Green and Bruckner 2000; Weil et al. 2002; Cróquer et al. 2003; Weil 2004). These outbreaks seem to vary across localities in many aspects such as: intensity (prevalence/incidence), virulence and dynamics (how many colonies and species are infected, amount of tissue killed and rates of tissue mortality), the habitats affected (different depths in different localities), and the timing of occurrence.

Results from this study support the hypothesis that WPD-II produced significant impacts in the coral community of the Madrizquí Key by significantly reducing the live coral cover and number of live

colonies and indirectly aiding an increased algal cover and bioerosion rates by fish grazing. Moreover, the high loss of tissue may significantly reduce the reproductive output of important coral species when live tissue areas in different colonies are reduced below the minimum reproductive size (Petes et al. 2003; Weil 2004).

Substantial work is still needed to understand how the frequency of WP-II epizootic events might affect the reef communities in a mid-to-long term basis, and to understand what set of environmental conditions might be triggering the outbreaks in the different localities around the Caribbean. This disease has a particular epidemiological behavior: suddenly increasing its incidence and prevalence, killing a wide range of different hosts and then, disappearing from the reef with only few infected individuals remaining. This behavior has been reported for several other infectious diseases in wildlife populations (Keeling 2000). Pathogens rapidly killing their hosts (i.e., high virulence) exhibit high contagious rates in order to persist within the host populations (Dobson and Hudson 1998). In colonies affected by WP-II, the mortality rate of living tissue ranges from 2 to 3 cm/day (Richardson et al. 1998a), which, depending on size of the colony, highly reduces the probability of survival once it is infected. Results from this study showed that WP-II removed almost 4 m of living tissue in 12 months in 15 infected colonies of major reef builders, thus significantly reducing live coral cover with the consequent increase in algae cover. An increase grazing pressure was noticeable as the number of colonies with bite marks increased in 2001. The coral mortality event and intense fish and urchin grazing on dead coral skeletons at the site have opened available space for coral recruitment (Edmunds 2000).

Because of its location in the Archipelago (i.e., close to Gran Roque island), the Madrizquí coral reef might be subjected to some human-induced stress. Many localities that constantly receive high numbers of tourists may be exposed to higher sources of pathogens. Recently, many problems of sewage contamination have been observed in Gran Roque Island, and there has been a fast population growth and excessive urbanization over the past decade. Even though the relatively new pressure of population overgrowth in El Gran Roque might be linked to the epizootic event, the factors responsible for triggering the WP-II outbreak still remain unclear and more research is needed to identify the causative agents to this and other outbreaks in the Caribbean. Due to its high virulence and wide host range; the cumulative impact (coral tissue mortality) of frequent WP-II events in many reef localities throughout the Caribbean over the years might end up having a more significant ecological impact than the white band event in the early 1980's.

Acknowledgments.—The authors would like to thank two anonymous reviewers for their suggestions and comments to improve this manuscript, the Fundación Científica Los Roques for logistical support and Pablo Mata and "Moncho" for their valuable help during the field campaigns.

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