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Temporal variability and impact of coral diseases and bleaching in La Parguera, Puerto Rico from 2003-2007

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Abstract.—Most coral reefs in the wider Caribbean have been showing alarming signs of decline in recent years. Coral diseases and bleaching were monitored seasonally from 2003 to 2007 using the modified CARICOMP protocol with a stratified design to assess spatial and temporal variability in community level coral disease prevalence (proportion of affected colonies) at six reefs along an inshore-offshore gradient in La Parguera, southwest coast of Puerto Rico. Temperature loggers were deployed in each reef to assess co-variation between disease prevalence and virulence, and temperature. Virulence was assessed in *Montastraea faveolata* by tagging colonies, marking diseased edges, photographing and following them through time. Overall, all major reef-building species and other common reef groups were variously affected by disease and bleaching. Eleven diseases affected up to 42 species of scleractinian corals, 5 octocorals, 2 hydrocorals, 3 zoanthids, 2 sponges and 2 crustose coralline algae. Bleaching affected 52 species of corals, 22 octocorals, 3 hydrocorals, 2 zoanthids and 3 sponges. The prevalence of the diseases compromised coral health and varied significantly between seasons within years as well as among reefs within years and across years for each reef. White plague, yellow band and bleaching were the most prevalent and damaging diseases. Three white plague, two yellow band outbreaks and three bleaching events of different intensities were observed during the five years of study. Yellow band disease increased in prevalence (from 4 % to 30%) and virulence (0.8 ± 0.2 to 3.9 ± 1.4 cm/month) over 6 years, becoming one of the most important agents of coral mortality. Higher disease prevalence was consistently found at the mid-shelf and shelf-edge reefs than in reefs closest to the shoreline. The combination of white plague disease outbreaks, the intensive bleaching of 2005 and the chronic yellow band caused an average 53 % of live coral tissue loss in four years, the highest coral mortalities ever recorded in southwest Puerto Rico.

Keywords.—Coral reef diseases, prevalence, spatial/temporal variability, Puerto Rico, Caribbean

Introduction

Increasingly stressful conditions from natural and anthropogenic factors are producing significant declines in coral reef communities around the World. Bleaching and biotic diseases are two natural occurrences that have become major factors in this process. Two Caribbean-wide epizootic events in the early 1980's produced massive mortalities of two important reef-building coral species, *Acropora palmata* and *A. cervicornis*, and a keystone echinoid, the black sea urchin *Diadema antillarum*. The potential of disease outbreaks to significantly change coral reefs was shown by these massive mortalities. In a relatively short time and over a wide geographic region, populations of these species suffered up to 95% mortality (Gladfelter 1982; Lessios et al. 1984, Carpenter 1990a,b) producing a cascade of significant ecological changes in the dynamics, function and structure of coral reefs at local and geographic scales (Hughes 1994, Harvell et al. 1999, Aronson & Precht 2001, Bruckner et al. 2002, Lirman et al. 2002, Weil et al. 2003, 2004).

Since these events, diseases affecting corals have increased in number, prevalence and spatial distribution in the Caribbean and other reef areas of the World (Richardson 1998; Harvell et al. 1999; Weil et al. 2002; Rosemberg and Loya 2004; Aronson and Precht 2006; Weil et al. 2006), prompting speculation that they result from global and local environmental stresses such as ocean warming, acidification, nutrient enrichment, deforestation and other local anthropogenic factors (Epstein et al. 1998; Carey 2000; Harvell et al. 2004, 2007, 2009; Weil 2004; Bruno et al. 2003, 2007; Hoegh-Guldberg et al. 2007).

Coral reef disease research in Puerto Rico in the mid 1990's and has yielded limited but important information on the number, distribution, prevalence and impact of the most common diseases at a few reef localities. The best studied areas include La Parguera on the southwest coast, Culebra on the east coast, where the best developed and extensive reef complexes in Puerto Rico are found, and reefs in the offshore islands, Mona and Desecheo off the west coast (Bruckner 1999; Bruckner and Bruckner 1997, 2006; Bruckner and Hill 2009; Weil et al, 2002, 2003; Weil 2004; Ballantine et al. 2005; Hernandez-Delgado et al 2006; Weil and Croquer 2009; Croquer and Weil 2009a,b; Toledo-Hernández et al. 2007; 2009; Weil et al. 2009; Flynn and Weil this issue).

Most diseases reported for the Caribbean are present in Puerto Rico and frequent epizootics of white band disease (WBD), white plague disease (WPD), Caribbean yellow band disease (YBD), white patches (= white pox, patchy necrosis) (WPA) and bleaching (BLE) have caused significant losses in coral cover (biomass and photosynthetically active surface area) in most reef areas around the island. These epizootic events usually occur during the Summer-Fall season and decline as temperature drops. In the late 1970's, extensive thickets of elkhorn coral *Acropora palmata* were present in 40 % of 35 reef localities surveyed around the island of Puerto Rico. Another 20 % of these reefs had dense patches and abundant colonies of staghorn coral *A. cervicornis.* However, qualitative surveys of more than 100 coastal and offshore localities around the island in the last 25 years indicate a significant decline in acroporid populations in most localities. Small, but short lived recovery, has occurred at some reef sites (Weil et al 2003).

A similar situation seems to have occurred with the *Montastraea* species complex during the last 10 years, with populations declining in many reefs due to WPD, YBD and BLE. Furthermore, other important biological groups of the coral reef community have shown signs of disease and have been affected by bleaching, including octocorals, hydrocorals (Weil et al, 2002, Weil 2004; Toledo-Hernández et al. 2007; 2009; Prada et al. 2009; Flynn and Weil 2009), crustose coralline algae (Weil 2004; Ballantine et al. 2005); zoanthids and sponges (Weil 2004, Weil et al. 2006).

The goals of this study were to identify the major disease problems affecting corals in reefs off La Parguera, assess the spatial, seasonal and temporal variability of biotic coral diseases and bleaching over five years and estimate their impact on coral populations and the reef community along an inshore-offshore gradient in La Parguera Natural Reserve, southwest coast of Puerto Rico.

Materials and Methods

Study area

The study was conducted in the La Parguera Natural Reserve, an extensive coral reef ecosystem located on the southwest coast of Puerto Rico. La Parguera is one of the driest areas along the southwestern coast of Puerto Rico, with an insular shelf that extends 8-10 km offshore (Morelock et al. 1977, Garcia et al. 1998). Two distinct lines of emergent reefs run parallel to the coastline dividing the insular shelf into inner, middle, and outer shelf areas (Morelock *et al*. 1977). Reefs located in the inner and middle shelf are typical fringing reef structures bordering mangrove keys with shallow areas of *Acropora* rubble and small crustose and massive coral colonies on the exposed reefs, with octocoral dominated platforms, and *Montastraea*-dominated fore-reefs and slopes extending to 15-20 m depth. Reefs in the outer shelf occur below 18m, with conspicuous spur and groove formations that extend over the platform edge. Reef localities included two inner fringing reefs close to shore [Pelotas (17° 57.442′ N; 67° 04.176′ W) and Enrique (17° 56.658′ N; 67° 02.213′ W)], two fringing reefs at the mid-shelf zone [Media Luna (17° 56.093′ N; 67° 02.931′ W) and Turrumote (17° 56.097′ N; 67° 01.130′ W)], and two shelf-edge spur and groove reefs [Buoy (17° 53.380′ N; 66° 59.090′ W) and Weinberg (17° 53.429′ N; 66° 59.320′ W)] (Fig. 1).

Methods

Data and observations on coral diseases have been collected in several reefs since the late 1990's. This effort was intensified in 2003 with the systematic sampling of nine reefs in La Parguera and other reefs of the southwest coast. Results from disease and bleaching data and observation collected in six of these reefs along an inshore-offshore gradient in La Parguera are presented here. The modified CARICOMP protocol (Weil et al. 2002) was used to assess the spatial and temporal variability in the number, distribution and prevalence of coral diseases and bleaching every summer-fall and winter-spring season from 2003 to 2007 along four permanent 10 x 2 m (20 m^2) band transects haphazardly marked at each of four depth intervals (reef habitats) (0-3, 4-8; 9-13 and >15 m). Sampling was done at each of the two inshore fringing reefs, Enrique and Pelotas (< 1 km from shore), and two mid-shelf fringing reefs, Turrumote and Media Luna (ca. 2 km from the shore). Sixteen permanent transects were surveyed at the two, shelf-edge, spur-and- groove reefs, Weinberg and the Buoy (7.2 km offshore) (Fig. 1, Table 1). Within each band transect, all coral, octocoral, zoanthid and sponge colonies were counted and checked for presence of diseases, bleaching, and predation/physical injuries. If no disease or predation signs were observed and the colony appeared healthy with normal coloration, it was considered to be healthy. Only scleractinian corals and a few octocorals and other reef invertebrates were identified to species level. The disease and acronym nomenclature used in the text and figures follows that of Raymundo et al. (2008) and Weil and Hooten (2008).

To assess virulence (rate of tissue loss) of the two most damaging diseases (WPD and YBD), over two hundred colonies of *Montastraea faveolata*, *M. franksi*, *Dendrogyra cylindrus*, *Diploria strigosa*, *D. labyrinthiformis*, and *Colpophyllia natans* were tagged and photographed, and nails carefully hammered at the disease-healthy tissue boundary and colonies photographed. Distance from

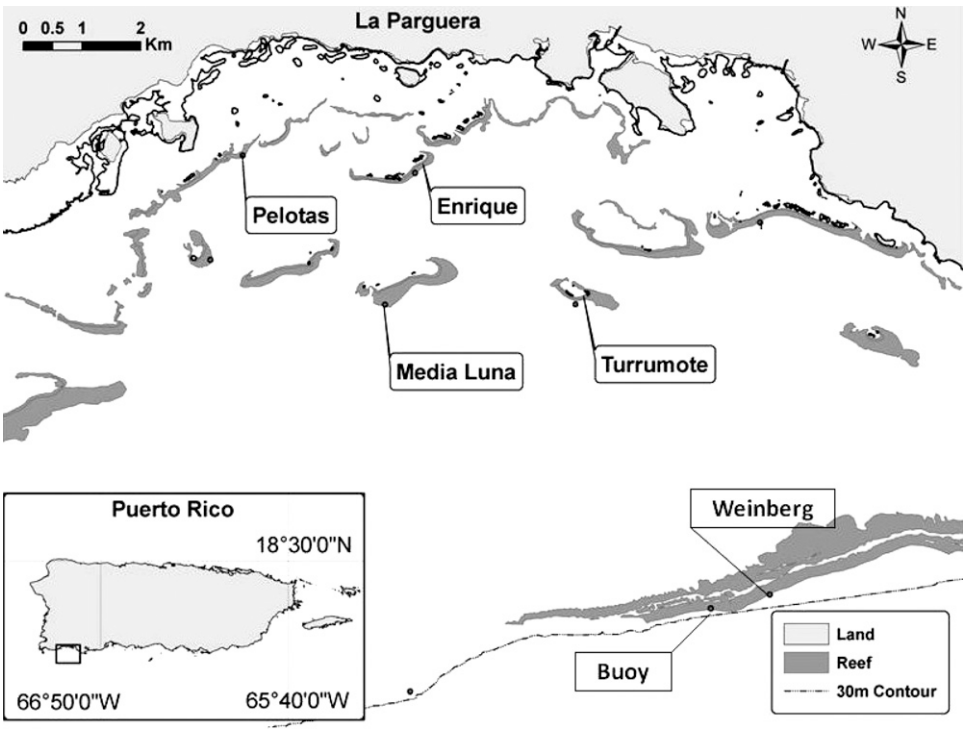

FIG. 1. Map of La Parguera showing the relative location of the six reefs sampled from 2003 to 2007.

Table 1. Geographic coordinates and information of the reef localities surveyed.

Reef	Latitud	Longitud	Type	Location	Depth range(m)
Enrique	17° 56.658	67° 02.213	Fringing	Inshore	1 - 17
Pelotas	17° 57.442	67° 04.176	Fringing	Inshore	1 - 16
Turrumote	17° 56.097	67° 01.130	Fringing	Mid-shelf	2 - 20
Media Luna	17° 56.093	67° 02.931	Fringing	Mid-shelf	3 - 20
Weimberg	17° 53.429	66° 56.320	Bank	Outer-shelf	18 - 25
Buoy	17° 53.328	66° 59.090	Bank	Outer-shelf	18 - 25

the nail to the new diseased tissue edge was measured (to the nearest mm) at weekly-monthly intervals and the average disease advance rate (= mortality rate of coral tissue = virulence) was calculated.

To assess potential co-variation between disease prevalence and outbreaks, and virulence with water temperature, Hobo Pro v.1 and v.2 temperature loggers were deployed at each reef off La Parguera in 2003. One logger was deployed at each of the shallow (1-2m) and deep (15-20m) habitats of the two inner- and two mid-shelf reefs. One logger was deployed at each of the two shelf-edge spur and groove reefs. Surface water temperatures (< 1m) have been measured weekly since 1997 in two reefs off La Parguera (Media Luna and the back, sea-grass area of Enrique).

Disease data was pooled and average prevalence calculated for each transect. Seasonal and yearly prevalence means were then estimated for total disease (all diseases pooled together), total bleaching (all bleaching pooled together), and the most common and damaging biotic diseases [YBD, WPD, dark spots disease (DSD), white band disease (WBD)] for each reef. Percent data was arcsin transformed and One way parametric and non-parametric ANOVAS were used to test the hypotheses that disease and bleaching prevalence was similar across all reefs during each particular year. Repeated measures ANOVA was used to test the hypotheses that disease and bleaching prevalence did not vary across years. A T-Test was used to test the hypothesis that disease and bleaching prevalence were similar between the winter-spring and summer-fall seasons of each year. Then, data from the two reefs in each of the inner- and mid-shelf reefs and the shelf edge reef was pooled and one way ANOVA was used to test the hypothesis that disease and bleaching prevalence did not vary across the inshore-offshore gradient. Pearson Correlation Coeficient was used to check co-variation between seasonal mean disease prevalence for major diseases and average monthly temperatures.

Results and Discussion

Disease infections and species affected.

All common coral reef diseases described for the wider Caribbean (Green and Bruckner 2000; Weil 2004, Sutherland et al. 2004) were observed in La Parguera. Overall, 38 species of scleractinian corals were susceptible to nine different biotic diseases (for which pathogens have been presumably identified), and several other affections (disease-like signs for which no pathogens have been determined) grouped in different categories (i.e. as compromised health) (Figs. 2A and 2B, Table 2) (Weil and Hooten 2008). Hydrocorals were suscep tible to three diseases, octocorals to six, zoanthids to two, sponges to four and crustose coralline algae (CCA) to two. Forty two scleractinian species, four hydrocoral, twenty four octocoral, two sponges and two zoanthid species were susceptible to bleaching (defined as an abiotic disease produced by environmental factors) events in 2003 and 2005 (Table 2).

Two diseases constitute new reports for this area, Caribbean ciliate infection (CCI) produced by halofoliculinid ciliates (*Halofoliculina* sp) (Croquer et al. 2006a, b), and *Gorgonia* purple spots (GPS), caused by an unidentified protozoan (Labyrinthulomycote) (Weil and Hooten 2008; Harvell person. comm.) (Table 2) (Figs. 2A and 2B).

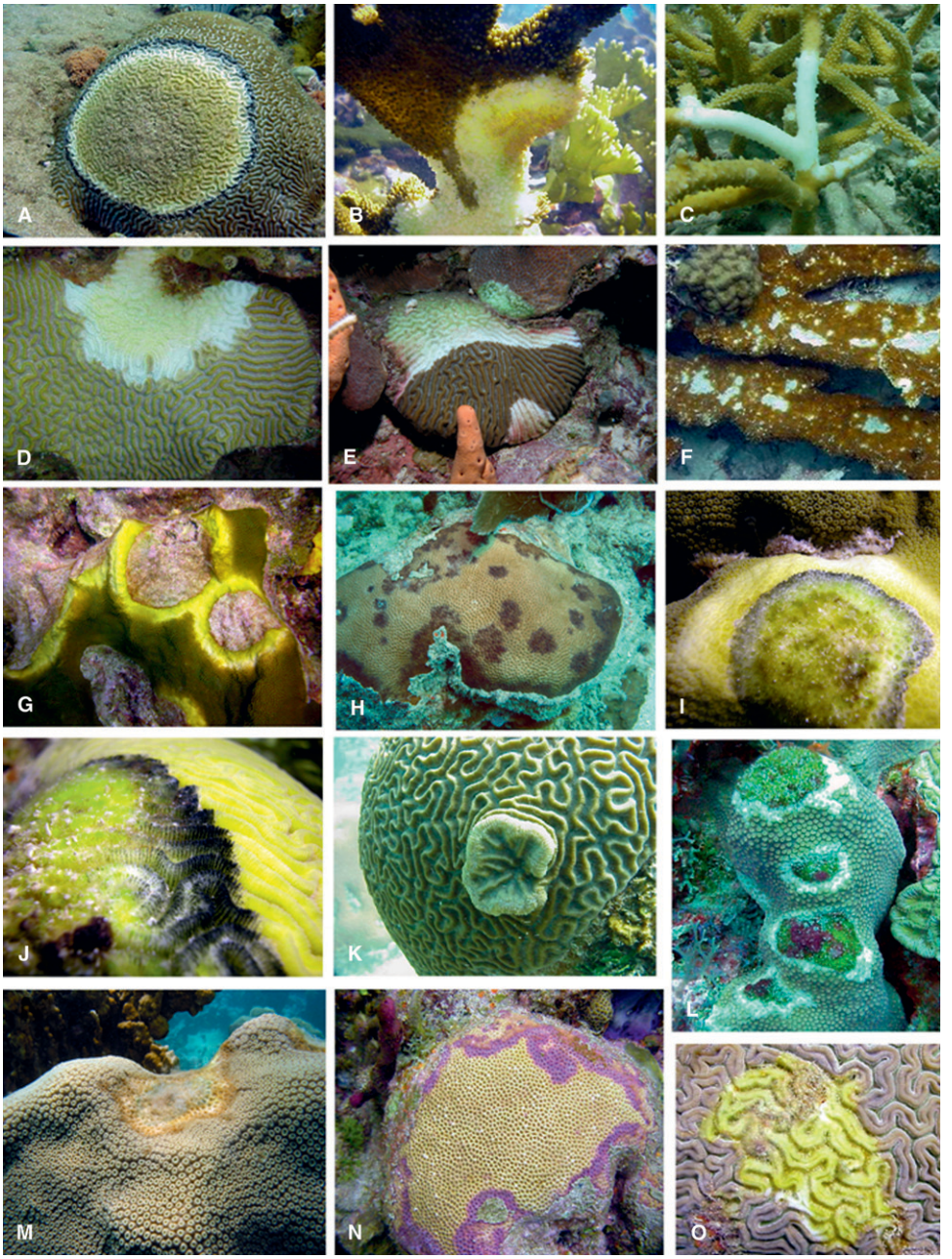

FIG. 2A. Common diseases affecting scleractinian corals in La Parguera including black band disease (A), white band disease (B, C), white plague disease (D, E), white patches (F), yellow band disease (G), dark spots disease (H), Caribbean ciliate infection (I, J), growth anomalies (K), white syndrome (L), and several compromised health problems (M, N, O).

The ciliate infections were first observed in Venezuela as a secondary opportunistic infection in few species of corals, further observations have brought the number of CCI susceptible coral species to 22 in several localities around the Caribbean (Cróquer et al 2006b; Weil et al. 2006; Rodriguez et al. 2009, Cróquer and Weil 2009). Only 9 coral

FIG. 2B. Recent new diseases affecting crustose coralline algae include coralline white band syndrome (A) and Caribbean coralline lethal orange disease (CCLOD) (B). Diseases affecting octocorals include *Gorgonia* purple spots (C), compromise health problems (D, E and H), red band disease (F, G), growth anomalies (K) and aspergillosis (R). Bleached *Pseuplexaura nutans* and *Acropora palmata* (I, J). Other organisms with disease signs were the zoanthid *Palythoa caribbaeorum* (L), the hydrocoral *Millepora complanata* (M) and sponges (O, P and Q).

species in Puerto Rico have been observed with CCI. In La Parguera, several colonies of *Montastraea* and *Diploria* were observed with CCI during our surveys in 2006 and 2007 following YBD and WPD infections. The ciliates eventually disappeared from the infected colonies. Gorgonia purple spots (GPS) is characterized by small purple spots

TABLE 2. Most common observed biotic and abiotic diseases affecting sessile coral reef organisms in La Parguera, their acronyms, putative pathogen (s) or causative agent and number of susceptible taxa of scleractinian corals (COR), octocoral (OCT), sponges (SPO), crustose coralline algae (CCA) and zoanthids (ZOA). * = Koch's postulates fulfilled.

Biotic Diseases	Acronym[1]	Pathogen	COR	HYD	OCT	SPO	CCA	ZOA
Black band disease*	BBD	*P. coralliticum*, *Desulfovibrio, Beggiatoa* sp	11	-	5	-	-	-
Red band disease	RBD	*Oscillatoria* sp. and other cianobacteria	4	-	3	-	-	-
White band disease*[1]	WBD	*Vibrio harveyi/charchariae*	2	-	-	-	-	-
White plague disease*[2]	WPD	*Aurantimonas coralicida*	22	2	-	-	-	-
White patches*[3]	WPA	*Serratia marsences* and others?	6	-	-	-	-	-
Caribbean yellow band disease[5]	YBD	*Vibrio* spp	7	-	-	-	-	-
Dark spots disease[5]	DSD	*Vibrio* sp?	10	-	-	-	-	-
Caribbean ciliate infection	CCI	*Halofoliculina* sp	4	-	-	-	-	-
Coral growth anomalies[4]	CGA	*A. endozoica* (algae) and other causes	5	-	-	-	-	-
Caribbean white syndromes	CWS	???	7	1	-	-	-	-
Compromised health corals	CHC	???	14	-	-	-	-	-
Aspergillosis*	ASP	*Aspergillus sidowii*	-	-	3	-	-	-
Gorgonia purple spots	GPS	Unknown Laberyrinthulomycote	-	-	1	-	-	-
Octocoral growth anomalies	OGA	?	-	-	6	-	-	-
Compromised health octocoral	CHO	?	-	-	8	-	-	-
Compromised health hydrocoral	CHH	?	-	2	-	-	-	-
Compromised health zoanthids	CHZ	?	-	-	-	-	-	2
Compromised health sponges	CHS	?	-	-	-	10	-	-
Coralline white band syndrome[5]	CWBS	?	-	-	-	-	2	-
Caribbean lethal orange disease	CCLOD	?	-	-	-	-	1	-
Abiotic Diseases								
Bleaching	BL	Temperature, UV light, sedimentation	42	4	24	2	-	2

[1]Includes white band types I and II.
[2]Includes white plague types I, II and III.
[3]Includes patchy necrosis and white pox.
[4]include hyperplasia and neoplasias. Samples currently under investigation to identify putative pathogens.
[5]Nomenclature and acronysms from Weil and Hooten (2008) and Raymundo et al. (2008).

on the blade of the sea fan *G. ventalina* (Fig. 2B-C) and was previously observed in Mexico and the Florida keys (Harvell pers. comm., Weil unpubl.). It was first observed at two shallow reefs off La Parguera in 2007, but has now spread to several other reef localities where prevalence has been increasing in recent years (Weil unpubl.).

Two recently reported diseases affecting crustose coralline algae (CCA), crustose coralline white band syndrome (CWBS) (Weil 2004; Ballantine et al. 2005) and Caribbean coralline lethal orange disease (CCLOD), a new report for the region (Weil and Hooten 2008, Weil unpubl.), were observed in this study. CWBS was first observed in the summer of 2001 in Grenada where shallow crustose coralline algae (CCA) showed concentric rings of dead tissue bordered by a conspicuous white

band (Weil 2004) (Fig. 2B-A). The condition was later found in deep reef habitats (18-25m at Weinberg and Buoy reefs off La Parguera) and many other reef localities throughout the Caribbean (Weil and Croquer 2009) affecting large numbers of the CCA *Neogoniolithon accretum.* Further observations indicated that this condition was also common in shallower reef habitats in other coral reefs off La Parguera and the Caribbean (Weil. unpubl.). Preliminary surveys at Weinberg indicated that close to 70% of the *N. accretun* were infected and some of these were nearly 80% dead (Ballantine et al. 2005). Further surveys have showed increasing prevalence of CWBS in CCA populations in many other reefs with significant mortality in CCA (data not shown). Caribbean coralline lethal orange disease (CCLOD) (Fig. 2B-B), has only been observed in a few crusts in deeper reef habitats (Weinberg and the Buoy) in La Parguera, Grand Cayman, and Mexico (Weil unpubl.). The signs are similar to "coralline lethal orange disease" (CLOD) reported in the Indo-Pacific (Littler and Littler 1994, 1995). The causative agent has yet to be determined.

Many of the important reef-building species (*Montastraea, Diploria, Colpophyllia*, *Acropora*, *Siderastrea*, and *Stephanocoenia*) were susceptible to multiple, simultaneous or continuous infections during the course of the study. For example, some colonies of the important reef-building *Montastraea faveolata* showed signs of YBD (Fig. 2A-G), WPD (Fig. 2A-D-E), DSD (Fig. 2A-H) and BLE simultaneously in 2003 and 2005. Other colonies were affected first by bleaching, developed WPD during the late summer and winter of 2005-2006 and when these signs disappeared, the corals were hit by YBD in the summer of 2006. Similar multiple infections have been observed in many other localities around the Caribbean (Weil et al. 2006, Weil and Croquer 2009, Croquer and Weil 2009). These simultaneous or continuous multiple infections significantly increased the rate of tissue mortality (data not shown), killing large colonies in a short time in many reefs, and affecting the reproductive output and fitness of important reef-building species (Weil et al. 2009), which could significantly delay the natural recovery of coral populations.

Spatial and temporal variability in prevalence of total coral diseases

Coral disease distribution and prevalence in La Parguera varied significantly within and across reefs as well as temporally. Similarly to other Caribbean locations, outbreaks of WPD, YBD, DSD, WPA, WBD, and aspergillosis (ASP), and chronic presence of YBD and CWBS were the most common problems affecting a large number of the main coral, octocoral and CCA species (Goreau et al. 1998; Green and Bruckner 2000; Gil-Agudelo and Garzón-Ferreira 2001; Rodríguez-Martínez et al. 2001; Weil et al. 2002; Weil 2004; Borger and Steiner 2005; Croquer and Weil 2009a).

Significant differences (ANOVA and Kruskal-Wallis, $P<0.05$) in the yearly average prevalence of all pooled diseases (= community-level disease prevalence) was found among reefs every year from 2003 to 2007 (Fig. 3). In 2003, coral disease prevalence levels varied between 2.7% (± 0.37) at Media Luna and 10.4% (± 0.83) at the Buoy. Disease prevalence was similar in all four inshore and mid-shelf reefs. The two offshore reefs (Weinberg and Buoy) had similar but significantly higher (K-W, $p<0.05$) coral disease prevalence compared to the other reefs (Fig. 3) due to an outbreak of WPD (see below). A disease outbreak or epizootic (= epidemic) event is considered herein to be when average community and/or population disease prevalence levels reach 10% and/or 20% levels respectively. The outbreak affected mostly *Montastraea* (17.0 ± 6.1%), *Mycetophyllia* (31.5 ±1.1%), *Diploria* (19.8 ±10%) and *Siderastrea* (33.6 ± 8.1%) species producing significant tissue losses. White plague disease signs disappeared during the winter of 2004 and community disease levels decreased significantly (K-W, $p< 0.05$) at the offshore reefs (Fig. 4). In 2004, the average disease prevalence varied between 1.38% (± 0.3) at Enrique and 11.02% (± 1.41) at Turrumote, driving the decrease in the mean pooled disease prevalence (all reefs together) for the area from 5.41 ± 3.43% in 2003 to 3.88 ± 3.64% in 2004. No significant changes in

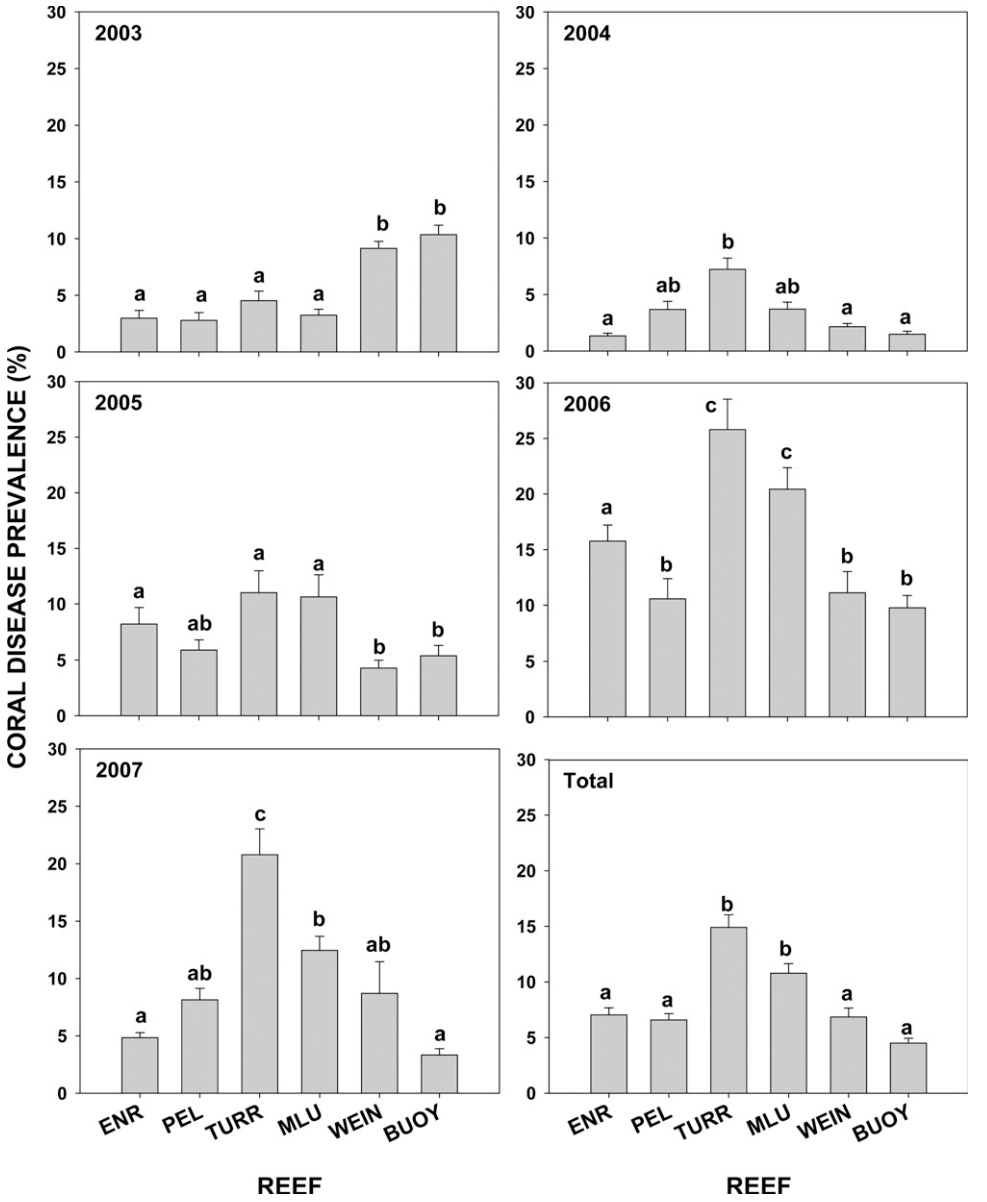

FIG. 3. Spatial variability of the mean (SE) prevalence of pooled coral diseases (all diseases recorded) in each of the six reefs for each year from 2003 to 2007. Different letters above columns indicate significant difference between that reef compared to other reefs.

prevalence was observed in either of the inshore and mid-shelf reefs (Figs. 3 and 4). The increase at Turrumote was due to an increase in prevalence of YBD, a disease first observed affecting few colonies of *Montastraea faveolata* during 1998-99 (Weil et al. 2002) (see below). With the exception of the Buoy and Weinberg in 2003 and Turrumote in 2004, the average coral disease prevalence was generally low and similar to those reported for other Caribbean reef localities (Weil et al. 2002; Borger and Steiner 2005; Weil and Croquer 2009).

In 2005, all reefs showed an increase in prevalence with inshore and mid-shelf reefs showing significantly higher levels

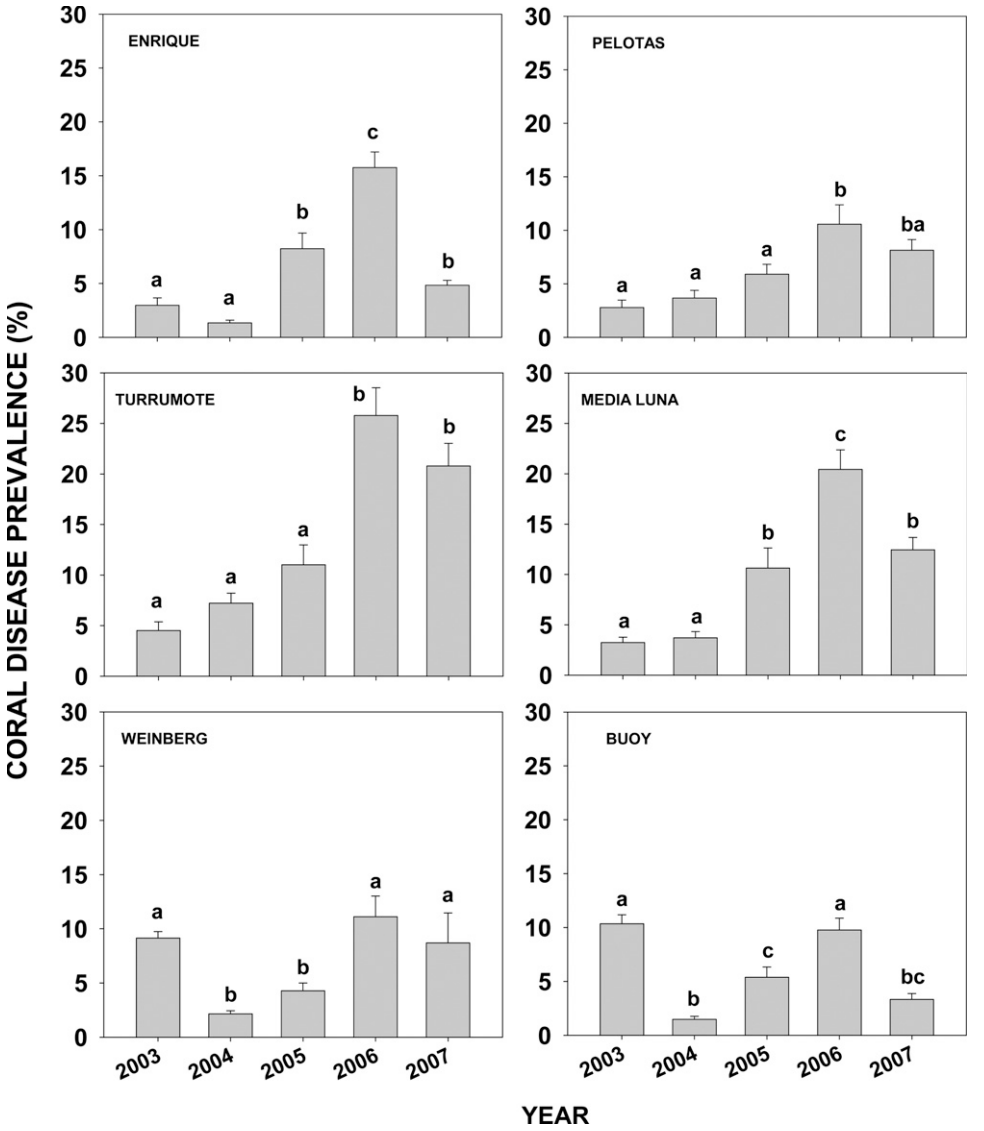

FIG. 4. Temporal variability in the mean (SE) prevalence of pooled coral diseases (all diseases recorded) for each reef between from 2003 to 2007. Different letters above columns indicate significant difference between years.

of disease compared to the two shelf-edge reefs (Figs. 3 and 4). Average prevalence for all reefs increased to 6.15 ± 1.3% and varied between 4.28% (± 0.6) at Weinberg to 7.82% (± 1.7%) at Enrique. Significant increases (K-W, $p<0.5$) in prevalence occurred at Enrique, Turrumote, Media Luna and the Buoy (Fig. 4). The most severe bleaching event recorded in the eastern Caribbean (McClanahan et al. 2009; Croquer and Weil 2009b) occurred during this year. This event was followed by the third and most intense WPD outbreak in the later months of the year which affected mostly mid-shelf and shelf-edge reefs (see below). All these outbreaks were correlated with higher than normal winter and summer water temperatures (Fig. 5). The WPD outbreak

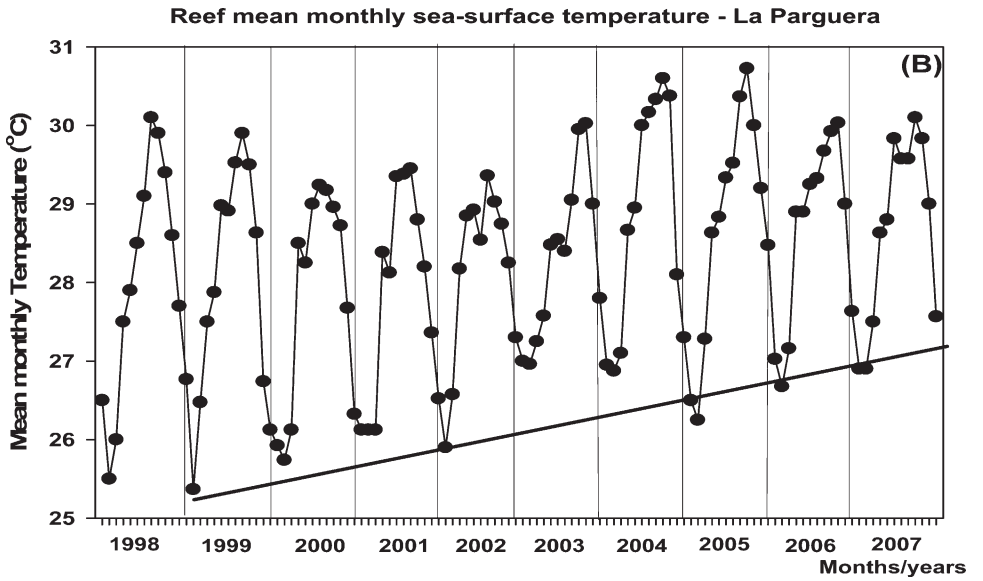

FIG. 5. Yearly variability in surface water temperature (°C) for la Parguera area. Line shows increasing trend in average winter water temperatures in the last 9 years.

continued through February-March of 2006 probably aided by the higher than normal water temperatures driving a significant increase (T-test, $P<0.01$) in mean coral disease prevalence from 6.15% in 2005 to 15.66 ± 6.34% in 2006, the highest mean disease prevalence recorded during the five years of this study.

The mid-shelf reefs showed significantly higher (K-W, $p<0.05$) disease prevalence than the inshore and shelf-edge reefs (Fig. 3), with all six reefs showing significant increases in prevalence from 2005 to 2006 (Fig. 4) due to the WPD outbreak and a new outbreak of YBD at Media Luna and Turrumote in the summer of 2006 (See below). Turrumote was the most affected reef (disease prevalence of 33.8 % during the summer) mostly by YBD. This disease has been a chronic problem at this reef since 2003, with seasonal outbreaks and high prevalence in *M. faveolata* all year long (see below). Media Luna had the next highest mean disease prevalence in 2006 driven also by the ongoing YBD epizootic event which intensified after the extended bleaching of 2005, and some remaining WPD infected colonies. Intensive bleaching followed by an outbreak of WPD was also reported for the Virgin Islands (Miller at al. 2006; 2009; Rogers et al. 2008) for 2005-2006 with high mortalities of coral tissue in *M. annularis* and *M. faveolata* and other important reef-builders.

Enrique, Media Luna and the Buoy reefs showed significant decreases (K-W, $p<0.5$) in mean disease prevalence in 2007 compared to 2006. The other three reefs also showed a decrease in prevalence but they were not statistically significant (Fig. 4). Turrumote had significantly higher mean disease prevalence compared to the other reefs due to the high prevalence of YBD during 2007. Even though summer temperatures were back to normal levels, mean winter water temperatures were still above normal in 2007-2008 (Fig. 5). Enrique and the Buoy showed the lowest disease prevalence (Fig. 3) in 2007 compared to the other reefs.

Seasonality and impact of white plague disease, yellow band disease and total disease.

Seasonal variability was readily observable in the field for WPD, YBD and other common diseases. Significant differences (T-test, $p<0.05$) in mean prevalence of WPD, YBD

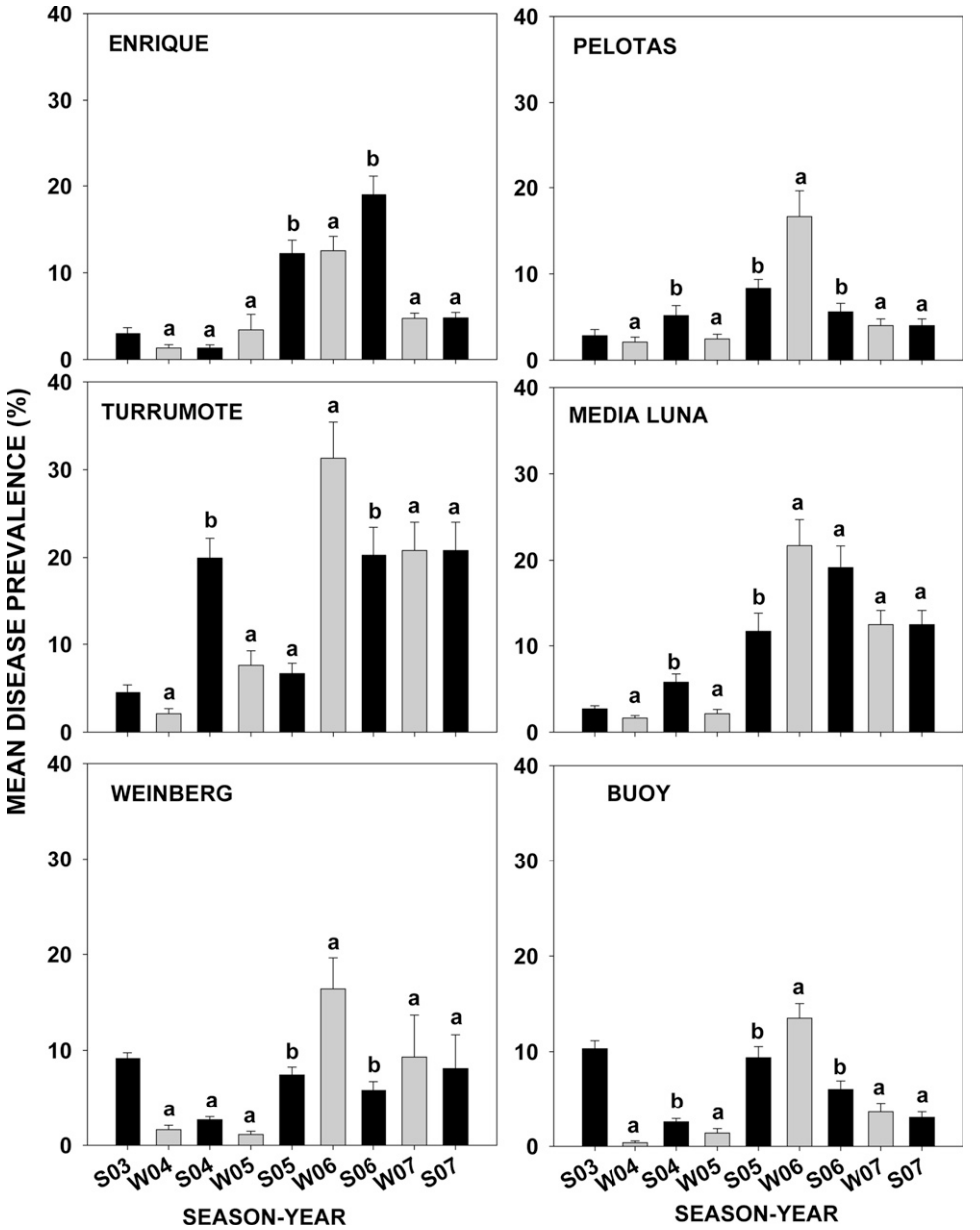

FIG. 6. Seasonal variability in the mean prevalence (SE) of pooled coral diseases (A), mean prevalence (SE) of white plague disease (B) and mean prevalence (SE) of yellow band disease for each reef from the summer-fall of 2003 to the summer-fall of 2007. Letters above columns indicate significant differences between seasons for each year only.

and all pooled diseases between the winter-spring and the summer-fall seasons were found for every year for each reef. However, there were years when no differences were found between seasonal mean disease prevalence (Figs. 6A-6C).

Average total coral (pooled diseases) disease prevalence was in general significantly higher (T-test, $p<0.05$) in the summer months for Pelotas, Media Luna, Turrumote and the Buoy compared with winter prevalence values in 2004. No difference was

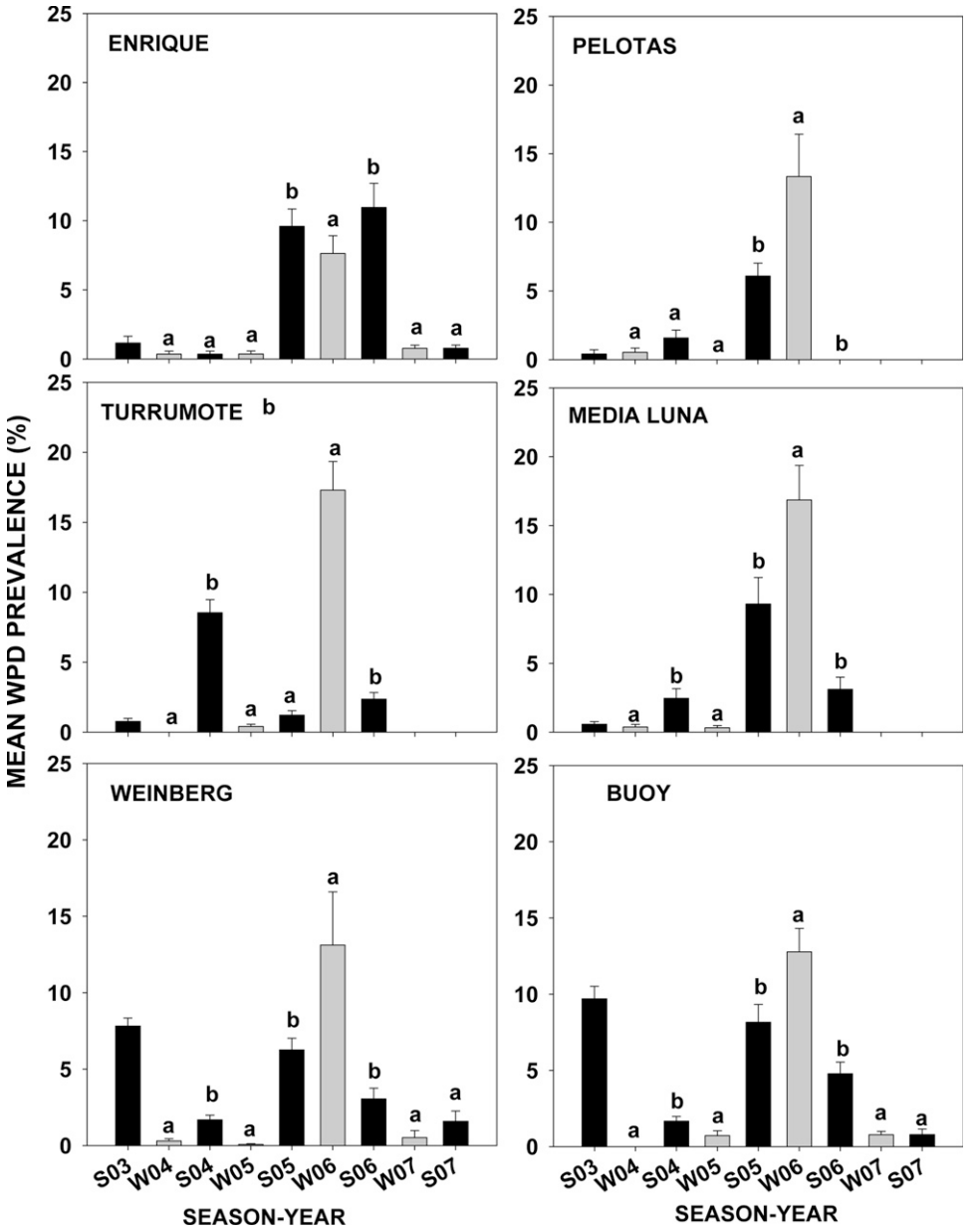

FIG. 6B. Continued.

observed between Enrique and Weinberg. Similar patterns were found for all reefs but Turrumote in 2005 (Fig. 6A). This was mostly due to the chronic presence of YBD in *M. faveolata* at this reef, which maintained total disease prevalence similar throughout the year unless there was a disease outbreak during the summer-fall. The highest mean prevalence of coral disease occurred in the winter-spring of 2006, when the peak of the WPD outbreak occurred and YBD was developing in some colonies affecting many coral species at all reefs but Enrique, which showed a higher disease prevalence

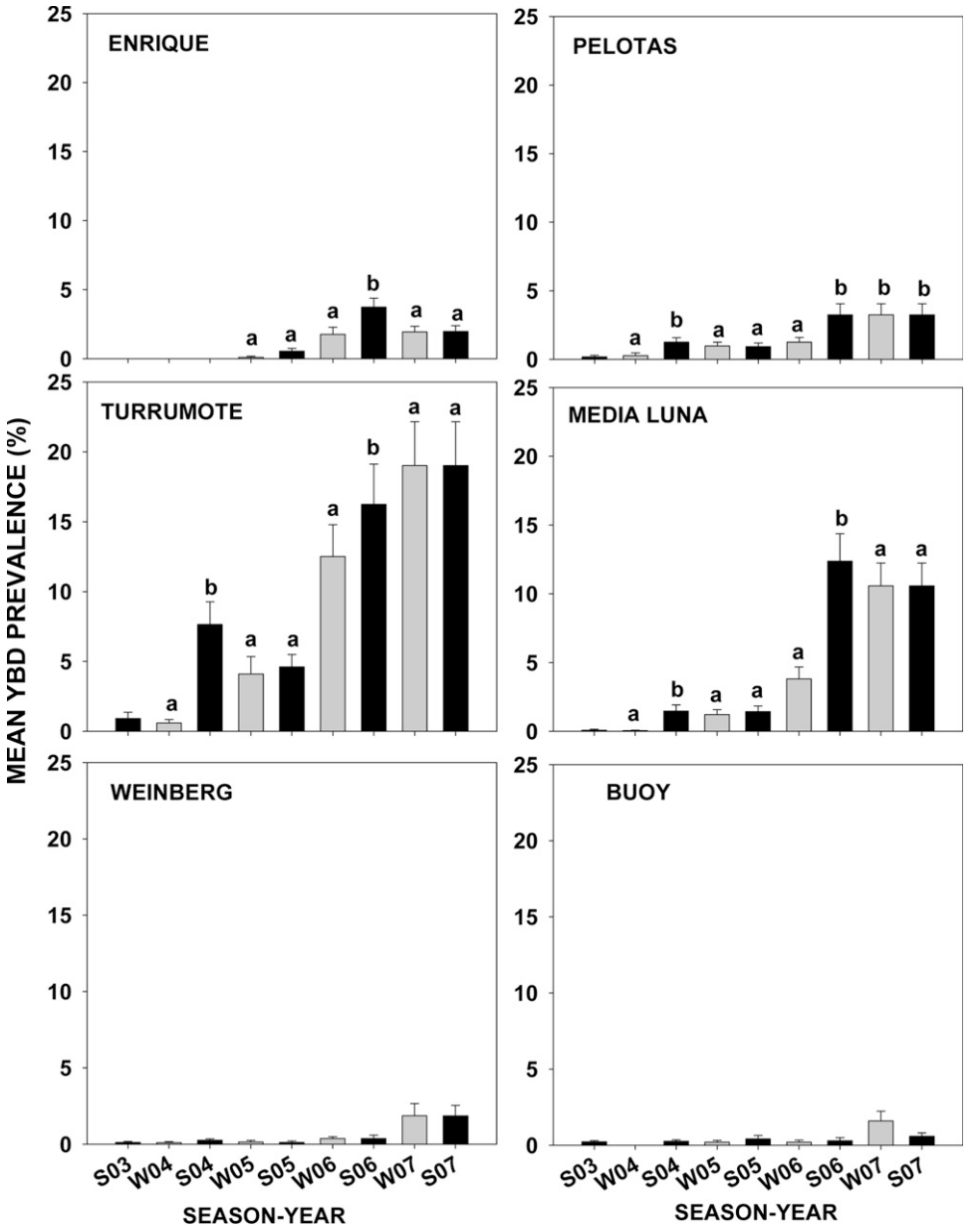

FIG. 6C. Continued.

during the summer of 2006. Mean disease prevalence decreased at all reefs with the exception of Turrumote and Weinberg in 2007 and prevalence levels were similar during the summer and winter seasons. High prevalence of YBD in Turrumote and presence of WPD infected colonies at Weinberg kept the average prevalence of pooled disease at similar levels to those of 2006 (Fig. 6A).

White plague disease

White plague disease was first reported from La Parguera in 1995, and has since been observed throughout Puerto Rico and

its offshore islands (Bruckner and Bruckner 1997, 2006; Weil et al. 2002; Weil 2004; Hernandez-Delgado et al. 2006). Over 22 local scleractinian species seem to be susceptible to this disease (Table 2) compared to over 40 coral species that have been observed with WPD signs Caribbean-wide (Weil 2004; Sutherland et al. 2004). It is considered one of the most damaging to coral populations because of its frequent outbreaks, wide host range, and high virulence (Richardson 1998; Richardson et al. 1998a,b; Richardson and Voss, 2005; Weil et al. 2006; Miller et al. 2003, 2006; Croquer and Weil 2009). The disease has been highly seasonal, developing during the high summer-fall temperatures and disappearing during the winter, when temperatures dropped.

Many species were affected by the WPD outbreaks observed in La Parguera. The disease showed significantly higher (K-W, $p<0.05$, T-test, $p<0.05$) mean prevalence during the summer-fall of 2005 and the winter-spring of 2006 compared to other seasons and years (Fig. 6B). The first significant outbreak in La Parguera occurred in late August and September of 2003, when warm waters (29.5 °C) were prevalent at intermediate-deeper habitats (>10m). The species most affected were *Montastraea faveolata* (20.7 ± 2.8%), *M. franksi* (11.8 ± 1.24%), *M. cavernosa* (3.6 ± 1.2%), *Diploria strigosa* (13.1 ± 11.8%), *D. labyrinthiformis* (16.7 ± 5.1%), and *Colpophyllia natans* (46 ± 9.7%). The average WPD virulence varied across species from 0.2 to 3.1 cm/day killing small colonies in less than a week and a few medium size colonies, mostly *D. strigosa*, *S. siderea*, *C. natans*, and *M. faveolata*, over the two months the event lasted. Rates of lesion advance were similar to those reported in other regions of the Caribbean (Richardson et al. 1998a; Weil 2004; Miller et al. 2006). Disease signs disappeared after water temperatures declined in early 2004.

The 2005-2006 WPD outbreak affected as many as 22 coral species with most reefs showing significantly higher (K-W, $p<0.05$) WPD mean prevalence in the winter-spring of 2006 (from 7.6 ± 1.29% at Enrique to 17.4 ± 2.05% at Turrumote) compared to the summer-fall of 2005 (from 1.23 ± 0.31% at Turrumote to 9.59 ± 1.21% at Enrique) (Fig. 6B). This seasonality pattern was different compared to the 2003 event when higher WPD prevalence was found in late early-mid fall. The outbreak produced high tissue and colony mortality in many of the most important reef-building taxa (*Montastraea*, *Diploria*, *Siderastrea*, *Colpophyllia*, *Stephanocoenia*, etc.), and other common species (*Mycetophyllia* spp, *Meandrina* spp. *Eusmilia fastigiata, Agaricia* spp. and *Madracis* spp). in La Parguera and other reefs off the west coast. On the east coast, WPD caused a net 20-60% decline in living coral cover within a period of approximately six months (Hernandez-Delgado et al. 2006; Garcia et al. 2008). Nearly 100% of the colonies of important reef-building coral species suffered significant partial colony mortality at Culebra Island (Ballantine et al. 2008, Croquer and Weil 2009a, b, Hernandez-Delgado et al., in press).

Colonies were already stressed by a long period (14 degree heating weeks= # of weeks with water temperatures at least 1°C above average) of high water temperatures (and bleaching) which could have increased colony susceptibility and facilitated the WPD-like epizootic to develop. Contrary to the characteristic seasonality of this disease, the 2005 outbreak lasted longer, with many colonies showing disease signs until March-April of 2006 in La Parguera. Similar observations were reported for the Virgin Islands (Miller et al. 2006, 2009; Rogers et al. 2008, Rothenberger et al. 2008). The unusually active WPD during the winter months was probably aided by the unseasonal high water temperatures recorded for winter-spring of 2006 (Fig. 5). No WPD outbreaks have been observed in La Parguera since 2006 and signs of the disease have only been observed in a few colonies of *M. cavernosa*, *Montastraea franksi* and *M. meandrites* at the shelf-edge reefs.

The possibility of other pathogenic bacteria eliciting similar signs to those of WPD infected with *Aurantimonas coralicida* (Richardson et al. 1998a, Denner et al. 2003) was recently proposed after analyses of several diseased tissue samples collected from *M. faveolata* colonies showing the typical signs of WPD in Turrumote in

2005, failed to find *A. coralicida* (Sunagawa et al. 2009). Results of this study support the idea that other pathogenic bacteria could have been the cause of the 2005-06 WPD outbreak and that the bacteria community seems to be highly dynamic and changing in the coral holobiont, not only in WPD, but in other common diseases as well (Gil-Agudelo et al. 2006; Ritchie 2006; Voss et al. 2007; Sunagawa et al. 2009). Descriptions of many coral diseases are limited and often confounded by the lack of clear diagnostic criteria with no pathological observations, so that similar disease signs may emerge in multiple coral species (Weil 2004, Raymundo et al. 2008; Work and Aeby 2006; Work et al. 2008). Moreover, the WPD putative pathogen *A. coralicida* has only been verified for a small number of species (Richardson 1998b; Pantos et al. 2003; Sutherland et al. 2004) and Koch's postulates only fulfilled for the pathogen isolated from *Dichocoenia stokesi* (Denner et al. 2003). There is no experimental data showing that the over 40 reported susceptible species to WPD, have been actually infected by *A. coralicida.*

Yellow band disease

Caribbean yellow band disease was first observed in Puerto Rico in 1996 with very low prevalence (Bruckner and Bruckner, 1997a; 2006). However, over the years, YBD has become the most important disease on reefs of La Parguera and other Caribbean localities (Cervino et al. 2001; Gil-Agudelo et al. 2004; Weil 2004; Weil et al. 2006; Bruckner and Bruckner 2006; Weil and Croquer 2009; Croquer and Weil 2009a). The disease is apparently produced by a complex of *Vibrio* bacteria that affects the zooxanthellae (Cervino et al. 2004, 2008; Weil et al. 2008). The etiology and dynamics of YBD have changed over the years, for example, from 1998 to 2002 the disease was highly seasonal, appearing in the summer-fall months, like WPD, and then arresting (disappearing) during the winter, when colonies did not show signs of it. Few previously affected colonies showed disease signs the next summer (reactivation of the infection or new infections) (data not shown). This seasonality disappeared around 2003, when the disease became chronic and colonies showed YBD-disease signs all year long (Fig. 6C). Even though the prevalence levels have been steadily increasing in the four inshore and mid-shelf reefs until the summer-fall of 2005, the disease was significantly more prevalent in the mid-shelf reefs (Turrumote and Media Luna), where populations of the susceptible *Montastraea faveolata* and *M. franksi* had higher densities and larger colonies compared to the inshore reefs (Weil unpub. data). Populations in the shelf-edge reefs seem to be less susceptible (low YBD prevalence over the years), possibly because of differences in light conditions (which could affect zooxanthellae), more resistant/different zooxantellae strains, depth-susceptible pathogens or any combination of these and/or other factors.

An intensive YBD outbreak started to develop in early spring 2006, peaking during the late spring and summer of 2006 in Media Luna and Turrumote. Disease mean prevalence were significantly higher (K-W, $p<0.01$) during winter and summer of 2006 compared to previous years (Fig. 6C). Data collected since 1998 in La Parguera showed that the average prevalence of YBD in the *Montastraea* species complex has significantly increased over time, from an average of 3.2 % (± 4.6) in 1998 to 42.7 % (± 26.9) in 2007. The yearly increase in mean YBD prevalence co-varied positively and significantly ($r^2 = 0.79$; $p<0.05$, Pearson's Coefficient) with increasing yearly average water temperatures (Fig. 7A).

Other evidence of the changing dynamics of YBD include an increase in the average number of focal lesions per colony in tagged colonies from two to seven, with some large colonies showing up to 35 disease lesions over their surface in 2007. More importantly however, YBD virulence measured in tagged colonies over the years, increased significantly (ANOVA, $p<0.05$) from 0.6 (± 0.8) cm/month in 2001-2003 to 2.19 (± 0.2) cm/month in 2007 (Fig. 7B), with some individual colonies showing average rates of lesion growth as high as 3.25 (± 0.8) cm/month. Virulence was significantly and positively correlated (Pearson Correlation Coeficient, $p<0.01$) with the increase in seasonal average temperatures (Fig. 7B).

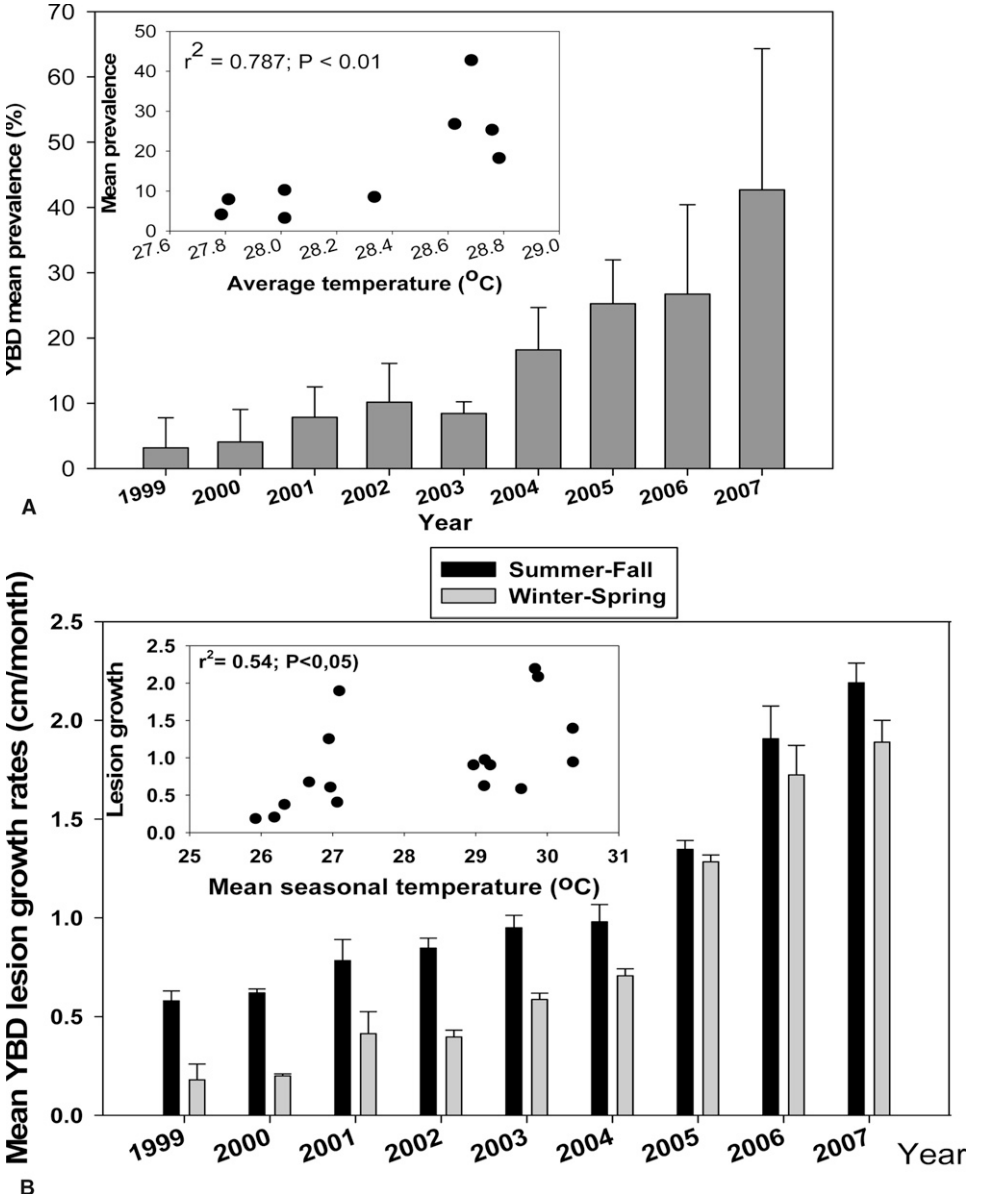

FIG. 7. (A) Temporal change in mean summer prevalence (SD) of YBD affecting *Montastrea spp.(M. faveolata, M. annularis and M. franksi* in La Parguera and Desecheo Island from 1999 to 2003 and in La Parguera from 2003 to 2007. Inset represents the correlation between average YBD prevalence and mean seawater temperatures over 9 years, from 1999 to 2007. (B) Seasonal variability in the rate of tissue mortality (virulence = lesion advance in cm/month) produced by YBD over nine years (1999-2007) in reefs of Desecheo and La Parguera. Inset shows the positive and significant correlation between virulence and the increase in average winter water temperatures.

The combination of higher number of lesions per colony, the chronic status of the disease, and the increase in linear mortality rates resulted in a significant increase (ANOVA, $p<0.01$) in overall YBD-induced tissue mortality, killing many small to large-sized colonies of *Montastraea faveolata*, *M. annularis*, and *M. franksi* over the last 5 years in several reefs off la Parguera. Similar observations have been reported for YBD

infected colonies in Mona and Desecheo islands off the west coast (Bruckner and Bruckner 2006; Bruckner and Hill 2009), and other Caribbean localities (Weil and Croquer 2009; Croquer and Weil 2009a).

Colonies infected with YBD bleached in 2005 with many of them showing a decrease or complete cessation of YBD signs while bleached through the winter of 2006. Many were infected with WPD for two-three months, and then, after WPD arrested, wider bands of YBD appeared, spreading over colonies during 2006 and 2007, increasing the area affected and the rates of tissue mortality (Fig. 7B). This pattern was somewhat different from the previously "normal" YBD signs observed in the same and other tagged colonies in Turrumote and Media Luna reefs.

Recent studies have shown that fecundity was significantly reduced in *Montastraea faveolata* colonies infected with YBD in at least four different ways: (1) loss of reproductive polyps due to tissue mortality, (2) reduction in fecundity in both affected and adjacent polyps, and in polyps away from healthy areas of infected colonies (a systemic effect), (3) increase of the "edge effect" by partial mortality over the colony (edge areas usually do not engage in sexual reproduction, thus polyps in these newly formed edges will not be contributing to the reproductive output), and (4) increase in the number of surviving patches (ramets) with sizes below the threshold for sexual reproduction (Weil et al. 2009). Reduced fitness could significantly hamper the natural recovery of the species and presumably, other reef-building species affected by disease. Similar reduction in fecundity has been reported for colonies of the sea-fan *Gorgonia ventalina* infected with aspergillosis (Flynn and Weil, 2009), and bleached *M. faveolata* colonies (Szmant and Gassman 1990).

The combination of disease and bleaching produced high coral mortality during 2006. It was difficult to separate the extent of mortality produced by the multiple factors. Overall, the abundance of large and medium sized colonies of *Montastraea faveolata*, *M. annularis* and *M. franksi* have been significantly reduced in most reefs off La Parguera, Mona, Desecheo, Guánica and the east coast of Puerto Rico (Bruckner and Bruckner 2006; Garcia et al. 2008; Weil unpubl.). Surviving ramets may have reduced fecundities and/or not reproduce for some time, significantly reducing the species fitness.

Seasonality and impact of coral bleaching

Signs of bleaching have been observed in different reefs almost every year since the intensive 1998 event. Two minor localized bleaching events occurred during the summer-fall of 1999 and 2001 in La Parguera. However, extensive and intensive bleaching events, with high bleaching prevalence in corals and other reef species occurred during the summer-fall of 2003 and 2005 (Fig. 8). The 2003 event affected up to 25 % of all reef coral species with many octocorals, zoanthids, hydrocorals, anemones, and some sponges becoming 100% bleached and/or translucent. Up to 90% colony mortalities were observed in populations of the hydrocoral *Millepora* in many shallow reef habitats of La Parguera (Velazco et al. 2003; McClanahan et al. 2008; Weil unpub. data). However, similar to previous bleaching events in Puerto Rico and other Caribbean localities, scleractinian coral mortality was low (1%), with partial mortality occurring in a small proportion of colonies of *Porites*, *Montastraea*, *Colpophyllia*, *Siderastrea* and *Agaricia* (McClanahan et al. 2009). Background bleaching (normal paling of colonies during summer-fall) were observed in 2004 at many reefs (Fig. 8). Even though the summer of 2004 had higher temperatures than 2003 and 2005 (Fig. 5), no major bleaching was observed. In 2005, the worst bleaching event on record for the eastern Caribbean started in late summer and early fall after a maximum of 14.3 accumulated degree heating weeks (DGWs) produced record-breaking warm sea surface temperatures (SSTs) up to 31.8°C at 30 m depth, and up to 33.1°C at reef crests (NOAA, Hernández-Delgado, unpubl.). A total of 82 cnidarian species bleached in Puerto Rico during 2005, including 52 scleractinians, 13 octocorals, four hydrocorals, four zoanthids, four actiniarians, three corallimorpharians and

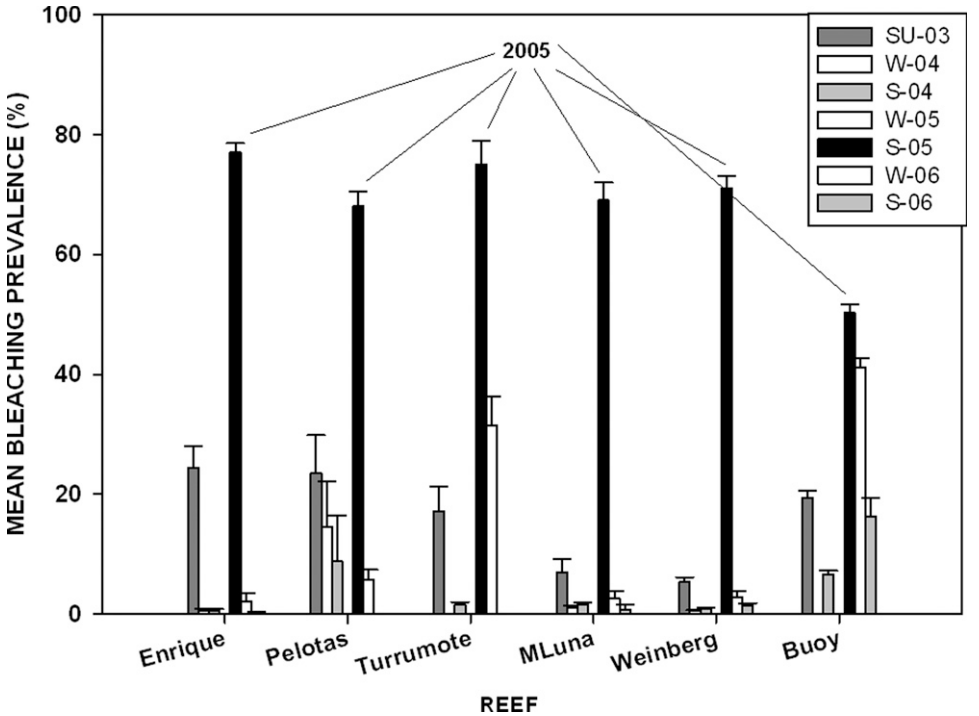

FIG. 8. Temporal (seasonal) variability in bleaching prevalence in each reef surveyed every summer-fall season from 2003 to 2006 in La Parguera, south-west coast of Puerto Rico. Reefs sequence indicates east-to-west location and the inshore off-shore transect. SU-03 = summer 2003, WI-04 = winter of 2004, SU-04= summer 2004, WI-05= winter 2005 and so on.

two scyphozoans (García-Sais et al., 2006; McClanahan et al. 2009).

As a result of the 2005 bleaching event, most coral reefs in La Parguera had mean coral bleaching prevalence exceeding 50%. One inshore reef (Pelotas), one mid-shelf reef (Turrumote) and one shelf-edge reefs had coral bleaching prevalence values higher than 70% (Fig. 8). Some populations of *Montastraea*, *Colpophylllia* and *Diploria* at these reefs had up to 95 % of all colonies totally bleached (data not shown). These results are similar to reports in other localities in the east coast of Puerto Rico (García-Sais et al., 2006) and the Virgin Islands (Miller et al. 2006, 2009; Rogers et al. 2008). These localities showed mean bleaching prevalence higher than in other Caribbean localities (Croquer and Weil 2009b). On the east coast of Puerto Rico, bleaching was significantly more severe and prolonged in protected (leeward) reefs than at reefs under moderate or strong water circulation, affecting 80-97% of the corals at leeward reefs, 60-80% at reefs with moderate circulation and only 20-60% at exposed reefs with stronger water circulation. A total of 37% of surveyed coral species in reefs of the east coast were 100% bleached, 24% of the species were 80-99% bleached, 29% were 50-80% bleached, and 10% were 20-50% bleached (Hernández-Delgado et al 2006; Garcia et al. 2008). Bleaching affected up to 42 coral species in 2005, including *Mycetophyllia* spp, *Madracis* spp. *Isophyllastrea* sp, *Scolymia lacera*, etc. that have not been observed bleached in La Parguera and other reef localities of Puerto Rico and the Caribbean (Aronson et al. 2002; Hernandez-Delgado et al. 2006; Cróquer and Weil 2009b) as well as a suite of other members of the reef community such as hydrocorals, octocorals, zoanthids and sponges (McClanahan et al 2009; Prada et al. 2009).

Many coral colonies and other zooxanthellated organisms regained partial coloration

within three-four months at reefs off La Parguera; however, a high proportion of coral colonies were 100% bleached for six months. Similarly to the WPD outbreak, the extended bleaching was probably a consequence of the higher than normal water temperatures during the winter-spring of 2006, which had the highest bleaching prevalence ever recorded for this season in La Parguera (Fig. 8). Even a year later, many colonies still exhibited signs of bleaching, with pale blotches and white areas over the colony surface at many reefs off La Parguera. Between 15 and 30% of the coral colonies had bleaching signs at Turrumote and the Buoy with other reefs showing lower, but still higher than normal bleaching prevalence (Fig. 8). Higher bleaching prevalence was found at intermediate and deep habitats at mid-shelf and shelf-edge reefs in 2006 compared to 2005 (data not shown), supporting the hypothesis that colonies frequently exposed to higher temperatures (i.e. shallower habitats) are better suited to recover faster from intensive bleaching events (Buddemier et al. 2004; Baird et al. 2008; Thomson and van Woesik 2009). No bleaching was observed during 2007 and 2008 at reefs off La Parguera.

The 2005 bleaching event and the subsequent outbreaks of WPD and YBD produced the highest mortalities of scleractinians ever recorded in La Parguera. Populations of *Agaricia*, *Acropora, Millepora* and the encrusting octocoral *E. caribaeorum* suffered high mortalities from bleaching, and *Mycetophyllia*, *Montastraea*, *Colpophyllia*, *Diploria*, *Siderastrea* and *Stephanocoenia* suffered high tissue and total colony losses from the initial impact of bleaching and WPD. Further mortality in the three species of *Montastraea* was compounded by the YBD outbreak after the peak of the bleaching and WPD outbreak. Results indicate that the *M. faveolata* populations lost an average of 50-60% of their live tissues at intermediate and deep habitats between 2003 and 2007 in Turrumote and Weinberg reefs. This represents an alarming rate of tissue loss for large, old (over 500 years old, some even older the 1,000 years) colonies of the larger species in such a short period of time.

Overall, reefs off La Parguera lost an average of 53% (± 3.5) of live coral tissue over a four year period (Fig. 9), the highest and fastest tissue loss ever recorded at this spatial level in La Parguera. Coral cover decrease from an average 22% to 10.5 % with most reefs losing over 50 % of their coral cover (Fig. 9). Similar losses were reported for the east coast of Puerto Rico where 20-60% decline in live coral cover was recorded within six months, most of this from *Montastraea* spp. (Garcia et al. 2006), and for the US. Virgin Islands, where up to 60% of live coral cover was lost over one year after the onset of the bleaching and WPD events (Miller et al. 2009). Nearly all of the colonies of important reef-building coral species suffered significant partial colony mortality.

Summary

The patchiness and irregular distribution of coral diseases and bleaching along the variable horizontal and depth related reef community, and/or reef locations, is related to differences in the distribution, composition and abundances of the susceptible coral species, the environmental conditions and sensibility of hosts and/or pathogens to disturbed environments, or combination of these and other factors (Harvell et al. 1999; 2004; Weil et al. 2006; Raymundo et al 2006; Bruno et al. 2007; Cróquer and Weil 2009a,b; Bruckner and Hill 2009). Development of pathogenesis in coral tissues might depend on lowered coral resistance resulting from anthropogenic or environmental stress (Peters 1997; Geiser et al. 1998; Harvell et al. 1999, 2002; Carey 2000). Even though when all years were pooled, no significant difference in the total mean coral disease prevalence was found between the inshore and offshore reefs (Fig. 3). When individual years, or individual diseases or populations are considered (Fig. 6), significant differences in mean prevalence across habitats and reef zones (data not shown) are present. White patches (= white pox, patchy necrosis) for example, was more prevalent in shallow, *A. palmata* dominated habitats in mid-shelf reefs in the early 2000's compared to

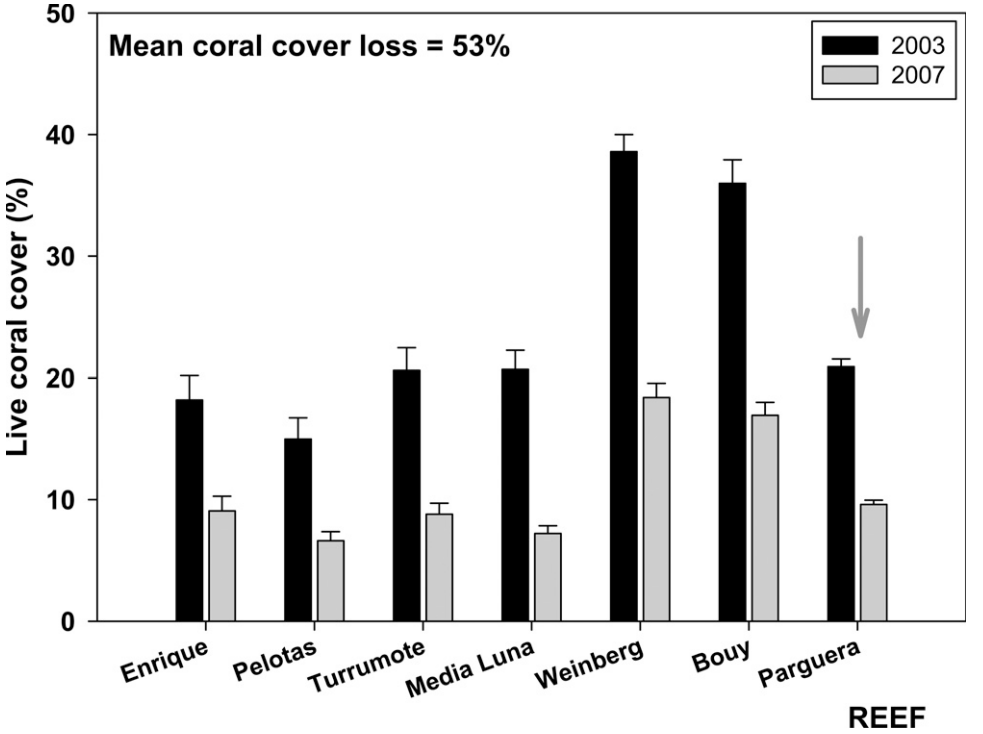

FIG. 9. Mean percent of live coral cover in each reef sampled in 2003 (black column) and 2007 (gray column) for the six reefs in La Parguera. Coral cover decreased from The average loss in percentage of live coral cover (%) was 53.7% (±7.2) in these six reefs from 2003 to 2007.

shallow habitats in inner reefs (Weil and Ruiz 2003) (data not shown). Significantly higher YBD and WPD prevalence was found at intermediate depths compared to shallow and deep habitats in both inshore and mid-shelf reefs, and at mid-shelf and shelf-edge reefs, where the habitats were dominated by high abundances of *Montastraea* susceptible colonies. Similar patterns were observed for some of the other reefs and individual diseases in Puerto Rico and other reef localities in the Caribbean (Cróquer and Weil 2009a,b; Bruckner and Bruckner 2006).

There is evidence that the microbial consortium responsible for BBD is more prevalent on corals subjected to anthropogenic stress compared to colonies in healthy reefs (Taylor 1983; Antonius 1985; Kuta & Richardson 2002), and correlative studies suggest that the incidence and severity of certain diseases increase under high nutrient concentrations (Bruno et al. 2003; Kaczmarsky et al. 2005) or anthropogenic influences such as coastal development, sewage discharges etc. (Green & Bruckner 2000; Sutherland et al. 2004). Environmental conditions in La Parguera have been changing (deteriorating?) over the last decades mostly due to local coastal development, overfishing, inland and coastal development in upstream areas (Garcia et al. 2008) and global warming. However, instead of higher numbers of disease affecting reef organisms and higher disease prevalence in inshore reefs compared to offshore reefs, results of this and other extensive local and geographic studies seem to indicate that the number and prevalence of diseases is higher in reefs far away from coastal developed centers (Weil et al. 2002; Weil 2004; Bruno et al. 2003; Cróquer and Weil 2009). Most of these studies however, report results of one or two years of surveys, a short time to characterize the spatial and temporal dynamics of the different diseases.

Results of this study underline the importance of long term surveys in the same localities by showing the high temporal and spatial variability in total disease prevalence and prevalence of individual diseases such as WPD and YBD. For example, in 2003, offshore reefs had higher prevalence of WPD and total diseases compared to mid- and inner-shelf reefs in La Parguera. In 2004, there was no significant difference between inner-, mid-shelf and shelf-edge reefs, and in 2005, 2006 and 2007, mid-shelf reefs showed significant higher mean YBD and total disease prevalence compared to inner-shelf and shelf-edge reefs (Figs. 3 and 6). More extended studies with quantitative data are needed.

The role of coral diseases as a primary cause of reef decline is controversial because for some, coral diseases are secondary conditions and not the primary cause of coral reef decline (Pandolfi et al. 2003, 2005; Lesser et al. 2007). Besides the WBD epizootic of the late 1970's and early 80's, which was responsible for the loss of many acroporid populations Caribbean wide, no similar pan-geographic disease outbreak occurred until recently, when yellow band disease, with its widespread distribution and high prevalence in *Montastraea* spp. populations from Bermuda to the southern Caribbean, seems to be threatening to significantly reduce populations of one of the most important reef-building genera in the region (Rogers et al. 2008; Weil and Cróquer 2009; Weil et al. 2009; Bruckner and Hill 2009, this study). The potential for more local outbreaks of other diseases and bleaching events exists. Sea water temperatures are increasing in addition to the problem of acidification, all of which will continue to affect coral reef communities, or what is left of them.

The continuous decline (in live cover and abundance) observed in populations of the *Montastraea* complex in Puerto Rico and throughout the Caribbean coupled with the significant reduction in fecundity in *M. faveolata* caused by bleaching and YBD (Szmant and Gassman 1990; Weil et al. 2009), and the combination with other factors could have prolonged negative effects on their reproductive success and net reef accretion rates, essentially hampering the potential for natural population recovery and reef recovery. The sudden collapse of entire assemblages of several coral species suggest the onset of a rapid Allee effect which could result in prolonged reproductive failure for reef-building species. Similar declines in other important structural and abundant species, the acroporids, have already caused significant phase shifts in Caribbean coral reef community structure in the past (Gladfelter 1982; Aronson and Prech 2001).

The events of the last decade throughout the Caribbean highlighted concerns regarding the reduction in resilience of coral reefs and their sensitivity to climate change (Hughes et al. 2003; Jackson et al. 2001; Gardner et al. 2003; Pandolfi et al. 2003; Bellwood et al. 2003, 2004; Pandolfi et al. 2005). This trend seem to be primarily driven by significant reductions in coral cover and population sizes of major reef-building genera, particularly, in the Caribbean region where coral diseases have played an important role in coral mortality (Aronson et al. 2004; Weil 2004; Miller et al. 2006, 2009; Bruckner and Bruckner 2006; Bruckner and Hill 2009). It seems that we could be witnessing local community "phase-shifts" in several reef localities, from what used to be *Montastraea*-dominated habitats and reefs for many years (> 100 years or more given the average colony sizes) to algae-dominated habitats and reefs. In La Parguera for example, live coral cover was reduced from an average 25.0 % (±9.1) in 2003 to 11.2% (± 5.1) in 2007 (Fig. 9), a net loss of 53.7% (± 8.3) in coral cover in nine reefs sampled from the inshore to the shelf-edge. This decrease in coral cover was matched by concominant increases of up to 80% in algae cover in some reefs (Ruiz and Ballantine, 2009, Weil unpub. data), presenting managers and decision makers with challenging problems in the near future.

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